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THESIS ADVISOR

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HYPOTHESIS

The experimental group with four cranial osteopathy treatments and exercise will show a faster time during the Timed Up and Go (TUG) test of function and mobility than the control group using exercise only with statistical significance of $p<.05$ or better.

ABSTRACT

Parkinson's disease afflicts primarily seniors, of whom two-thirds are likely to fall and be admitted to hospital. It will become a growing concern both financially and socially in the coming years as the population of Canada ages. Traditional paramedical treatment of Parkinson's disease has been directed at combating the symptoms or using coping techniques rather than addressing the underlying cause in the basal ganglia and brain.

The purpose of this study was to determine the effects of cranial osteopathic treatment on functional mobility of patients with idiopathic Parkinson's disease. A single blind between-group design was used to evaluate eleven Parkinson's patients between the ages of 55 and 82. The study used the Timed Up and Go (TUG) test to measure the change between a control group that received a standard exercise program and an experimental group that received four cranial osteopathic treatments in addition to the physiotherapist-supervised exercise program. The exercise program was taught and supervised by a registered physiotherapist in Ontario based on the exercises suggested by the Parkinson's Society of Canada. An osteopathic evaluation measuring cranial movement and vitality was assessed pre- and post-intervention for both groups.

A Kruskal Wallis (KW) Exact for the pre- and post-test of the Timed Up and Go (TUG) times was used. Due to the abnormal data distribution, a KW non-parametric test was used instead of the parametric t-test. The KW test is less powerful and thus yielded a higher p value of p=.23. The experimental group yielded a greater improvement than the control group. The four participants in the control group all improved in their TUG times, with a mean improvement of .22 seconds. The experimental group TUG times improved,

with a mean improvement of 1.50 seconds and the largest improvement being 5.5 seconds.

An osteopathic evaluation was added pre- and post-intervention documenting the severity of osteopathic lesions, based on the Canadian College of Osteopathy classification system, and the vitality of the structures directly related to the craniosacral mechanism. The experimental group showed statistically significant improvements in 29 of 38 structures measured in vitality and 17 of 38 structures of lesion classification in the structures assessed versus none in the control group. The structures that did not reach statistical significance were related to a better starting value rather than a lack of improvement. Although the structures improved in lesion classification and vitality, they did not improve enough to reach statistical significance. The control group had no change in their osteopathic findings, helping to support changes made to the structures with cranial osteopathic intervention. The most common osteopathic lesions were the left C0-C1, left petro-basilar suture, the left nasal bone and the sphenobasilar symphysis.

Improvement trends in the TUG test and the very strong results in blinded osteopathic findings should stimulate further research in the area of cranial osteopathy and its use for Parkinson's disease treatment. Based on the results of this pilot study, a full research protocol is warranted using this research methodology.

ABSTRAIT

La maladie de Parkinson affècte principalement les personnes âgées, dont les deux-tiers ont une chance de tomber accidentellement et d'être admis à l'hôpital. Avec la population canadienne vieillissante, ce problème deviendra sociallement et financièrement plus important dans les années à venir. Traditionnellement, le traitement paramédical de la maladie de Parkinson a été concentré sur les symptômes, ou encore utilise des techniques d'adaptation, plutôt qu'adresser les causes initiales du cerveau et du noyau gris central.

Le but de cette étude était de déterminer les effets de traitement ostéopathique crânien sur la mobilité fonctionnelle des patients atteints de la maladie de Parkinson idiopathique. Onze patients âgés de 55 à 82 ans, atteints de la maladie de Parkinson ont participé à une étude entre-groupe. Le test de mobilité et de fonctions « Timed Up and Go » (TUG) a été utilisé pour mesurer les changements entre un groupe de contrôle ayant reçu un programme d'exercises standard et un groupe expérimental ayant reçu quatre traitements ostéopathiques crâniens ainsi qu'un programme d'exercises suppervisé par un physiothérapeute. Ce programme d'exercises suggéré par la société Parkinson Canada, a été suppervisé et instruit aux candidats par un physiothérapeute enregistré en Ontario. Une évaluation ostéopathique mesurant les mouvements crâniens ainsi que la vitalité des structures a été effectuée pré-et post-intervention pour les deux groupes.

Un extrait de Kruskal Wallis (KW) sur le « pré» et « post» test du « Timed Up and Go » a été utilisé. Étant donné l'anormalité de la distribution de données, un test KW non-paramétrique a été utilisé au lieu du test-t paramétrique. Le test KW est moins efficace et puissant, et a ainsi un rendemment générallement d'une plus grande valeur « p » de

p=.23. Le groupe exéprimental a démontré une plus grande amélioration que le groupe de contrôle. Le groupe de quatre participants (dit groupe de contrôle) ont tous améliorés leur temps sur le test TUG avec une amélioration de .22 secondes. Le groupe expérimental a aussi vu ces temps du test TUG améliorés, avec une amélioration de 1.50 secondes et la meilleure amélioration étant de 5.5 secondes.

Une évaluation ostéopathique a été ajoutée pré-et post-intervention, documentant la sévérité des lésions ostéopathiques, basée sur le système de classification du Collège Canadien de la Médecine Ostéopathique, ainsi que la vitalité des structures directement reliées au mécanisme craniosacral. Le groupe expérimental a démontré une amélioration statistiquement significative sur 29 des 38 structures mesurant la vitalité, et une amélioration statistiquement significative sur 17 des 38 structures de classification des lésions évaluées, comparativement à aucune du groupe contrôlé. Les structures n'ayant pas atteintes d'amélioration statistiquement significative étaient reliées à une valeur de base plus élevée plutôt qu'un manque d'amélioration. Bien que les structures ont demontrées une amélioration dans la classification des lésions et de la vitalité, elles ne se sont pas ameliorées suffisamment pour atteindre le statut "statistiquement signaficatif". Le groupe de contrôle n'a vu aucun changement sur les résultats ostéopathiques, aidant ainsi à supporter des changements effectués aux structures dans l'intervention ostéopathique crânien. Les lésions ostéopathiques les plus communes ont été identifiées comme étant C0-C1, la suture pétro-occipitale gauche, l'os nasal gauche ainsi que la symphyse sphéno-occipitale.

Les tendances d'améliorations au test TUG, ainsi que les améliorations significatives démontrées durant les observations ostéopathiques a l'insu pourront

stimuler de plus amples recherches dans le domaine ostéopathique crânien et son utilité pour le traitement de la maladie de Parkinson. Basé sur les résultats de cette étude pilote, un protocole de recherche complet utilisant cette méthodologie de recherche est justifié.

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1 CHAPTER ONE: INTRODUCTION

There is a clinical understanding that cranial osteopathy is an effective treatment modality. However, only limited research exists to substantiate this clinical experience in the treatment of Parkinson's disease. This study seeks to demonstrate the positive effects of cranial osteopathy as a treatment by improving the gait and mobility of patients with Parkinson's disease as measured by the Timed Up and Go (TUG) test.

Parkinson's disease is the second most diagnosed degenerative neurological disease in the world, affecting approximately 3,765,000 people worldwide (Stern, 1993). Idiopathic Parkinson's disease primarily affects the senior population and occurs in one in every one hundred people over the age of 65 (De Lau & Breteler, 2006). From the year 1991 to 2016, the projected increase in Canadians over the age of sixty-five is expected to result in a 92 percent increase in the number of people with Parkinson's disease ("Population estimates and projections," 2008). Both neurologists and patients are less satisfied with current treatment of Parkinson's disease when compared with other chronic diseases despite the wide array of available treatment options for Parkinson's disease (Fargel, Grobe, Oesterle, Hastedt, & Rupp, 2007).

Parkinson's disease is a chronic neurodegenerative disease associated with selective loss of the dopaminergic neurons in the pars compacta of the substantia nigra (Lang & Lozano, 1998). Parkinson's disease is characterized by both motor and non-motor symptoms. The exact cause of the degeneration of Parkinson's disease is unknown. There are a number of working theories. These include inflammatory factors (McGeer & McGeer, 2004), oxidative stress (Jenner, 2003), and a possible genetic link (Gwinn-Hardy, 2002). Parkinson's disease is characterized by its motor symptoms: tremor,

rigidity, bradykinesia (slowing of movement), gait and postural disturbance. Non-motor symptoms include depression, personality changes, and olfactory changes.

The motor symptoms of Parkinson's disease occur primarily as a result of the destruction of the dopaminergic neurons in the substantia nigra and nigro-striatal pathways to the basal ganglia that are believed to control the sequencing of automatic, well-learned movements, such as gait and eating. The basal ganglia also aid in the preparation and maintenance of motor plans and keep these motor plans in a state of readiness for action (Robertson & Flowers, 1990). Newer studies and advances in imaging techniques support the multi-factorial neurodegenerative process in the rest of the cortex in addition to the dopamine depletion seen in the substantia nigra (Maguire-Zeiss & Federoff, 2010; Paschali et al., 2010; Qian, Flood, & Hong, 2010).

Gait and balance are affected by the impairment of postural reflexes and the associated difficulty in executing and timing automatic physical responses (Ashburn, Stack, Pickering, & Ward, 2001b). The slowing of movement, rigidity, and postural alterations impact both gait and function. The impairments caused by Parkinson's disease worsen with time, and as impairments worsen, activity limitations, participation restrictions, and psychosocial problems develop.

Those diagnosed with Parkinson's disease experience decreased function and a reduced quality of life (Ashburn, Fazakarley, Ballinger, Pickering, McLellan, & Fitton, 2007; Koller, Glatt, Vetere-Overfield, & Hassanein, 1989), with disorders of gait being one of the most common symptoms (Hoehn & Yahr, 1967). Disorders of gait can include a reduction in stride length, reduced velocity, a shuffling gait pattern, and falls. Gait disorders get progressively worse as the disease advances, limiting the patient's quality of

life and increasing their risk of falling. The incidence of falls in people with Parkinson's disease is as high as 68 percent (Wood, Bilclough, Bowron, & Walker, 2002) and an association has been demonstrated between functional mobility and balance confidence (Hatch, Gill-Body, & Portney, 2003). Parkinson's disease patients were found to fall more than three times as often compared to their age-matched controls (Ashburn, Stack, Pickering, & Ward, 2001a). Falls are one of the most common reasons for emergency room visits in the elderly, and complications of falling can lead to a fear of falling and a general loss of independence (Ashburn, Fazakarley, Ballinger, Pickering, McLellan, & Fitton, 2007; Johnell, Melton, Atkinson, O'Fallon, & Kurland, 1992). Forty-two percent of Parkinson's disease fallers have been reported to have a fear of future falls (Bloem, Grimbergen, Cramer, Willemsen, & Zwinderman, 2001). As Parkinson's disease progresses balance, falls, and the loss of mobility increase. The presence of gait disturbances was found to be associated with an increase in mortality risk (Bennett et al., 1996).

Current treatment with pharmacological agents, of which levodopa has been the first-line drug of choice, is no longer sufficient. Side effects of long-term levodopa use, such as dyskinesia and freezing and failure at end of use, contribute to the dissatisfaction reported by Parkinson's patients (Fargel, Grobe, Oesterle, Hastedt, & Rupp, 2007). Future drug treatment appears focused beyond the traditional replacement of dopamine with levodopa. In the search for a more effective treatment, and a cure, research today includes neuro-protective and neuro-restorative methods aimed at prevention of cell loss or death. By affecting the factors that may be contributing to the early cell death and sub-clinical features researchers hope to slow or stop this damage for those with Parkinson's

disease. Research in these areas of pharmacological treatment for Parkinson's disease offers hope through improved drug treatment in the future.

The TUG test is a functional measure used in research studies involving the elderly and those with Parkinson's disease. It is an excellent determinant of function in the elderly and has been used frequently in the study of function with those suffering with Parkinson's disease (Brusse, Zimdars, Zalewski, & Steffen, 2005; Ellis, Cavanaugh, Earhart, Ford, Foreman, & Dibble, 2011; Ellis, Katz, White, DePiero, Hohler, & Saint-Hilaire, 2008; Hirsch & Farley, 2009; Smithson, Morris, & Iansek, 1998; Steffen, Hacker, & Mollinger, 2002; Vereeck, Wuyts, & Truijen, 2008). The TUG test correlates well with other outcome measures and has been shown to be more useful in determining quality of life than simple strength and flexibility testing. The loss of locomotion has a detrimental effect on one's sense of self-worth and independence. Mobility is reported by Parkinson's patients to be the most problematic area and its loss has been shown to most adversely affect their quality of life (Fargel, Grobe, Oesterle, Hastedt, & Rupp, 2007). Ellis, Katz, White, Depiero, Hohler and Saint-Hilaire (2008) reported that outcome measures including functional testing, such as the TUG test, correlate best to one's quality of life.

There is need for further research into improving gait and mobility for Parkinson's patients both from a patient satisfaction and a safety perspective. The aim of this study is to demonstrate the effectiveness of cranial osteopathic treatment on functional mobility of those suffering with Parkinson's disease. There are preliminary studies looking at the relationship between osteopathic cranial somatic dysfunctions and the use of cranial osteopathic treatment with Parkinson's disease and other neurological

diseases (Frymann, 1976a; Frymann, Carney, & Springall, 1992; Greenman & McPartland, 1995; Rivera-Martineza, Wells, & Capobianco, 2002; Sandhouse et al., 2010).

Functional mobility is a chief concern with Parkinson's disease as improved gait and balance will improve independence, and will reduce falls and the fear of falling. This would improve the quality of life of those with Parkinson's disease and would reduce hospital admissions and minimize the burden on the healthcare system and society. This pilot study will attempt to provide some evidence, stimulate thought and discussion, and help guide future research for alternative care directed towards helping those with Parkinson's disease.

2 CHAPTER TWO: LITERATURE REVIEW

MEDICAL REVIEW OF PARKINSON'S DISEASE

Parkinson's disease is a neurodegenerative disorder that primarily affects voluntary, coordinated movement. The majority of Parkinsonian symptoms result from a loss of dopamine and its related receptors in the basal ganglia, or more specifically, the substantia nigra. The cause of this neurodegeneration is not yet known and a cure has not been discovered. Parkinson's disease may be a final outcome of a complex set of interactions. Research shows both genetic and environmental factors appear to play a role in the development and progression of Parkinson's disease (Gwinn-Hardy, 2002; Hardy, Cookson, & Singleton, 2003; Hirsch, 2007; Jenner & Olanow, 1998, 2006; Rakshi et al., 1999).

2.1 DIAGNOSIS OF PARKINSON'S DISEASE

The diagnosis of Parkinson's disease in the living patient is based on the results of clinical assessment. A clinical diagnosis of Parkinson's disease is made when at least two of the three cardinal signs (bradykinesia, rigidity, and resting tremor) are present (Hughes, Daniel, Kilford, & Lees, 1992; Rajput, 1993). True diagnosis of Parkinson's disease can only be done by pathological analysis; the disease is characterized by nerve cell loss in the substantia nigra and the presence of Lewy's intra-neuronal inclusion bodies (Forno, 1996). Lewy bodies in Parkinson's disease are considered brainstem Lewy bodies. They are spherical protein masses that develop inside nerve cells. The primary structural component is alpha-synuclein, which is thought to represent an excessive protein accumulation as a result of the cell degradation that the cell is unable to remove (Tanaka, Kim, Lee, Junn, Iwatsubo, & Mouradian, 2004). Unfortunately, this pathological evidence is not available in most clinical settings, and therefore a definitive

diagnosis of Parkinson's disease is difficult for the clinician. If two of the three cardinal signs are present and there is an asymmetrical onset and the patient has a good or excellent response to the medication levodopa, the diagnostic accuracy of Parkinson's disease is increased. Levodopa is a precursor to dopamine which can cross the blood-brain barrier and increase the available dopamine in the brain and basal ganglia and is the most common medication used to treat Parkinson's disease (Jankovic, 2008).

The discovery of biomarkers has improved diagnosis of Parkinson's disease. This has allowed for early identification and hope that a cure may be found in the future (Jankovic, 2008). Newer imaging techniques such as single photon emission computed tomography (SPECT) have helped the clinician more definitively diagnose Parkinson's disease in vivo (Paschali, et al., 2010). The technique uses a gamma ray–emitting isotope that is tagged to a cocaine derivative (ioflupane, I-FP-CIT). This binds to the pre-synaptic dopamine reuptake site in the striatum (caudate and putamen). Using a SPECT scan allows dopamine uptake to be seen. The uptake is reduced in those with Parkinson's disease compared to normals. This serves as a useful differential diagnostic tool to differentiate Parkinson's disease from other neurological disorders such as intention tremor, which is often confused and misdiagnosed in the early stages of Parkinson's disease (Clarke, 2007). Biomarkers have been used in other neurological degenerative diseases (Tuppo & Forman, 2001) to determine disease causes and have provided researchers with proof that those neurodegenerative disorders are a result of oxidative stress.

2.2 SIGNS AND SYMPTOMS OF PARKINSON'S DISEASE

The most recognizable and characteristic symptom of Parkinson's disease is bradykinesia. It is usually associated with a basal ganglia disorder. One of the roles of the basal ganglia is to maintain the set-related 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 and gait. The issue of balance in Parkinson's disease appear to be linked to these set-related neurons. Assessment of bradykinesia includes tests that require the patient to perform rapid, repetitive, alternating movements of the hand or fingertips, or heel taps. Not only is slowness observed, but also decrementing amplitude. This differs from other basal ganglia disorders such as Huntington's disease and dystonia. Basal ganglia disorders include difficulties with planning, initiating, and executing movement and with performing sequential and simultaneous tasks (Berardelli, Rothwell, Thompson, & Hallett, 2001). In Parkinson's disease this slowness in movement occurs in conjunction with a reduction in the amount of spontaneous movement.

Although tremor, rigidity, bradykinesia or akinesia, and postural instability are the telltale signs of Parkinson's disease, they are not the only symptoms. There is a broad spectrum of clinical manifestations including depression. Parkinson's disease is no longer believed to be associated only with movement disorders as a result of dopamine deficiency in the basal ganglia. It has been shown to incorporate disorders of the neurotransmitter chemicals serotonin and norepinephrine as well as dopamine, including neurotransmitter dysfunction in the gastrointestinal system, which may help to explain some of the digestive symptoms in those with Parkinson's disease. Other features of Parkinson's disease are now believed to arise from a malfunctioning limbic system, the

region responsible for behaviours and emotions, the nucleus coeruleus, and other areas such as the frontal lobe (Rakshi, et al., 1999). This has led to the discovery of some predicting signs such as prolonged anxiety or unexplained depression. Intestinal dopamine cells can degenerate in people with Parkinson's and this may be important in the gastrointestinal symptoms that present as part of the disease. Other changes in the Parkinsonian brain include less activation in the anterior supplementary motor area or premotor area, the anterior cingulate cortex, the dorsolateral prefrontal cortex, the basal ganglia, and the thalamus, compared to the normal brain (Jenkins et al., 1992). Newer imaging techniques such as single-photon emission computerized tomography (SPECT) have improved the understanding of the cortical changes that occur in the brain of a Parkinson's disease patient by measuring the degree of perfusion in the brain. In the early stages of Parkinson's disease hypo-perfusion is seen in the basal ganglia and frontal lobe, and as the disease progresses hypo-perfusion progresses to the parietal and temporal lobes (Paschali, et al., 2010). Neuropsychological performance seems to decline in parallel with this loss of perfusion (Paschali, et al., 2010). This supports the multi-factorial degeneration process in Parkinson's disease.

Symptoms of Parkinson's disease only become evident once there is a greater than 80 percent loss of striatal dopamine (Marsden, 1990). Marsden (1990) suggested that the disease might exist for up to thirty years before symptoms present themselves. The loss of dopamine occurs primarily in the substantia nigra, reducing the available dopamine in the basal ganglia. The condition is characterized by a depletion of dopaminergic neurons and a reduction in dopamine receptor sites in the striatum

(Marsden, 1994). Wang, Aziz, Stein, Bain, and Liu (2006) showed that there was an in vivo difference on PET scan imaging between early and late stage Parkinson's disease.

2.3 PATHOPHYSIOLOGY OF PARKINSON'S DISEASE

The pathophysiology of this slow and progressive damage to the basal ganglia is thought to be the result of several degenerative mechanisms. Basal ganglia damage has been reported to result from mitochondrial dysfunction. There is support for an oxidative stress model. The presence of pro-inflammatory cytokines is suggestive of chronic inflammation (Jenner, 2003; Jenner & Olanow, 1998, 2006). Abnormalities in the processing of cell proteins related to the ubiquitin-proteasome system are linked with the presence of Lewy bodies (McNaught, Olanow, Halliwell, Isacson, & Jenner, 2001). A slow destruction of neurological function occurs in other neurological pathologies and is not unique to Parkinson's disease. Pre-clinical signs and symptoms, such as olfactory loss in those with Parkinson's, may lead to early detection before the cardinal signs of tremor and rigidity occur and may lead to improved results with neuroprotective care. These early pre-clinical signs of neural dysfunction have been shown in other conditions in both the peripheral and central nervous systems. Minor changes in motor, sensory, and sudomotor activity have been shown to occur with neuropathy. These minor sub-clinical signs begin before the true neurological signs appear (Gunn, 2009; Low, Sandroni, Fealey, & Low, 2006). Diabetic neuropathy has also shown that minor damage to the neurological tissues can lead to early detection of Type I diabetes. Hendriksen, Oey, Wieneke Bravenboer, and van Huffelen (1993) found that symptoms of temperature and vibration were lost in Type I diabetes on 60 otherwise symptom-free patients.

Hatano (2009) found selective loss of mitochondrial complex I and the alpha-ketoglutarate dehydrogenase complex in the nigral neurons of patients with Parkinson's

disease. These observations have helped establish the connection of mitochondrial defects with Parkinson's disease. Mitochondrial respiratory failure induces oxidative damage in neurons, associated with an increase in hydroxynonenal and 8-oxo-deoxyguanine, indicating oxidative damage in the nigral neurons of Parkinson's disease. These abnormalities can trigger apoptotic cell death. The primary events that induce mitochondrial failure and oxidative damage are not known; however, it has been postulated that the interaction of genetic risk factors and environmental factors could initiate the degenerative process.

Other authors (Bastianello et al., 2011; Plasmati, Pastorelli, Fini, Salvi, Galeotti, & Zamboni, 2010; Zamboni et al., 2009) have shown that vascular insufficiencies are more prevalent in neurological disorders and are a factor in the cell death associated with the disease. This is a new finding in the area of research investigating vascular insufficiency as a possible contributing cause to neurodegenerative disorders such as multiple sclerosis. A search of the literature in PubMed, CINAHL, and Google did not produce any investigations with respect to Parkinson's disease. The acceptance of surgical intervention to correct venous insufficiency has not yet been statistically proven in any random control trials (RCTs) to date, but Health Canada has committed funding to create its own study of its efficacy. To date there have not been similar investigations into its connection to Parkinson's disease.

A closer look at Parkinson's disease and toxicity was made possible in the late 1970s because of the association of its sudden clinical onset with use of a narcotic, injectable substance call MPTP (1-methyl-4-phenyl 1,2,3,6- tetrahydropyridine). This discovery ultimately led to animal models of drug-induced Parkinson's disease. It was

revealed that MTPT was neurotoxic to dopaminergic neurons by way of a reaction through the active metabolite called MPP (1-methyl-4-phenylpyridinium), which was formed by the action of monoamine oxidase in glial cells. This increases the risk of oxidative damage significantly (Langston & Ballard, 1984) (Figure 1).

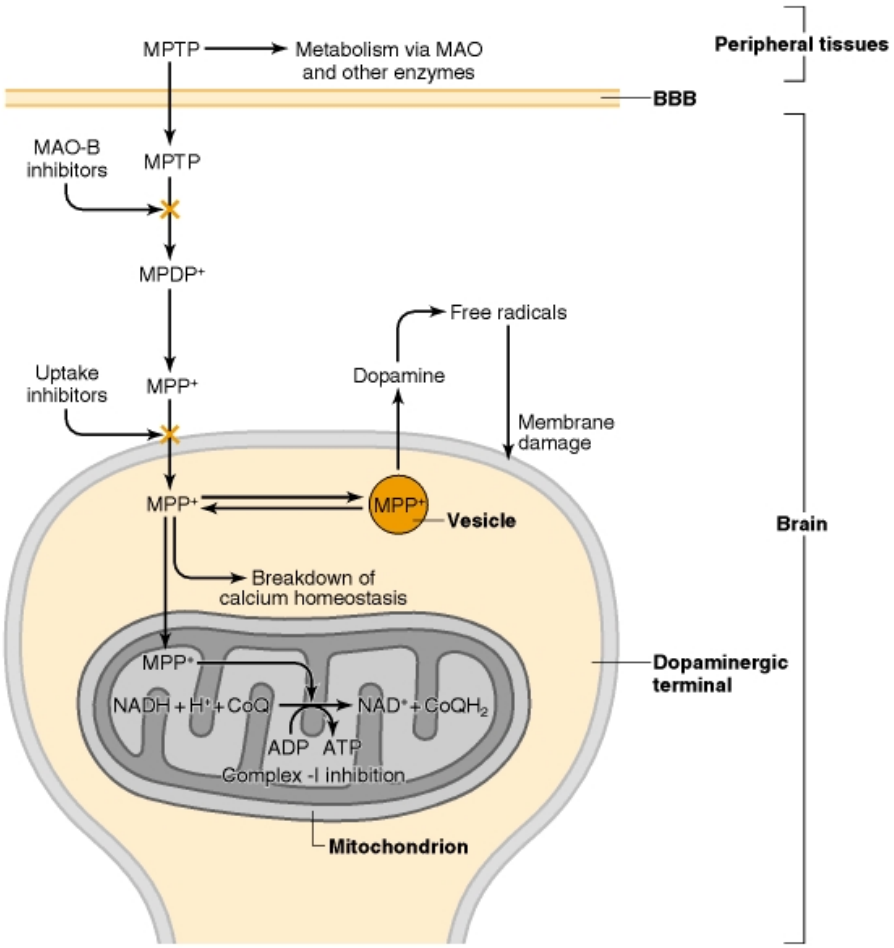

Figure 1. Schematic representation of the mechanisms involved in toxicity of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). *BBB,* blood—brain barrier; *MPDP*$^{+}$, 1-methyl-4-phenyl-2,3-dihydropyridinium; *MPTP,* 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine; *MPP*$^{+}$, its four-electron oxidation product 1-methyl-4-phenylpyridinium; monoamine oxidase (MAO). (Gerlach & Riederer, 1996)

Although it is believed that an inflammatory or oxidative component may lead to the cell death seen in the dopaminergic cells in the substantia nigra, a truly effective pharmacological treatment directed at this pathology has not been discovered. As seen on Figure 1 some success has been found using MAO inhibitors, but a Cochrane review of the use of non-steroidal anti-inflammatory drugs (NSAIDs) in the treatment of Parkinson's disease revealed no significant improvement (Rees et al., 2011). There is no current evidence for use of NSAIDs at this time for the treatment of Parkinson's disease.

Other pathological processes have been linked to cortical destruction. Elevated homocysteine levels in the brain have been linked with Parkinson's disease and other neurodegenerative disorders such as Alzheimer's and amyotrophic lateral sclerosis (Levin, Blatzel, Giese, Vogeser, & Lorenzl, 2010; Seshadri et al., 2002). Homocysteine elevation has also been correlated with other pathology such as vascular disease (Wald, Morris, & Wald, 2011). In Parkinson's disease these elevated levels of homocysteine are connected to cortical cell death and contribute to the production of Lewy bodies. The presence of these Lewy bodies in the basal ganglia is used to definitely diagnose Parkinson's disease post-mortem.

2.3.1 GENETICS

Although there has been significant advancement in understanding the role genetics plays in the development of Parkinson's disease, there is no *single* Parkinson's gene and it is not directly caused by a single genetic source. There are several genetic factors that have been discovered that are believed to be components in the development of Parkinson's disease. Genetic factors linked to toxin removal and its ability to oxidize proteins may contribute to an understanding of who develops Parkinson's disease in the future (Gwinn-Hardy, 2002; Hardy, Cookson, & Singleton, 2003). A longitudinal

evaluation of twins with late-onset Parkinson's disease via F-dopa and positron emission tomography (PET) suggest that genetics plays some role in the pre-symptomatic disease (Rakshi, et al., 1999).

Researchers are exploring the idea that loss of cells in other areas of the brain and body contribute to the symptoms characteristic of Parkinson's disease. Researchers have discovered that the hallmark sign of Parkinson's disease, Lewy bodies, are not only found within the substantia nigra, but also in the olfactory bulb, the hypothalamus, and the mesolimbic and mesocortical pathways (Forno, 1996; Jager, Hartog, & Bethlem, 1960; Lang & Lozano, 1998). They also present in the dorsal motor nucleus of the vagus, the nucleus basalis of Meynert, the locus ceruleus, the Edinger-Westphal nucleus in the midbrain, the raphe nuclei, cerebral cortex, and autonomic ganglia (Jager, Hartog, & Bethlem, 1960).

These areas of the brain correlate to non-motor functions such as sense of smell and sleep regulation. The presence of Lewy bodies in these areas helps explain some of the non-motor symptoms experienced by some people with Parkinson's disease before any motor sign of the disease appears (Jager, Hartog, & Bethlem, 1960).

Genetics helps in the understanding of the biology of the disease and may better guide future research by characterizing the protein products of genes associated with Parkinson's disease and the biochemical mechanisms affected. Five regions on chromosomes 5, 6, 8, 9, and 17 are associated with susceptibility to Parkinsonism (Hardy, Cookson, & Singleton, 2003). There has also been a link to genetic factors that may reduce the ability to rid the body of toxins and, it has been suggested, contribute to the oxidative process and cell destruction of the dopamine-producing cells. This supports the

connection to a neuro-oxidative pathology of Parkinson's disease. A detailed description of genetics is beyond the scope of this paper, however. Hardy et al. (2003) summarize the connection of genetics and its role in Parkinson's disease: "We can consider neurodegenerative disease the final outcome of a sequence of cellular events. At each stage the process is likely modified at the cellular level, due to genetic or environmental factors."

Although there is growing evidence that genetics plays a role in Parkinson's disease, the connection of the environment cannot be ignored. Twin studies substantiate the large role the environment may play as a contributing factor in the development of Parkinson's disease (Tanner, 2003). There is a higher incidence of Parkinson's in certain geographical areas, and a large incidence of toxic exposure in those with early-onset Parkinson's further supports the role the environment may play in the pathological process of Parkinson's disease.

2.4 CURRENT MEDICAL TREATMENT

Current medical treatment is focused on symptomatic improvement of the patient by balancing the excitatory and inhibitory stimuli from the basal ganglia to the supplementary motor cortex using medication and/or newer surgical procedures such as deep brain stimulation and fetal tissue transplantation (Farris, Ford, DeMarco, & Giroux, 2008; Goetz, Poewe, Rascol, & Sampaio, 2005; Guttman, Kish, & Furukawa, 2003; Liu et al., 2002; Ostergaard, Sunde, & Dupont, 2002; Rocchi, Chaiari, & Horak, 2002; Weaver, Stern, & Follett, 2006). Pharmacological treatment is effective by either stimulating more dopamine production or reducing the actions of the antagonistic cholinergic neurons (Jankovic & Stacy, 2007; Olanow & Jankovic, 2005).

2.4.1 MEDICATIONS

Medications are the first line of treatment for Parkinson's disease and are usually recommended once motor symptoms interfere with everyday life. There are three first-line drug classes: levodopa, dopamine agonists, and monoamine-oxidase-B inhibitors (e.g., rasagiline, selegiline). The monoamine-oxidase inhibitor medications support the oxidative principles described earlier in the oxidative stress model. Other pharmacological treatment classes include COMT inhibitors (entacapone and tolcapone), amantadine, and anticholoinergic agents (benztropine or trihexyphenidyl) (Baker et al., 2009; Fargel, Grobe, Oesterle, Hastedt, & Rupp, 2007; Goetz, Poewe, Rascol, & Sampaio, 2005; Jankovic & Stacy, 2007; Myllyla, Sotaniemi, Vuorinem, & Heinonen, 1992; Piccini et al., 1999; Talati, Reinhart, Baker, White, & Coleman, 2009).

Levodopa is still considered the gold standard for treatment of symptoms of Parkinson's disease. When levodopa eliminates or reduces the cardinal signs of Parkinson's disease (tremor, bradykinesia and rigidity, and postural instability), the clinical diagnosis may be confirmed. Levodopa is a precursor to dopamine that crosses the blood-brain barrier and binds to the dopaminergic receptors of the basal ganglia. Unfortunately, levodopa does nothing to stop the relentless destruction of dopaminergic cells in the substantia nigra. There are two possible negative consequences with levodopa use: one, levodopa may be toxic, and two, it may directly cause future motor complications (Perez et al., 2009). Levodopa is toxic to cultured dopamine neurons, and this may be a problem in Parkinson's disease where there is evidence of oxidative stress in the substantia nigra. However, there is little evidence to suggest that levodopa is toxic in vivo in Parkinson's disease (Jankovic & Stacy, 2007). Levodopa is associated with motor complications and dyskinesia. Increasing evidence suggests that they are related, at

least in part, to the short half-life of the drug and its potential to induce pulsatile stimulation of dopamine receptors rather than to specific properties of the molecule and that the motor complications may be a result of impacting the nonadrenergic receptors (Jankovic & Stacy, 2007). This imbalance may contribute to the dyskinesia.

Newer but cost-prohibitive pharmacological treatments include Apomorphine. This medicine is a fast-acting dopamine agonist used for treating occasional episodes of immobility associated with Parkinson's disease. Apomorphine can be injected under the skin when muscles become *stuck* or *frozen* and the patient is unable to rise from a chair or perform daily activities (Goetz, Poewe, Rascol, & Sampaio, 2005; Jankovic & Stacy, 2007; Jenkins, et al., 1992).

Continuous dopaminergic stimulation throughout 24 hours has shown to reduce motor complications resulting from prolonged levodopa use. This is thought to work by avoiding pulsatile stimulation of dopamine receptors. The dopamine agonist, Rotigotine, has been formulated in a transdermal delivery system that provides 24-hour stimulation (Watts et al., 2010). Parkinson's symptoms are equally reduced using Rotigotine with fewer side effects versus other dopamine agonists. Pramipexole and ropinirole are other drugs undergoing clinical trials (Pahwa et al., 2007).

2.4.2 DEEP BRAIN STIMULATION

The use of deep brain stimulation for the treatment of Parkinson's disease has shed light on the complexities of the brain and the areas affected by the disease. Deep brain stimulation of the subthalamic nucleus, globus pallidus, and thalamus continues to be performed and researched for the treatment of Parkinson's disease (Farris, Ford, DeMarco, & Giroux, 2008; Goetz, Poewe, Rascol, & Sampaio, 2005; Ostergaard, Sunde, & Dupont, 2002; Rocchi, Chaiari, & Horak, 2002; Weaver, Stern, & Follett, 2006).

Although this treatment was traditionally performed on the globus pallidus, there has been a shift to other areas of the brain. Stimulation of the part of the brain called the subthalamic nucleus has now been recognized as the most effective surgical treatment for Parkinson's disease. This addresses not only tremors, but also the full range of the disease's symptoms, including rigidity, slowness of movement, stiffness, and walking concerns (Ostergaard, Sunde, & Dupont, 2002). The surgery required to place a stimulator in the subthalamic nucleus is generally less complicated than surgeries for the thalamus or globus pallidus. Ostergaard (2002) did not report any adverse effects with deep brain stimulation of the subthalamic nucleus. The participants in the study were able to reduce their levodopa dosage by 19 percent and reduce off periods associated with levodopa use. This study had some subject attrition and, of the drop-outs, three were because patients refused to turn off their stimulator. This is suggestive of a reward of symptom reduction as a result of the stimulation.

People affected by involuntary movements such as dyskinesia often experience a marked reduction of these involuntary movements primarily because they are able to reduce their medications following deep brain stimulation surgery. Stimulating areas of the brain in some cases significantly reduced these involuntary movements (Ostergaard, Sunde, & Dupont, 2002). Deep brain stimulation of the globus pallidus seems to be somewhat less effective for problems with walking and balance, and patients remain on the same average dose of medications following surgery. Deep brain stimulation of the thalamus is only effective for tremor and rigidity and consequently, deep brain stimulation of the thalamus is no longer performed for patients with Parkinson's disease (Weaver, Stern, & Follett, 2006).

Liu et al. (2002) found the effects of deep brain stimulation could depend on the frequency of the applied stimuli. The study also showed that unilateral stimulation could have bilateral effects. The brain tissue was shown to be frequency-dependent; 100 Hz induced tremors while 130 Hz suppressed tremors. Lui et al. (2002) was able to correlate deep brain stimulation and local field potentials. This non-invasive measurement of local field potentials could allow for assessment and functional modulation of neural activity without the risks of deep brain stimulation (Sindou, 2001).

2.5 CURRENT THEORIES OF FUTURE MEDICAL TREATMENT FOR PARKINSON'S

Current theories regarding the treatment of Parkinson's disease are being directed at the pre-clinical destruction of cortical cells. The concepts of neuroprotection and neurorestoration, and the role of genetics are receiving greater attention in the literature (Gwinn-Hardy, 2002; Hardy, Cookson, & Singleton, 2003; Hattori, 2004; Hirsch, 2007; Hirsch & Farley, 2009; Liu, Nguyen, Hulleman, Li, & Rochet, 2008; Schapira, Cooper, Dexter, Clark, Jenner, & Marsden, 1990; Schapira, Holt, Sweeney, Harding, Jenner, & Marsden, 1990).

Neuroprotection refers to inverventions 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.

2.6 PARAMEDICAL TREATMENT

Other treatments for Parkinson's disease include physiotherapy, general exercise, relaxation, and occupational therapy (Keus, Bloem, Hendriks, Bredero-Cohen, & Munneke, 2007). Studies suggest that the priority for physical therapy interventions should be muscle strengthening and balance training in order to prevent falls (Ashburn,

Fazakarley, Ballinger, Pickering, McLellan, & Fitton, 2007; Koller, Glatt, Vetere-Overfield, & Hassanein, 1989; Verma & Pickett, 2001). Most of these therapies look at treating the symptoms of the disease or prevention of impairments from those symptoms, rather than its underlying cause (Fisher et al., 2008; Schaafsma, Giladi, Balash, Bartels, Gurevich, & Hausdorff, 2003).

The review of physical therapy in the treatment of Parkinson's disease literature is complicated by the large degree of variance in the types of intervention classified under the umbrella of physiotherapy. Interventions include gait re-education, improvement of balance and flexibility, and exercise to enhance aerobic capacity. Visual cueing, general exercise, and instruction are all employed to achieve goals of improved movement initiation, functional independence, and safety. Reviewing the effectiveness of physical therapy in the treatment of Parkinson's disease is also difficult, due to the many definitions of physical therapy. Differences in session length, group sizes versus individual sessions, and the different outcome measures used in the studies (Deane, Jones, Playford, Ben-Shlomo, & Clarke, 2001; Goede, Keus, Kwakkel, & Wagenaar, 2001; Iansek, 1998; Keus, Bloem, Hendriks, Bredero-Cohen, & Munneke, 2007; Koller, Glatt, Vetere-Overfield, & Hassanein, 1989) further complicate the ability to use literature to determine the effectiveness of physical therapy in the treatment of Parkinson's disease.

Two independent Cochrane reviews looking at physiotherapy as a treatment for Parkinson's disease (Deane, Jones, Playford, Ben-Shlomo, & Clarke, 2001b; 2001a) concluded there was not sufficient evidence for or against the use of physical therapy in the treatment of Parkinson's. These reviews highlight the lack of randomized controlled

trials in the care of such a complex, multi-causal, long-duration neurodegenerative disease like Parkinson's disease. Future literature would do well to define what physiotherapy treatment is, employ consistent outcome measures, and use more rigorous methodology and design.

Occupational therapy includes maintenance of work and family roles, improved transfers, and mobility. Therapy is directed at improving personal self-care activities such as eating, drinking, washing, and dressing. It includes environmental education and cognitive assessment. Studies looking at the effectiveness of occupational therapy are typically paired with physical therapy. Together they have been found to increase quality of life beyond the mobility gains made through physical therapy (Gage & Storey, 2004).

Speech and language therapists' goals for clients with Parkinson's disease are designed to prevent or minimize aspiration and improve speech. The therapy has been shown to be effective in improving loudness of speech and pitch. It improves the ability of a Parkinson's patient to make them understood when speaking (Fox, Ramig, Ciucci, Sapir, McFarland, & Farley, 2006; Whitehill & Wong, 2007).

A review of the literature in other movement therapies such as dance and Tai Chi has shown their effectiveness in the treatment of mobility of Parkinson's patients (Hackney & Earhart, 2008; Hackney, Kantorovich, Levin, & Earhart, 2007). Results of these studies, and the ease with which those with Parkinson's disease can participate in these movement therapies, provide insight into the complexity of brain activity required in coordinated movement. Visual cueing has been shown to improve gait, and this suggests that gait disturbances are a result of central nervous system changes (Lewis, Byblow, & Walt, 2000; Nieuwboer et al., 2007). Enhancing the function of the neural

connections in the central nervous system may bring about a change in mobility peripherally (Lewis, Byblow, & Walt, 2000).

Mobility in the Parkinson's patient is reduced by a number of factors, particularly in the later stages of the disease. Rigidity, bradykinesia, and postural instability all negatively impact gait and mobility performance. Canon and Rosenblueth (1949) showed that after denervation, the muscles supplied by the injured nerve became hypersensitive to acetylcholine, the neurotransmitter responsible for muscle contraction. They referred to this reduced threshold of contraction as supersensitivity. The supersensitivity can result from neuropathy or degeneration. In the motor system, the supersensitivity manifests as muscle banding and tension. A similar mechanism of spasticity is seen in other central nervous system injuries such as stroke and spinal cord injury. What is consistent is the impact on functional mobility as a result of central, but possibly also peripheral nervous tissue dysfunction.

Rigidity results in reduced postural flexibility and contributes to the Parkinsonian posture and reduced mobility. With denervation or motor disruption a resultant hypersensitivity to acetocholine in the peripheral muscles leads to inter-segmental stiffening. 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 and reduces their functional mobility.

2.7 OSTEOPATHIC LITERATURE ON PARKINSON'S DISEASE

Andrew Taylor Still (1910) discussed *shaking palsy,* which was described as a "shaking and tremor in muscles, accompanied by in-coordination" p137. Still refuted all theories on the causes of shaking palsy at the time, and reasoned that there was a mechanical cause. Still (1910) stated 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" (Still, 1910, p. 140).

2.8 OSTEOPATHIC STUDIES ON PARKINSON'S

A review of the available cranial osteopathic literature for the treatment of Parkinson's disease patients was completed. The author primarily used the internet and local libraries for research. Google, pubMed, cinahl, pedro, and ostmed were visited using *Parkinson's disease*, *cranial*, and *osteopathic treatment* as key words. The librarian and researcher at the A.T. Still library in Kirksville was contacted and she attempted to locate cranial and osteopathic treatment and Parkinson's disease sources. These web searches produced few studies on cranial treatment in Parkinson's patients. The few studies that were found did not outline a specific cranial treatment protocol, nor did they utilize specific techniques or methodology taught at the Canadian College of Osteopathy.

Wells et al. (1999) measured gait in people with mild to moderate Parkinson's disease and determined that after a single treatment of a standardized osteopathic manipulative treatment, there was a statistically significant increase in stride length, cadence, and velocity between the pre- and post-gait evaluation of the patients with Parkinson's. The possible mechanisms responsible for these improvements were not described. This study reinforces the need for further research into the role of cranial and other osteopathic treatment for mobility of Parkinson's patients.

Rivera-Marineza, Wells, & Capobianco (2002) suggest 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. This is consistent with the new paradigm in the medical literature directed at neuroprotection and neurorestoration. The

mechanisms of how the brain and human body compensate and protect themselves are still unknown. Rakshi et al. (1999) compared the dopamine uptake in the various parts of the brain and the ways they differ from early and late stage Parkinson's disease. The authors were able to show an increase in dopaminergic release in the anterior cingulate that they believe may be compensatory. They also showed a compensatory dopamine uptake by the serotonergic and non-andrenergic receptors in the dorsal midbrain and pontine regions of the brain, suggestive of compensatory mechanisms activating in the late stages rather than early stages of Parkinson's disease.

Other studies looking at the effectiveness of osteopathic manipulative treatment for Parkinson's disease showed some promise. One was set up as a single-blind placebo controlled study. There was no statistical significance; however, there was a trend towards being clinically significant (Snider et al., 2007). The applicability of their findings is limited as they did not include the specific osteopathic treatment given to the experimental group. Elster (2004) looked at 81 patients with multiple sclerosis and Parkinson's disease and the effects of upper cervical manipulation on subjective symptoms of the participants. Though there is no precise description of the *adjustment*, nor statistical analysis, subjective symptomatology was improved in 92 percent of the Parkinson's disease cases. Due to the limited research in cranial osteopathy and Parkinson's disease a case study was also included in the literature review. After twelve months of chiropractic craniovertebral manipulation the case study subject showed a 43 percent improvement in the unified Parkinson's disease rating score (UPDRS) as well as subjective improvement in sleep and range of motion and a reduction in rigidity and stiffness (Elster, 2000). Although no definitive conclusions can be drawn from a case

study, it supports the need for further investigation of cranial treatment for Parkinson's disease.

Comella, Stebbins, Brown-Toms, & Goetz (1994) found that the greatest effect of physical therapy was on patients with moderate Parkinson's disease. They suggest that this is most likely due to the ability of the brain to recruit dopamine from other areas earlier in the disease process. For example, the anterior cingulate in the frontal cortex secretes dopamine in early-stage Parkinson's disease and is no longer present in late stages of the disease (Lewis, Byblow, & Walt, 2000). Directly treating and improving the environment of the brain, the damaged basal ganglia, and their neighbouring structures resulted in an observable improvement in the execution of automated tasks such as gait. Osteopathic treatment effects could be neuro-restorative in nature to both the damaged basal ganglia and the nigro-striatal pathways. It may also improve the connections between the basal ganglia and other areas of the brain and may improve the compensatory capacity of the other areas o the brain like the anterior cingulate. There is a correlation between the degree of destruction and the severity of disease. Observable improvement from treatment could be a result of functional improvement of the basal ganglia or possibly could prevent any further cell destruction from occurring.

In the treatment of mobility and gait in those with Parkinson's disease, the effects of manual cranial therapy may not be only a result of improving the damaged dopamine and striatal pathway, but rather, an improvement may be the result of improving the neighbouring healthy tissue and other brain functions.

A full understanding is lacking of both the brain and the basal ganglia and its functions in Parkinson's disease. Not all Parkinson's patients present with the same

symptoms. Some have tremors while others may not. The variance in symptoms may be explained by the complexity of the basal ganglia themselves. The literature shows that stimulation of the brain has different effects. Although using only a small sample size (n=6), Rocchi et al. (2002) were able to show how levodopa and deep brain stimulation have differing effects on postural stability. They were able to show that combination therapy (deep brain stimulation and levodopa) work better together than either in isolation. No literature was found looking at the combined therapy of cranial osteopathy and medications, nor cranial therapy and exercise, but there is evidence that cranial manipulation can improve peripheral symptoms. Similar improvements in peripheral symptoms are achieved with deep brain stimulation.

Anecdotal evidence supporting craniosacral therapy as a treatment for Parkinson's disease is readily available on the Internet. These unpublished, non-peer-reviewed studies are poorly described in terms of methodology, treatments given, and controls. While it would be impossible to repeat these studies, they do succeed in stimulating discussion and thought. They show a descriptive improvement with video evidence (Rogers, 2009) of before and after for six Parkinson's patients.

2.9 CRANIAL THERAPY

In order to support the use of cranial osteopathic treatment for Parkinson's disease, a critical review of cranial assessment and treatment with its physiological effects and its use with other neurological disorders is helpful. The following review looked at not only the scientific evidence on osteopathic cranial therapy, but also the literature devoted to supporting the four tenets of cranial osteopathic therapy described by Magoun (1976) and Sutherland (1939). They describe four aspects required for a proper functioning cranial mechanism:

1. The articular mobility of the cranial bones
2. The inherent mobility of the brain and spinal cord
3. The fluctuation of cerebral spinal fluid
4. The mobility of intracranial and intraspinal membranes

Specific literature to substantiate the use of cranial therapy in the treatment of Parkinson's disease is limited. Since there is limited research in this area, there is also insufficient literature available to suggest discontinuing the use of cranial therapy in the treatment of Parkinson's disease. There are many case reports supporting the use of cranial osteopathic treatment. These are considered level 4 literature. Ideally, level 1 research should be used but in the absence of higher quality research, these case studies deserve consideration (Phillips et al., 2009). In the following literature review of cranial osteopathic treatment, there is sufficient evidence to support the principles that govern cranial therapy. The proposed therapeutic effects of cranial therapy based on these principles lend support to its use in the treatment of Parkinson's disease.

2.9.1 LIMITATIONS OF CRANIAL OSTEOPATHIC TREATMENT

Many of the studies supporting the use of craniosacral therapy as a treatment intervention have limitations to the research. One such limitation is the poor inter- and intra-rater reliability of cranial osteopathy (Moran & Gibbons, 2001; Rogers, Witt, Gross, Hacke, & Genova, 1998; Wirth-Pattullo & Hayes, 1994). These authors showed that practitioners were unable to accurately and consistently palpate the rhythm described by other osteopathic authors. A reliable method of evaluation is required to determine the validity of therapeutic interventions. The human interaction between patient and therapist continues to complicate the ability to measure small sub-clinical changes (Green, Martin, Bassett, & Kazanjian, 1999). As imaging capable evaluating cranial perfusion come

down in price, there will hopefully be more objective measurable effects to support the improvements seen by patients and therapists.

The Province of British Columbia did a systematic review of craniosacral therapy and concluded there was insufficient evidence to support the use of this treatment modality (Green, Martin, Bassett, & Kazanjian, 1999). The authors were unable to find sufficient evidence from primary literature to support the use of cranial osteopathic treatment as an effective treatment modality. They were also unable to find sufficient evidence to negate its use. In the absence of high-quality literature, clinical experience, case studies and other lower-quality research should be incorporated into an evidence-based practice.

Another limitation of cranial research arises from its origin. The original description of cranial lesions and their treatment by means of cranial manipulation were were based on clinical observations. Original studies in cranial therapy lacked the sensitive measuring devices and outcome measures needed for today's research standards. The studies describe movement of the bones with little or no scientific data to corroborate these findings. Sutherland (1939) and Magoun (1976) describe in great detail the movement of the bones and their effects on health, but the measurement of these movements was based on palpation and anecdotal evidence. One study measured client satisfaction: 74 percent of patients participating in the study *reported a valuable improvement* (Harrison & Page, 2011). Upledger (2007) describes the effects of the craniosacral mechanism but the studies do not stand up to critical evaluation. The studies have many limitations. These include difficulty to reproduce due to incomplete or absent methodology. Upledger's study entitled *Mechano-electric patterns during craniosacral*

osteopathic diagnosis and treatment concludes that there is a measurable effect on other parts of the body that match the subjective impression of changes in the craniosacral mechanism. He used electromyography (EMG) studies in the lower extremities, electrocardiograms (ECG), and bioelectric activity to correlate with what the osteopath felt. This study does not state how many subjects were tested, nor does it describe the type of intervention being performed. There is neither statistical analysis to determine whether these measureable changes are statistically significant nor descriptions of how the subjects or the evaluators were blinded. No significant conclusions can be drawn from this type of literature.

2.10 THE FOUR PRINCIPLES OF CRANIAL OSTEOPATHIC THERAPY

If cranial osteopathic therapy is broken down into subunits, better literature is available to support the foundation upon which osteopathic cranial therapy is based.

2.10.1 THE ARTICULAR MOBILITY OF THE CRANIAL BONES

Several studies have shown that there is movement between cranial bones (Downey, Barbano, Kapur-Wadhwa, Sciote, Siegel, & Mooney, 2006; Heisey & Adams, 1993; Hubbard, Melvin, & Barodawala, 1971; Kostopoulos & Keramidas, 1992). Literature is available showing that sutural movement does exist and that this cranial movement coincides with global physiological changes. To show the existence of sutural movement in the living, Heifetz and Weiss (1981) used two comatose patients to measure skull expansion with increased intracranial pressure. They were able to detect small movements at the cranial sutures. Adams, Heisey, Smith, and Briner (1992) used invasive measures at the skull including a 20 gauge needle inserted into the lateral ventricle and a cannula inserted directly into the femoral artery, and also measured distance changes between two needles affixed to the parietal bones. This study used external forces to

produce cranial bone movement. The results of this study were more impressive than prior studies looking at cranial movement, and the authors believe it was a result of how they measured the movement. It differed from prior cranial movement studies in that it looked at units of distance rather than units of force. The Adams et al. (1992) study used units of distance to show that movement at the parietal bones existed with both external and internal cranial force changes; they used two microfoil strain gauges attached directly to bone and not skin. Lastly, Greenman and McPartland (1970) were able to correlate roentgen findings with clinical cranial findings in seven patients, while three were in disagreement. Frymann (1971) made electronic tracings of cranial bone motion but the study was not well described and therefore assessing the quality is difficult. Heisey and Adams (1993) 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.

2.10.2 THE INHERENT MOBILITY OF THE BRAIN AND SPINAL CORD

The movement of the brain can be measured using cine and echo-planar MR imaging (Poncelet, Wedeen, Weisskoff, & Cohen, 1992). Researchers found the motion of the brain occurred primarily in cephalocaudal and lateral directions. Pulsations of the brain parenchyma occurred in conjunction with the cardiac cycle and there was rapid brain motion during systole with a slow diastolic recovery. The rate of movement found in this study differs from the primary respiratory rhythm (PRM) described by osteopathic practitioners. PRM is described at a slower rate with an equal phase length of motion, and recovery is described as flexion and extension or inhalation and exhalation phase. The velocity was measured in different areas of the brain and in specific locations: 2mm/sec in the brainstem and 1.5mm/sec in the central thalami – differing again from the global

expansion and contraction described in osteopathic cranial therapy (Poncelet, Wedeen, Weisskoff, & Cohen, 1992). Maier, Hardy, and Jolesz (1994) used real time magnetic resonance imagining (MRI) 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 recoil that appeared slower, consistent with Poncelet's findings.

Greitz et al. (1992) used MR imaging to measure velocity and movement of the brain, and 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. At the brainstem it was 1.5 mm per second. The researchers concluded that the encephalon moves due to arterial expansion, which in turn causes expansion of the brain that compresses the ventricular system, pushing cerebrospinal fluid into the spinal cord.

NASA looked at the effects of 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, Ballard, Shuer, Yost, Cantrell, & Hargens, 1998). This device was used in a study with six healthy volunteers placed in various tilt positions; 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).

2.10.3 THE FLUCTUATION OF CEREBROSPINAL FLUID

Studies support the phenomenon of cerebrospinal fluid movement and the fact that the cranial pulse or rhythm is different from the cardiac and respiratory rate. Studies supporting this phenomenon are measurable by encephalogram, magnetic resonance

imaging, and intracranial and intraspinal pressure monitoring. Li, He, Yao, and Wen (1996) used an observational study to determine the connection between cerebrospinal pulse waveforms and intra-cranial pressure. Feinberg and Mark (1987), observing 25 healthy subjects and 5 patients using magnetic resonance imaging, showed reproducible magnitudes and directions of CSF flow. Moskalenko (1980) showed a connection between the fluctuation of cerebrospinal fluid and the dynamics of blood in dogs. Using a Plethysmograph, the researchers measured the blood volume and cerebrospinal fluid within the cranium, as well as the intracranial pressure. Results showed that 10 percent of the total cerebrospinal fluid shifts from the cerebrum to the spinal cavity with every exhalation, returning on inspiration. The flow of the cerebral spinal fluid is shown in Figure 2.

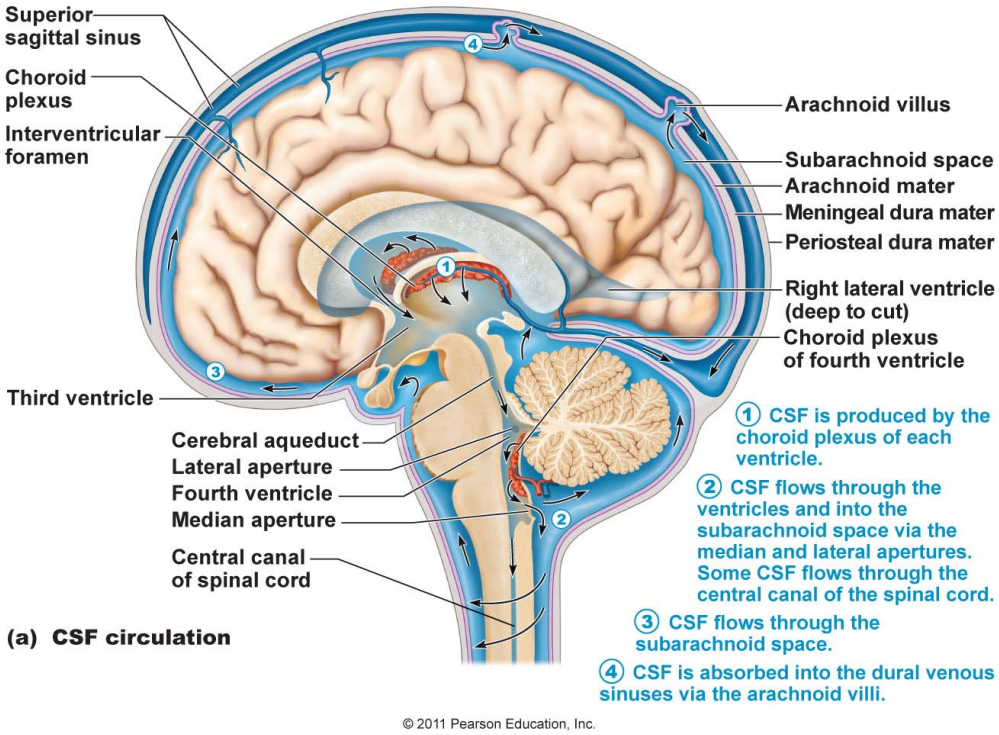

Figure 2. Flow of Cerebrospinal Fluid: (Marieb & Hoehn, 2011)

These studies provide evidence that the primary respiratory mechanism in the cranium may be a combination of, arterial pressure and the pressures created by inspiration and expiration.

2.10.4 THE MOBILITY OF INTRACRANIAL AND INTRASPINAL MEMBRANES

Pia, arachnoid, and dura make up the protective membranes of the brain and spinal cord. Sutherland (1939) wrote that these structures attach to all bones of the neurocranium and are responsible for their movement. Evidence is available to support the attachment of the dura to the upper cervical vertebrae and posterior cranial fossa (Mitchell, Humphreys, & O'Sullivan, 1998). There is a suggestion of connective tissue crosslinks between the dura and the upper cervical vertebrae. Upon dissection it appears that the dura is a component in limiting the motion of the cervical spine, particularly in rotation (Bashline, Bilott, & Ellis, 1996; van Dun & Girardin, 2006).

Studies show that alterations in the attachments of the dura to the cranium and vertebral column may impact symptomatic pathology. Levy et al. (1988) showed that cord motion was decreased and pulsative velocity was reduced in subjects with tethering or cord compression compared to normal control subjects. On dissection, Bashline (1996) was able to conclude that variations of attachments of the meningovertebral ligaments appear to impact disc herniation, degeneration, and possibly symptomatic relationships.

Kostopoulos and Keramidas (1992) looked at the impact external cranial pressure had on intracranial membranes in an embalmed cadaver with the brain removed. They measured the change in distance of the membranes with different osteopathic cranial techniques. They found statistically significant change with the frontal and parietal lift. Smaller changes in distance were noted with sphenobasilar compression and decompression. The limitation of this study is that with the brain removed the impact on

the membranes was measured without the brain, and it may differ significantly from the impact on the membranes with the brain and dura still present. Nonetheless, the evidence in this study supports the attachments of the dural membranes to the cranium and the impact on the dural membranes by osteopathic cranial techniques.

2.10.5 CRANIAL THERAPY IN THE TREATMENT OF NEUROLOGICAL DISEASE

Physiological changes and the use of cranial therapy as a treatment modality have been shown in the literature. Frymann (1998) believed that impairment of physiological motion of the cranium and a disturbance of the inherent mobility could lead to the manifestation of progressive neurologic dysfunction within the central nervous system. Frymann (1998) observed that children who had no previously recognized neurological inadequacies demonstrated improvement in their academic performance following an osteopathic treatment program. This would lend merit to cranial treatment of Parkinson's patients at the onset of the earliest signs or even prior to symptoms in a prophylactic manner if possible. Still (1910) discussed the treatment and clinical presentation he called *shaking palsy* (Still, 1910)(p. 140). These thoughts are substantiated by current theories in neurodegenerative diseases related to a reduction in perfusion to the brain seen on more sensitive cranial imaging tests. Magoun (1976) describes a mechanical treatment for the cranium directed towards providing a proper supply of nutrients to the brain and states that "dural tension, therefore, presents a greater portent for circulatory stasis and toxic reaction, congestion and dysfunction" (p. 198). Although this is not primary source literature, it does show others are postulating similar hopes for cranial therapy and the treatment of disease. Hypotheses are borne out of clinical practice.

Cranial therapy has been shown to be effective in the treatment of other brain or central nervous system disorders (Duncan, 2007; Frymann, 1976a, 1976b; Frymann,

Carney, & Springall, 1992; Greenman & McPartland, 1995; Magoun, 1976; Moskalenko et al., 2001; Woods, 1973). Greenman and McPartland (1995) used a retrospective case series study to look at the cranial strain patterns of those with traumatic brain injury seen in an outpatient therapy department. An experienced osteopathic physician recorded the cranial rhythm interval and found that both amplitude and rate were decreased with traumatic brain injury when compared to normals. Lateral and vertical strain patterns were also more prevalent when compared to the healthy population. These strains are considered to be non-physiologic lesions and are considered to have a more serious impact on the health of the patient. They are more commonly a result of trauma. Woods and Woods (1961) found a similar pattern in those patients with psychosis.

2.10.6 PHYSIOLOGICAL EFFECTS OF CRANIAL THERAPY IN PARKINSON'S DISEASE

There is limited evidence in the literature of cranial osteopathy in the treatment of Parkinson's disease. There is insufficient evidence for, or against, its use. However, there is evidence to support the belief that the four principles of the craniosacral mechanism can have a positive effect as a neuroprotective treatment. As the research into the pathology of Parkinson's disease and the possible oxidative factors contributing to the pathology of Parkinson's disease increase, the need for neuroprotective treatment will grow. As measuring devices become more sensitive and pick up small changes in perfusion, the quality of studies supporting or negating the use of cranial therapy will increase.

Understanding the specific physiological changes that result in the brain with cranial osteopathic therapy will help substantiate or refute its use in the prevention of cell death and thus clinically in the treatment of neurodegenerative disorders such as Parkinson's disease. If an anti-oxidative response or perfusion improvement is possible

with osteopathic treatment there could be a neuroprotective role for its use in Parkinson's disease. A loss in perfusion correlates with destruction of the dopaminergic cells in the substantia nigra and the motor symptoms of Parkinson's disease (Zhang et al., 2010). As an example, first-line medications are being used that address early oxidation believed to contribute to the neurodegenerative process. These are currently part of the pharmacological treatment of Parkinson's disease. Monoamine inhibitors are directed at minimizing the damage as a result of oxidation. Anti-inflammatory medications are being researched in the treatment of Parkinson's disease, as there is evidence of an inflammatory component, shown by the presence of cytokines. The effect of altered arterial flow to, and venous drainage from, the cranium has been shown to impact neurodegenerative disorders. More recently, the use of vascular surgery to improve venous drainage from the cranium is receiving a great deal of attention, and Health Canada has recently announced that they will be doing a study to determine its efficacy (Health Canada, 2011).

The symptoms of Parkinson's disease, and its diagnosis, occur long after the destruction of dopaminergic cells has taken place. By better assessing who may be susceptible, or who may already be in the process of cell destruction, researchers will be able to focus their treatments on the underlying causes of the cell destruction occurring in the basal ganglia and other locations in the brain. By assessing and treating the cause of the cell death it is hoped that Parkinson's disease can be slowed, halted, or cured. The physiological effects of improving cellular perfusion, improving blood supply and removing toxins removal will cause the health of the target tissues to be improved. If the

target tissues are the basal ganglia and the brain, its improved health should minimize cell destruction, slow the disease progression, and improve symptoms.

2.10.7 SUMMARY OF CRANIAL THERAPY

There is a large void in the literature concerning general osteopathic care and, in particular, cranial osteopathic treatment for those individuals suffering with Parkinson's disease. This pilot study hopes to lay a foundation of the effects of cranial treatment on mobility in those afflicted with Parkinson's disease. Applying the principles of osteopathy to the known location of pathology (the brain) and the hypothesized reasons for that pathology, including toxicity and vascular insufficiency, will result in a global effect and a measurable improvement in mobility. The hope is that this research may lead to an improvement in the quality of life of those individuals, and add to the body of knowledge on which future research may be built.

The supporting evidence for the movement of cranial bones, the rhythmical fluctuation of cerebrospinal fluid, and the dural attachments in the cranium and sacrum provide the physiological and anatomical evidence to support the mechanics behind cranial osteopathic treatment. Current evidence and case study evidence exists supporting the use of cranial therapy in the treatment of Parkinson's disease, but there is insufficient literature to support either its use or its non-use. Further research is needed in this area.

2.11 SUMMARY OF LITERATURE REVIEW

Parkinson's disease is a prevalent neurodegenerative disorder affecting the functional mobility of a growing number of Canadians. There is no known etiology and current treatment of the disease remains limited. Presently there is no known cure.

Parkinson's disease is characterized by tremor, rigidity, bradykinesia, and postural instability affecting the functional mobility of its sufferers. The symptoms of Parkinson's

disease primarily result from the cell death of the dopaminergic neurons in the substantia nigra. However, newer imaging techniques now make it possible to detect minor perfusion changes in the brain associated with neuronal loss in other areas of the brain in addition to that in the basal ganglia. Parkinson's disease is now known to be more diffuse in nature than previously thought. This more diffuse neuronal cell death better explains the more diffuse nature of symptoms associated with Parkinson's disease such as depression, and olfactory and digestive alterations, and points to a more global approach to treatment.

The cause of the cell death is not fully understood but is thought to include environmental factors, oxidative stress, and genetic predispositions which are presumed to create a toxic environment leading to the neuronal loss. This concept of toxicity and slow destruction as a result of a suboptimal environment lends credence to the theories of osteopathy. Cranial osteopathic treatment is thought to improve the vitality and health of the whole patient. Cranial osteopathic treatment is a safe, non-invasive, and well-received treatment that if shown to be beneficial would be a useful adjunct treatment for those with Parkinson's disease.

Current medical treatment of Parkinson's disease is effective in temporarily curbing symptoms of tremor, bradykinesia, and rigidity. Deep brain stimulation for younger patients and dopamine replacement pharmacological treatment are the primary methods of treatment. Unfortunately there are side effects to pharmaceutical treatment, and none have been proven to slow the progression of the disease. Deep brain stimulation is a surgical procedure that has shown some dramatic improvements. However, this

surgery is available only to younger candidates with severe motor fluctuations, and has its risks.

Physical therapy is a common treatment provided to Parkinson's disease patients either before or in addition to pharmaceutical intervention. Studies have indicated various results on the treatment of gait and functional mobility in Parkinson's disease.

There is very limited research directly supporting the use of cranial osteopathic treatment for Parkinson's disease. Four studies looking at osteopathy and Parkinson's disease were reviewed, showing promising results. The use of cranial osteopathy in treating other neurological conditions such as traumatic brain injuries and learning disabilities were reviewed to add support to its use. It is a non-invasive, well-received treatment with no known side effects. Research on the efficacy of cranial osteopathy is also limited. Current literature is able to support the principles of cranial osteopathy, but its therapeutic effects in the treatment of Parkinson's disease are neither supported nor negated. The mechanical principles of cranial osteopathic treatment are supported in the literature, and with newer imaging techniques research in this area should become more valuable.

3 CHAPTER THREE: OSTEOPATHIC JUSTIFICATION

Parkinson's disease is a complex neurodegenerative disorder with no known cure. The substantia nigra undergoes a long, slow destruction thought to occur over many years. The resulting loss of dopaminergic function in the substantia nigra is the primary cause of the symptoms associated with Parkinson's disease. The pathology process responsible for this dopaminergic destruction may result from toxic exposure, ischemic trauma, inflammatory processes, deposition of proteins responsible for the formation of Lewy bodies, and/or a number of other factors.

Osteopathy is ideally positioned to treat complex neurodegenerative disorders due to the holistic approach of osteopathic care. Andrew Taylor Still first described osteopathy in 1910; he believed that the patient's body, mind, and spirit were most aptly designed to promote one's own health (Still, 1986). By removing restrictions or blockages in the body, whether they are joint, fluid, or neural restrictions, the osteopath is able to free the patient's body's own healing capacity. This in turn promotes healing or restoration of health, and prevents further disease. In the case of Parkinson's disease the primary area in which cell destruction takes place is the substantia nigra of the basal ganglia in the brainstem. Improving the mobility and health of the cranial structures related to the basal ganglia and its surrounding tissues would put the Parkinson's patient's body in the best position to heal these damaged areas, promoting compensatory mechanisms and preventing further destruction.

The concept of cranial movement was discussed by Still (1986), advanced by Sutherland (1939), and documented by Magoun (1976). Several authors discuss the impacts of immobility of the cranium, locations of compromise, and their resultant impacts on health (Magoun, 1976; Sutherland & Wales, 1998; Upledger & Vredegoogd,

1983). Sutherland (1939) hypothesized that the cranial lesions or restrictions in cranial movement would impact the health and function of the brain and brainstem, as well as the vessels entering and exiting the brain, and thus the health of the whole person. This study will potentially improve the functional mobility of Parkinson's patients by applying the principles of osteopathy to the areas directly and indirectly related to the basal ganglia.

The four principles of osteopathy are discussed below in relation to improving the health and function of the Parkinsonian patient.

3.1 THE PRINCIPLE OF THE RULE OF THE ARTERY IS ABSOLUTE

For living tissues, muscles, nerves, or cells to function properly they must receive ample nutrition and have appropriate waste removal. Proper arterial flow, venous return, lymphatic drainage, and cerebrospinal fluid are required to maintain health. Proper cellular perfusion requires optimal venous drainage for adequate arterial supply to occur. Thus, all fluid flow must be working optimally to maintain optimal health. This is *the rule of the artery* (Still, 1910).

The primary pathological process in Parkinson's disease is the destruction of the dopaminergic cells in the basal ganglia, or more specifically, the substantia nigra. The pathological destruction of the substantia nigra is believed to occur very slowly over many years (Adler & Ahlskog, 2000). It has been shown that a loss or reduction in blood supply to the brain can lead to cell death (Lazzaro, Chen, Christoforidis, & Mohammad, 2011; Lee et al., 2009), cause inflammation (Luchtman, Shao, & Song, 2009; McGeer & McGeer, 2004; Qian, Flood, & Hong, 2010), and increase iron content (Haacke et al., 2009; Zhang, et al., 2010). This slow destruction and cell death as a result of poor environment support the current theories about the pathological process responsible for

Parkinson's disease and support the principle of the rule of the artery in the treatment of Parkinson's disease.

The brain requires a large vascular supply. Numerous areas of the brain must be activated in order for coordinated movement, required for gait and balance, to occur properly. Although the loss of dopamine in the substantia nigra and basal ganglia are the primary reasons for the loss of coordinated movement in Parkinson's disease, other areas of the brain must be adequately supplied by nutrients. The compensatory mechanisms available in the human body and brain and the development of collateral compensatory vascular supply provides support for a treatment approach encompassing all the vascularity to the brain rather than confining treatment to the brainstem. In addition, the complex nature of Parkinson's disease and the diffuse nature of its symptoms supports the likelihood of complex neuronal cell destruction. The connections and integrations in the brain as well as the ability to develop compensatory pathways would indicate a need to look at the blood supply of the whole brain and not only the basal ganglia.

By improving fluid flow to and from the brain and brainstem, cranial osteopathic treatments may put the Parkinson's patient's tissues in a better position to reduce inflammation, remove toxins, and restore health. It is known that the incidence of Parkinson's disease increases with age. This correlates with the normal reduction in vascular supply that occurs with normal aging, which is accompanied by a reduction in brain tissue volume (Folstein & Folstein, 2010; Ge, Grossman, Babb, Rabin, Mannon, & Kolson, 2002; Meltzer et al., 2000; Moskalenko, 1980). In order to understand how cranial osteopathy can impact Parkinson's disease the anatomy of the arterial, venous, and lymphatic system must be well understood.

3.1.1 ARTERIAL SUPPLY TO THE BRAIN AND BASAL GANGLIA

The arterial blood flow to the brain comes from two pairs of arteries: the paired carotid arteries and the vertebral arteries. Eighty percent of cranial blood supply is believed to come from the carotid system and the vertebral arteries supply the remaining twenty percent. The brain is one of the most metabolically active organs in the body, as it receives approximately 17 percent of the cardiac output and uses 20 percent of the body's oxygen (Krakow, Ries, Daffertshofer, & Hennerici, 2000; Lee, et al., 2009). These statistics are indicative of the nutrient needs of the brain. Figure 3 illustrates the complexity of the arteries passing through the neck to supply the brain and Figure 4 is a close up of the circle of Willis.

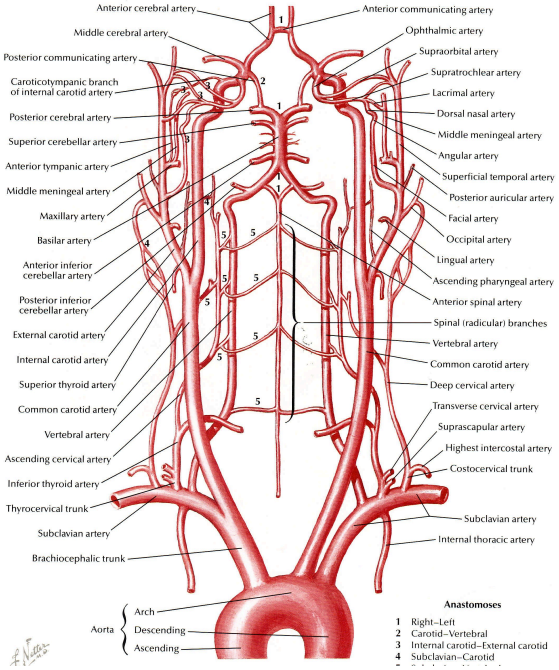

Figure 3. Arteries to the brain (Netter, 1989. Plate 131)

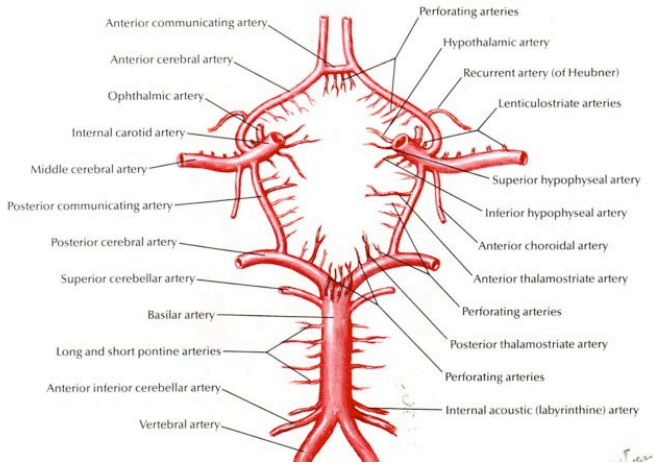

Figure 4. Circle of Willis (Netter, 1989. Plate 133)

The vertebral arteries originate from the subclavian arteries and travel within the transverse foramen, beginning at the sixth cervical vertebra. At the second cervical

vertebra the vertebral artery moves laterally and upward to the transverse foramen of the first cervical vertebra. It then loops posteriorly along the articular process of the first cervical vertebra in a deep groove, passes beneath the atlanto-occipital ligament, and enters the foramen magnum, where the anastomosis of the left and right vertebral arteries form the basilar artery at the base of the pons and become part of the circle of Willis (Figure 4, Figure 5, and Figure 6). The circle of Willis is an arterial loop consisting of the anterior cerebral, the internal carotid, and the posterior cerebral arteries located at the base of the brain (Figure 5 and Figure 6). Some of the blood supply to the basal ganglia originates from the circle of Willis. The posterior cerebral artery, which extends from the basilar artery, supplies the mid-brain, including the substantia nigra (Fix, 2009).

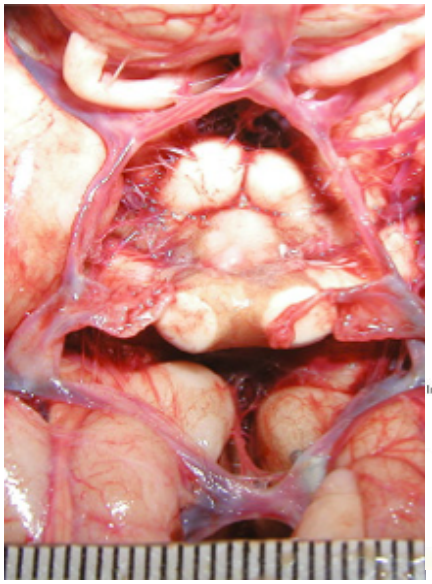

Figure 5. Normal cerebral arterial circle. The metric gauge placed on the plane of the vessels is shown in the bottom of the image. This meter has been used for the measurement of the diameters (Eftekhar, Dadmehr, Ansari, Ghodsi, Nazparvar, & Ketabchi, 2006, p.1203)

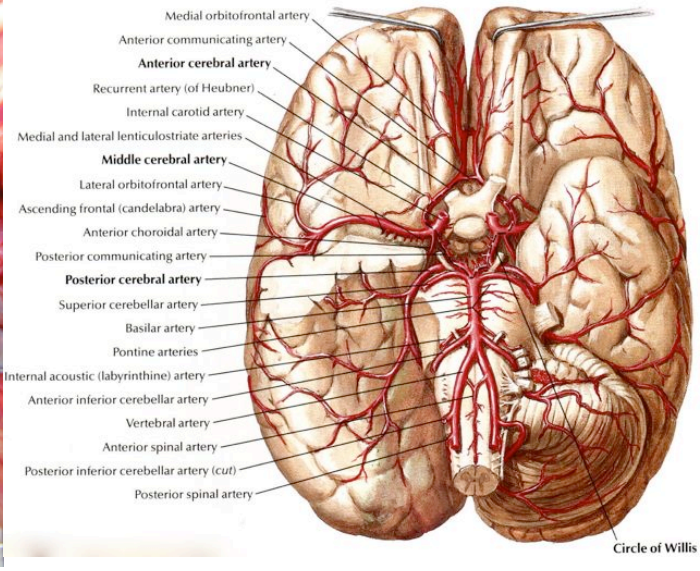

Figure 6. Inferior view: Arterial supply to the brain (Netter, 1989. Plate 132)

The path of the vertebral artery in the upper cervical region is tenuous and susceptible to compromise in this area by aberrant movement, sustained end range positions**,** and instability. The path of the vertebral artery can be seen in Figure 7 and Figure 8. Also note the close proximity of the nerve ganglion. Although this is considered to be the most common path of the vertebral artery, there is great variability in the arterial path of the vertebral arteries (Wang, Cai, Liu, & Wang, 2009), and therefore it is possible that different lesions in different Parkinson's patients may affect the flow in different ways. Somatic dysfunctions in the craniovertebral region due to instability and dysfunction can affect the flow of the vertebral arteries and thus affect the blood flow to the spinal cord, brainstem**,** and brain (Yamazaki, Okawa, Hashimoto, Aiba, Someya, & Koda, 2008). Gross pathology to the craniovertebral region has been shown to be relevant to peripheral neurological symptoms. Goel and Sharma (Goel & Sharma, 2004)were able to improve neurological symptoms in all of their surgical patients. Osteopathic treatment is believed to improve minor alterations of the joints and fascia in this area and optimize vascular supply. *The Osteopath seeks first physiological perfection of form, by normally adjusting the osseous frame work, so that all arteries may deliver blood to nourish and construct all parts* (Still, 1910, pg 28).

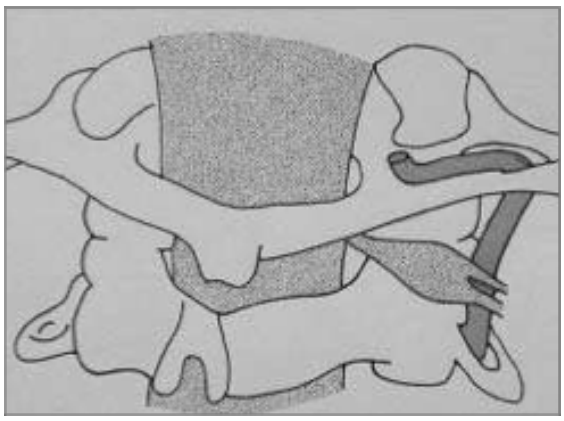

Figure 7. Line drawing showing the relation of second cervical ganglion to the vertebral artery and atlantoaxial facet joint (Goel, Sharma, Dange, & Kulkarni, 2005, p.527)

Figure 8. Cadaveric dissection of an injected specimen showing the relationship of the vertebral artery and the C2 ganglion to the atlantoaxial joint (Goel, Sharma, Dange, & Kulkarni, 2005, p.527)

The basal ganglia and substantia nigra's blood supply come from the cerebral arteries originating from the internal carotid system (Figure 3 and Figure 4). The internal carotid artery enters the cranium through the foramen rotundum in the cranial case and meets the vertebral arteries, which enter the cranium at the foramen magnum as part of the circle of Willis. The internal carotid arteries bifurcate into the middle and anterior cerebral arteries. Magoun (1976) described the importance of using cranial osteopathy to eliminate circulatory stasis in the brain, and this concept has been supported by more recent literature. Moskalenko's (1980) research was able to show that structural impedance improves circulatory flow. He also found that the movement of cranial bones had an effect on vascular circulation in the brain.

The anterior choroidal artery, the primary supplier of the basal ganglia, typically originates from the internal carotid, but has been shown to branch off the middle cerebral artery (Nolte, 1981). It 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). Note (Figure 9) that the posterior cerebral artery, which in turn is supplied primarily by the internal carotid, supplies the substantia nigra.

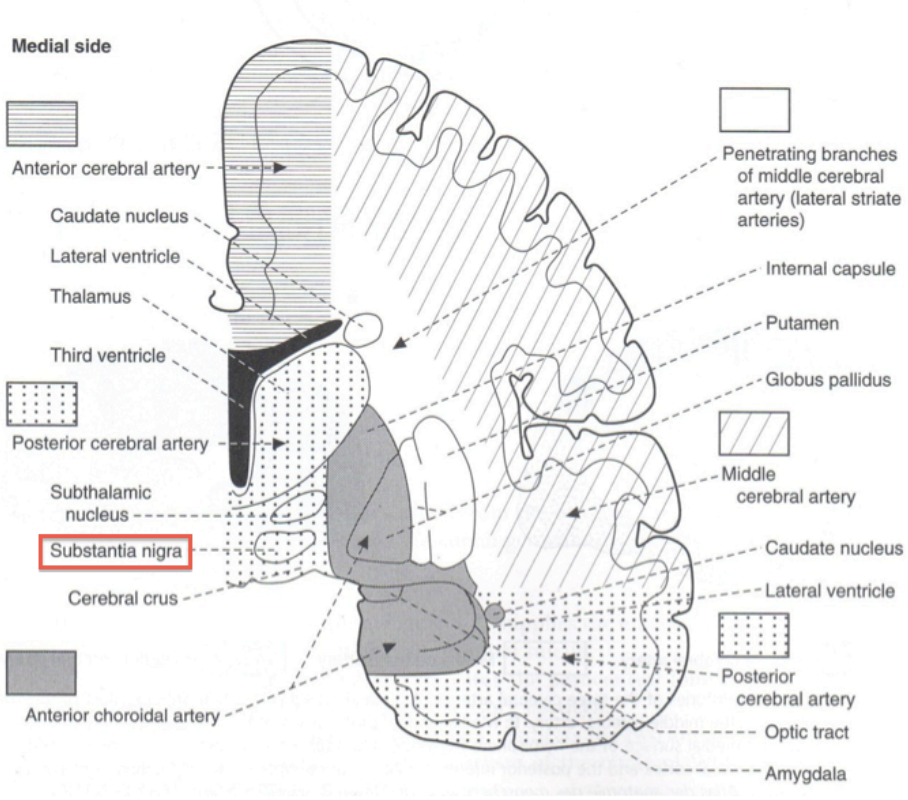

Figure 9. Coronal section through cerebral hemisphere at internal capsule and thalamus, showing distribution of arterial supply (Fix, 2009, p.40).

3.1.2 OSTEOPATHIC LESIONS RELEVANT TO THE RULE OF THE ARTERY

The osteopathic principle of the rule 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 (Magoun, 1976). This principle is of utmost importance as it applies to the brain. Because the brain requires more oxygen and glucose than any other organ, it may be more sensitive to vascular insufficiency. This could result in neural cell necrosis, causing brain damage and serious functional sequelae (Nolte, 1981). Cranial osteopathy may have an effect on the arterial supply, venous drainage, and flow of cerebrospinal

fluid within the cranium. Maximizing motion of the occiput, petrous portion of the temporal bones using cranial osteopathy in Parkinson's patients can potentially increase blood flow to the affected basal ganglia (Magoun, 1976; Moskalenko, 1980). Removing fascial tension through which the vessels of the basal ganglia flow will reduce the tension on the vessels and thus reduce the vascular impedance.

There is evidence to suggest that somatic dysfunctions in the cervical spine can alter the blood flow of the vertebral artery. For example, a lesion of the first or second cervical vertebra or a malposition of the occipital condyles can lead to strain on the vertebral artery in its most tenuous pathways, before it enters the foramen magnum (Bonati, Wetzel, Kessel-Schaefer, Buser, Lyrer, & Engelter, 2010; Licht, Christensen, Svendsen, & Hoilund-Carlsen, 1999; Mitchell, 2009; Mitchell & Kramschuster, 2008). Cervical position such as sustained end range rotation of the cervical spine can result in a reduction of blood flow through the vertebral arteries (Goel, Sharma, Dange, & Kulkarni, 2005; Licht, Christensen, Svendsen, & Hoilund-Carlsen, 1999). Further, cervical degeneration can alter the vascular function of the vertebral arteries as they ascend the cervical spine and pass through the upper cervical spine. Instability caused by trauma, rheumatoid arthritis, or boney and ligamentous malformation and malpositioning of the atlas, axis**,** or occiput. Instability can impact the neighbouring neural structures and vertebral arteries and thus can lead to spinal cord and brainstem damage (Goel, Sharma, Dange, & Kulkarni, 2005; Vetti et al., 2010).

There is a great deal of collateral circulation in the brain due to the vast demands on the brain and the importance of the structures. 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 (Fix, 2009).

The rule of the artery is one way to understand the pathological process of Parkinson's disease. If adequate perfusion of the basal ganglia and substantia nigra could be maintained, the cell death that occurs in Parkinson's disease might be reduced or eliminated. Conversely, if there is toxic exposure to the basal ganglia or if there is an injury to the area involving inflammation, a good vascular system is essential for the Parkinson's, or pre-Parkinson's patient. Improving fluidic supply and fluid turnover may be neuroprotective. Optimal blood supply is needed to reduce inflammation and promote healing. A reduction in blood flow can lead to an ischemic insult causing inflammation, tissue injury, and eventually cell death; however, early loss of blood flow may not always produce symptoms immediately. Maintaining ample blood flow and maximizing neuroprotective therapies are new and important approaches in treatment of Parkinson's disease. Parkinson's disease inflammation and perfusion loss are thought to occur years before true symptoms appear. There may therefore be a role for identifying and treating subclinical symptoms in a neuroprotective manner. In diabetes, for example, an early reduction in blood flow has been shown to produce subclinical signs and symptoms (Meh & Denislic, 1998) before more clinical symptoms present. By enhancing the *rule of the artery*, the body's own healing capacity may produce new cells and improve cell health, giving the Parkinson's brain a chance of collateral function and repair (van Praag, Kempermann, & Gage, 1999).

Magoun (1976) and Sutherland (1990) discuss how the mechanical pressures on the vascular tissues from the cranial bones can adversely affect the circulation in the

brain. Torsions and side bending rotation lesions of the sphenobasilar symphysis can affect the middle cerebral artery, the main source of nutrition to the basal ganglia and to the choroid plexuses of the cranium. Magoun (1968) also mentions that the sphenoid can affect the blood supply to the basal ganglia. Magoun (1968) 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). Other possible areas of compromise include the middle cerebral artery as it passes along the lesser wing of the sphenoid where it can be subjected to mechanical pressure with a torsion or side bending rotation lesion of the sphenobasilar symphysis. This would result in an alteration of the circulation to the sensory and motor regions around the central fissure (Kimberly, 1954; Magoun, 1976). Direct pressure to the brain tissues would likely also result in a local reduction of blood flow similar to other ischemic insults that occur with direct mechanical pressure.

Lesions of the upper cervical spine, the temporal bone, and occiput can affect the path of the vessels entering and exiting the cranium. The path of the vessels to and from the brain and brainstem lies within the neck and the upper cervical spine, within the dura, and along the cranial bones and sutures. The path of these vessels must be maintained in order to maximize the Parkinson's patient's tissue health and prevent neuronal death.

3.1.3 PERFUSION CHANGES IN THE PARKINSON'S DISEASE BRAIN

A reduction in perfusion to certain areas of the brain may correlate with the reduced vitality palpated by the osteopathic practitioner. Measurements of vitality are subjective sensations of health and respiration of the tissues palpated by the osteopathic

practitioner. At the time of Sutherland and Magoun, perfusion testing sensitive enough to measure these small changes in tissue health was not available.

With advances in magnetic resonance (MR) imaging, minute changes in cellular respiration and small changes in perfusion in the brain, previously non-detectable, can now be measured with more sensitive and very specific imaging techniques. Susceptibility weighted imaging (SWI) using magnetic resonance is one such advancement in perfusion analysis of the brain. It can detect very small changes in perfusion by measuring the iron content of the venous return seen after cellular respiration and can show very small reductions in vascularity to specific areas of the brain (Chalian, Tekes, Meoded, Poretti, & Huisman, 2011; Haacke, et al., 2009). With new imaging techniques available, the possibilities of correlating what the osteopathic practitioner palpates with cellular perfusion may be possible.

By measuring brain iron concentration in the substantia nigra, it has been shown that poor perfusion correlates well with functional Parkinson's disease outcome measures, such as the unified Parkinson's disease rating score (UPDRS) (Zhang, et al., 2010). This substantiates the role of fluidic reduction in Parkinson's disease etiology and is consistent with the rule of the artery principle. If osteopathic treatment improves the perfusion to the substantia nigra it is reasonable that the functioning of the Parkinson's patient would improve. There are no studies presently correlating vitality of the cranium with specific perfusion measurements. Osteopathy believes that cellular respiration is evidence of the life force of the tissue, representing the vitality of the structure. Unfortunately, there are no studies presently correlating vitality of the cranium with specific perfusion measurements; however, osteopathy believes that cellular respiration

dependent on a good blood supply is evidence of the life force of the tissue, representing the vitality of the structure. Studies have shown that the severity of Parkinson's symptoms correlate with reduced perfusion in Parkinson's disease, especially in the later stages of the disease (Paschali, et al., 2010). This supports the possibility that the osteopath's subjective measure of vitality and the corresponding treatment should improve the perfusion of the brain.

A future advantage of brain perfusion mapping would be early diagnosis of Parkinson's disease using SWI magnetic resonance imaging. If it would be possible to correlate an osteopathic assessment with imaging, this would provide a cost-effective early diagnostic tool for Parkinson's disease. Early detection, years before the onset of Parkinson's symptoms, could provide a means for Parkinson's disease prevention or an early therapeutic target to slow Parkinson's disease progression.

Although it is now possible to see areas of the brain with suboptimal perfusion, these newer imaging techniques used to detect such subclinical changes are very expensive, and presently are not feasible for this or comparable studies.

An example of how SWI can be used to map the brain of a neurodegenerative disorder is shown in Figure 10. Using specific weighted imaging, very minor changes in blood flow can be detected. The majority of SWI has been completed on multiple sclerosis (MS) patients. The similarities in MS brain lesions and reduction in perfusion supports the comparison. The white lesion areas seen in the image are MS lesions and are similar to Lewy body formation seen in Parkinson's disease (Gupta, Saini, Kesavadas, Sarma, & Kishore, 2010). These lesions are seen on a traditional MRI image, shown on the right in Figure 10, labeled *Flair.* A definitive diagnosis of Parkinson's disease is

based on the presence of Lewy body lesions, similar to those seen in MS. These changes can only been seen on an MRI once enough destruction and iron deposition have occurred. The image on the far left, labeled *Similarity map: nulling veins*, shows the MS lesions in red, indicating an increased iron content as a result of decreased perfusion. Also, in the image on the left of Figure 10, it is possible to see the other areas of the brain that have similar levels of perfusion as the MRI image brain lesion site. The areas of the same colour (red) indicate similar levels of perfusion. This type of mapping is not available for a Parkinson's brain at this time, but the similarities of MS and Parkinson's, and the importance of global perfusion changes in the neurodegenerative brain, prompted a comparison. It is hoped that this type of imaging will expand in the search for more insight into the global nature of Parkinson's disease.

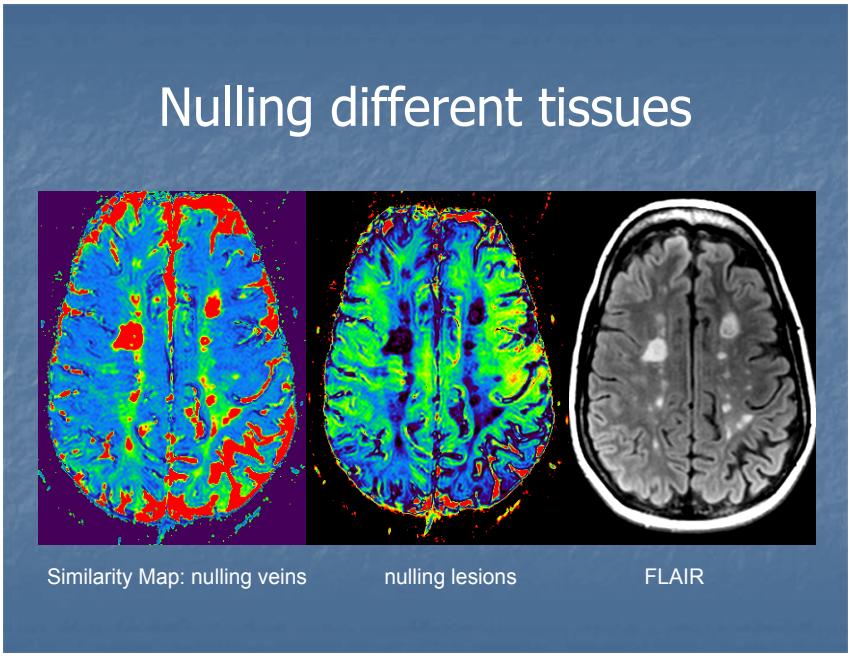

Figure 10. Susceptibility weighted imaging (SWI). The flair image shows two MS lesions seen on a typical MRI. The left image is able to show very sensitively small changes in iron content indicative of perfusion changes. Note All the red areas have similar perfusion to the MS lesion but that have yet to show changes on a traditional MRI. (Haake, 2010, p.32)

The images seen in Figure 10 support the diffuse reduction in perfusion in neurodegenerative disorders, and the consequent need to treat the whole cranium to maximize nutrition for the whole brain and not just one aspect of the brain. Assessing and treating disorders of the brain are more complicated than addressing one factor or area, such as the basal ganglia in Parkinson's disease. These diffuse perfusion changes seen in the brain indicate the possible cause of the diffuse symptoms seen in Parkinson's disease. The diffuse reduction in the arterial supply to other areas of the brain in addition to the basal ganglia lends support to the theory that Parkinson's disease is more than a basal ganglia problem and a more global treatment such as cranial osteopathy may be aptly designed to assist in its treatment.

Interestingly, perfusion changes differ at the early and later stages of insult (Rakshi, et al., 1999). In MS patients with acute lesions there is an initial increase in cerebral blood flow that appears as an apparent healing response to the lesion. Over time those same lesions become ischemic, and brain tissue that is ischemic will result in cell death. This is very similar to the disease process seen in Parkinson's disease, where an acute insult using MPTP produces a local inflammatory reaction (McGeer & McGeer, 2004). However, the glial cells involved in inflammation appear to continue to be active long after the removal of the toxin (McGeer & McGeer, 2004).

It is hypothesized by the author that cranial osteopathic treatment can assist the damaged areas of the brain. There is insufficient evidence to determine what result endocranial treatment may have on these tissue, but it can be hypothesized that by improving blood flow to and from areas of compromise the osteopath can minimize further damage and promote possible repair.

3.1.4 OSTEOPATHIC SIGNIFICANCE OF THE VENOUS SYSTEM

Magoun (1976) discussed the importance of using cranial osteopathy to eliminate circulatory stasis in the brain. Fifty percent of the function of our vascular system is the venous drainage. Magoun (1976) noted that the main cause of circulatory stasis within the brain is a blockage of vascular drainage due to the anatomical vulnerability of the venous vessels. These vessels are particularly vulnerable to distortion because, unlike other veins, they do not have muscular walls to maintain their shape.

The venous drainage of the basal ganglia enters the straight sinus via the great cerebral vein (Gray, Williams, & Bannister, 1995). Therefore, a lesion of the straight sinus could have an effect on the drainage of the basal ganglia. Adequate venous return is necessary for maximal perfusion and cellular respiration. From the straight sinus the venous return flows through the petrosal sinuses, where it exits the cranium via the jugular foramina. 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, Leonhardt, & Starck, 1991). Any reduction in space of these foramina by mechanical stress from the cranial bones can affect the cranial nerves IX, X, and XI as they exit the jugular foramina via the jugular vein or may impact the efficiency of the venous drainage itself (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 occipitomastoid lesions, closely related to the jugular foramen.

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.

3.1.5 VENOUS DRAINAGE AND NEURODEGENERATIVE DISORDERS

Impairment of drainage of the cranium and brainstem can occur within the cranium or in the vessels of the neck into which the cranium is drained. Zamboni et al. (2009) described chronic cerebrospinal venous insufficiency in patients with multiple sclerosis in a number of ways. First, reflux in the jugular veins and the deep cerebral veins can be seen. Second, a type of narrowing of the venous vessels can occur, resulting in a decreased venous return. Lastly, it was shown that the venous drainage of the cranium differs between sitting and standing. The jugular system is primarily being used in supine, while the vertebral vessels are normally producing the majority of the drainage in an upright position (Fisher, Davis, Srikusalanukul, & Budge, 2005; Millson & Tepper, 2004). This may have implications for osteopathic treatment, as the majority of osteopathic treatment is done with the patient supine.

Somatic cranial dysfunctions affecting the jugular foramen are thought to impact the structures passing through it. Of particular interest is the cranial mobility of the temporal bone and occipital bone that form the jugular foramen. Lack of mobility of these bones may affect the function of the petrosal sinus and jugular vein (Magoun, 1976). After exiting the cranium the veins must pass through the cervical spine before finally draining, typically into the subclavian vein.

Zamboni et al. (2009) were able to successfully improve chronic cerebrospinal venous insufficiency through surgical repair of the veins in the neck. They demonstrated the existence of many variances in anatomical and functional drainage of the brain, and by improving the function of the veins of the neck they were able to improve the

neurological functioning of patients suffering from multiple sclerosis. This substantiates the belief that an improved neurological environment can be accomplished in neurodegenerative diseases and that neurological symptoms may be the result of a poor environment. By improving the neuronal environment a symptomatic improvement can be accomplished.

A search of *chronic cerebrospinal venous insufficiency (CCVI)* and *Parkinson's disease* yielded no results in a MEDLINE and CINAHL search. This author believes this will begin to change as our understanding of brain pathology improves. Although Zamboni et. al. (2009) look exclusively at MS and chronic cerebrospinal venous insufficiency, a similar mechanism may work for Parkinson's patients. Their study compared MS patients with three other groups, which included three Parkinson's disease patients. This group did not show statistical significance and they concluded that these diseases did not have CCVI. Upon more rigorous analysis of the *other neurological disease* category it became clear that it would be premature to exclude the possibility that Parkinson's disease may have similar findings. Within this category other neurodegenerative diseases, conditions such as amyotrophic lateral sclerosis (ALS) were included. This is not a fair comparison, as the pathology of ALS lies in the spinal cord and would not be affected by a reduction of cerebral venous action. ALS differs from Parkinson's disease, which is more similar to MS pathology, with both resulting from cerebral neuronal cell destruction.

In both MS and Parkinson's disease, a reduction in venous drainage contributes to fluid stasis, leading to homocysteine build-up. Homocysteine is toxic to the cells and is thought to contribute to cell destruction in Parkinson's and other chronic diseases (Levin,

Blatzel, Giese, Vogeser, & Lorenzl, 2010; Seshadri, et al., 2002; Wald, Morris, & Wald, 2011). Osteopathic treatment directed towards removing blockages of venous drainage could improve the health of the cranial environment in a manner similar to the surgical procedures described by Zamboni and his team. More importantly, cranial osteopathic treatment is safer and may be more indicated in the subclinical stages of Parkinson's disease for neuroprotective purposes.

3.1.6 ANATOMY OF THE VENOUS SINUSES

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

The paired internal cerebral veins are the chief veins 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 pass through the transverse cerebral fissure and form the great cerebral vein of Galen. The great cerebral vein travels a short distance superiorly to join the inferior sagittal sinus, and together they form the straight sinus. Prior to forming the straight sinus, the great cerebral vein is joined by the paired basilar veins responsible for draining the brainstem. The middle cerebral vein is responsible for drainage of the insula and the inferior portion of the basal ganglia and 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 the great vein. The majority of the venous drainage then exits at the straight sinus, which drains into the internal jugular veins (Frick, Leonhardt, & Starck, 1991; Nolte, 1981). The venous sinuses are located within the dura and are susceptable to tension changes within the dura. Note the straight sinus located at the confluence of the falx cerebri and tentorium cerebelli (Figure 9).

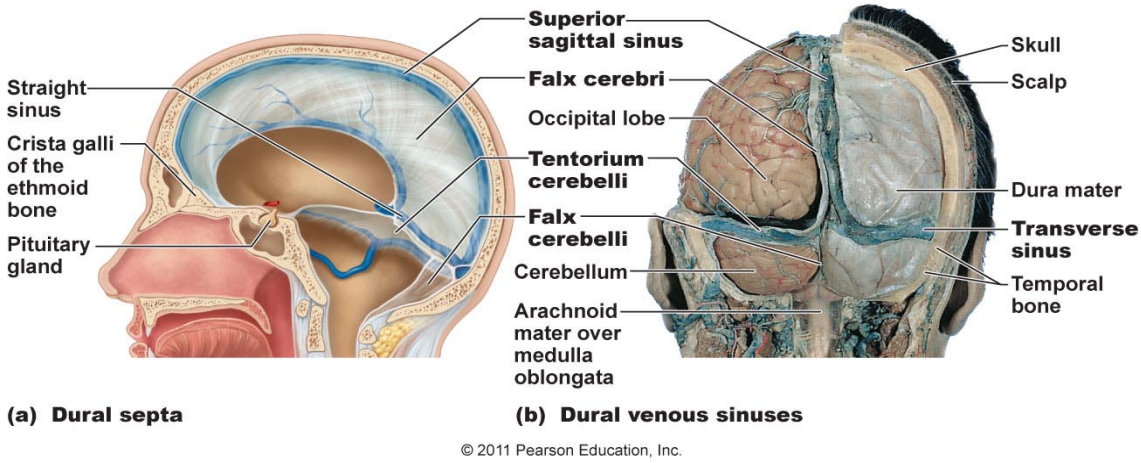

Figure 11. Venous System of the Brain (Marieb & Hoehn, 2011).

3.1.7 ANATOMY AND PHYSIOLOGY OF CEREBROSPINAL FLUID

The cerebrospinal fluid (CSF) bathes the brain and spinal cord and contributes to the nutrition and health of the brain, including the basal ganglia and associated brain areas damaged in Parkinson's disease. Epithelial cells in the choroid plexus produce the CSF. The nutrition from the arterial system is transferred to the CSF by diffusion in the ventricles of the brain (Brown, Davies, Speake, & Millar, 2004).

3.1.8 OSTEOPATHIC SIGNIFICANCE OF CEREBROSPINAL FLUID

Osteopathically, the fluctuation of the cerebrospinal fluid (CSF) is important to the primary respiratory mechanism and is required for the health of the central nervous

system (CNS) (Sutherland & Wales, 1990). Newer studies show that the propagation of CSF extends to all peripheral tissue (Brookfield, Randolph, Eismont, & Brown, 2008; Brown, Davies, Speake, & Millar, 2004; Feinberg & Mark, 1987), perhaps an indication of its significance in all tissue health. This propagation may also relate to the cellular respiration the osteopath feels in all tissue. Becker (1948) described the cerebrospinal fluid as necessary for normal nerve impulses to occur. He believed a change in chemical make-up or restriction of movement would result in a change in nerve impulses and he believed the CSF acted as a neuroprotective barrier. As long as cerebrospinal fluid was healthy, a normal discharge of nerve impulses could occur from the brain and spinal cord (Becker, 1948). The health and normal propagation of CSF may promote neuroplasticity and neuroprotection in Parkinson's patients.

Peripheral inflammatory markers are now being discovered in the brain, indicating that perhaps a peripheral inflammatory condition may lead to inflammation in the brain. According to one theory, inflammation contributes to the pathological process of Parkinson's disease. This new view of communication between the peripheral environments of the body and the environment of the brain supports a symbiotic connection between the peripheral tissue environment and that of the central nervous system. As well, extracranial lymphatics have shown connections to the CSF and connections to intracranial pressures (Koh, Nagra, & Johnston, 2007; Nagra, Koh, Zakharov, Armstrong, & Johnston, 2006; Zakharov, Papaiconomou, Koh, Djenic, Bozanovic-Sosic, & Johnston, 2004). Peripheral lymphatic alterations can influence the environment of the brain itself. This connection between central and peripheral function

supports certain principles of osteopathy, such as the functional unit, and the fluidic connections involved in the rule of the artery.

One attempt to explain the flow of CSF is the Monro-Kellie doctrine (Millson & Tepper, 2004; Neff & Subramaniam, 1996). It states that the central nervous system and its accompanying fluids are enclosed in a rigid container whose total volume tends to remain constant. This differs from the cranial concept, supported in the literature, according to which the container contains some resilience, and can absorb some increase in pressure (Adams, Heisey, Smith, & Briner, 1992). Another explanation for the flow of CSF describes the propagation as a combination of arterial pressure and the pressures induced by respiration (Adams, Heisey, Smith, & Briner, 1992; Heisey & Adams, 1993; Kuhlwein, Balmer, Cannizzaro, & Frey, 2011). Arterial flow to the brain and the production of CSF have an impact on intracranial pressure. An increase in volume of one component (i.e., blood or CSF) will elevate pressure and decrease the volume of the other (Adams, Heisey, Smith, & Briner, 1992). The greater the resilience of the cranium, the lower the pressure elevation in the cranium. If osteopathic therapy is able to improve the resilience of the cranium it should improve the fluidic exchange of its contents: the brain.

3.2 THE PRINCIPLE OF STRUCTURE GOVERNS FUNCTION

This osteopathic principle relating to Parkinson's disease concerns the structures related to the substantia nigra, the basal ganglia, and their function. The upper cervical spine, the cranium, and the soft tissue container of the spinal cord and brain must all be maximized in order for the dopaminergic function of the substantia nigra to be optimized.

Still (1910) described the connection between how a structure moved and its subsequent effect on how that structure and its related tissues functioned. Magoun (1976) in Osteopathy in the Cranial Field described Sutherland's work and the effects cranial

lesions would have on the function of the structures associated with those somatic dysfunctions. Thus, decreased mobility of the cranium will alter the function of the brain and brainstem, including the basal ganglia and substantia nigra. It is known that sub-optimal functioning of the basal ganglia leads to the movement disorders affecting the functional mobility of those suffering with Parkinson's disease, such as freezing, loss of balance, righting reflexes, dyskinesia, and tremor. The peripheral tissue dysfunctions seen in Parkinson's disease are secondary to the altered central nervous system dysfunction and result in further loss of mobility and function. This may possibly lead to further loss of structure and function.

3.2.1 EMBRYOLOGICAL 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), the mesencephalon (midbrain), and the rhombencephalon (hind-brain). The forebrain divides into the telencephalon 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, Leonhardt, & Starck, 1991).

3.2.2 ANATOMY OF THE BASAL GANGLIA

The basal ganglia are paired structures consisting of two groups of cerebral nuclei that are situated in both the left and right cerebral hemispheres. The basal ganglia include the amygdala, the caudate, the putamen, 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). There are numerous connections between the structures of the basal ganglia as well as with other parts of the brain. These integrated connections are responsible for the natural movements that those without Parkinson's disease take for granted. These numerous connections are illustrated in Figure 10.

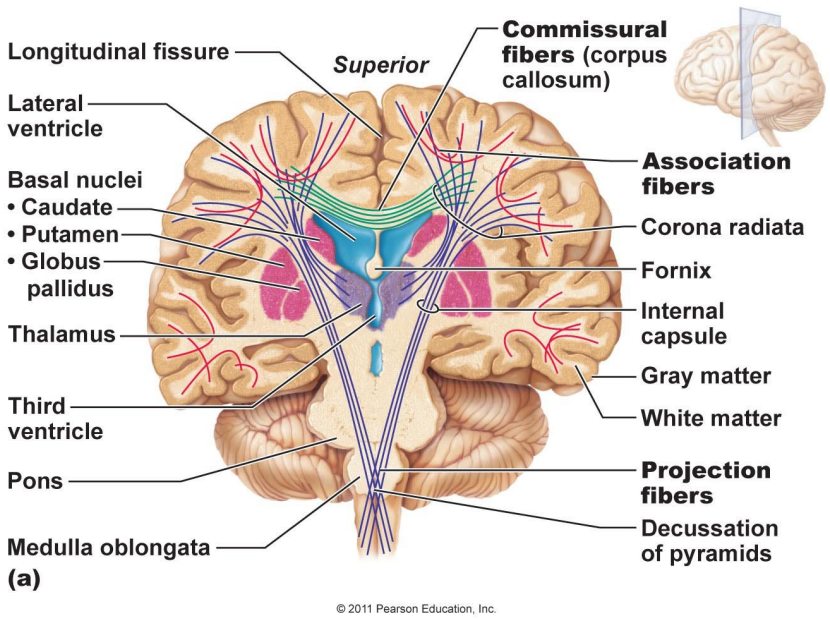

Figure 12. Anatomy of the basal ganlia (Marieb & Hoehn, 2011)

The caudate nucleus of the basal ganglia is a C-shaped structure that encircles the other nuclei of the basal ganglia (Fix, 2009; Nolte, 1981). 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 is the globus pallidus. The caudate is in close proximity

to the lateral wall of the lateral ventricles. This close proximity to the ventricular system may have some connection to the nutritional assistance provided by osteopathic cranial treatment, such as those to be discussed in the autoregulation section and the ventricular techniques performed in this study.

At the end of the tail-like caudate lies the almond-shaped amygdala. The amygdala lies within the temporal lobe just anterior to the inferior horn of the lateral ventricle. The amygdala is considered to be a structure of both the basal ganglia and the limbic system (Frick, Leonhardt, & Starck, 1991). The limbic system is responsible for emotions; the altered structure and function of the basal ganglia, perhaps in the amygdala, provides some connection between the cardinal motor symptoms of Parkinson's disease and the associated emotional affect occurring with the disease (Frick, Leonhardt, & Starck, 1991; Marieb & Hoehn, 2011).

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, Leonhardt, & Starck, 1991). The substantia nigra lies in the midbrain (mesencephalon) and communicates with the striatum. The substantia nigra is the largest nucleus in the midbrain and is divided into two parts: the pars reticulata and the pars compacta (Gates, Torres, White, Fricker-Gates, & Dunnett, 2006). It lies in the midbrain, lateral to the red nucleus, and is easily identified by its dark (nigra) colour (Figure 13). The cell death of the dopaminergic neurons in the pars compacta is primarily responsible for the cardinal symptoms of Parkinson's disease (Gates, Torres, White, Fricker-Gates, & Dunnett, 2006;

Paschali, et al., 2010). The primary role of the pars compacta is to have an indirect influence on motor control (Nolte, 1981).

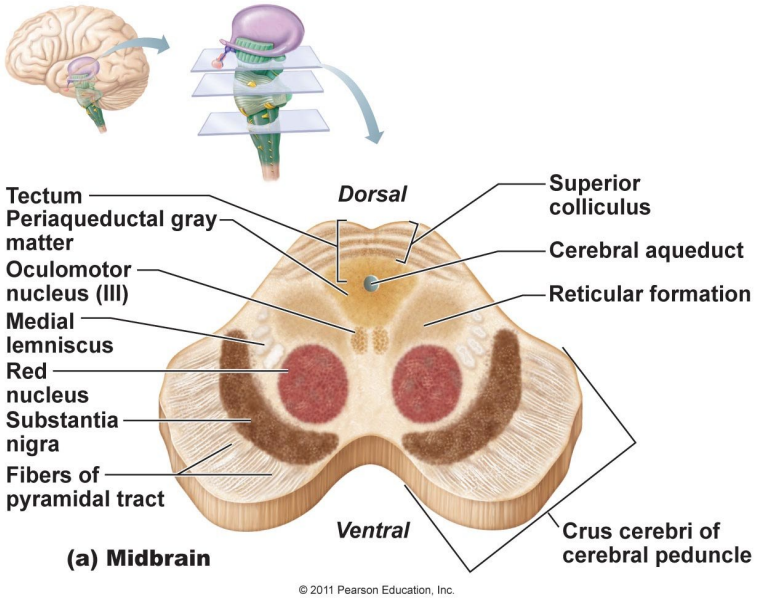

Figure 13. Cross-section of the substantia nigra (Marieb & Hoehn, 2011)

3.2.3 OSTEOPATHIC SIGNIFICANCE OF THE STRUCTURE

By treating the cranial sacral mechanism and improving the mobility of the structures within the cranium, the osteopathic treatment should theoretically maximize the Parkinson's patient's own disease-fighting systems. The substantia nigra and basal ganglia, primarily responsible for Parkinson's disease, are in direct relationship with the cranial base. The cranial base and their foramina provide the entrances and exits for the vessels responsible for supplying the brainstem and brain with the required nutrients and waste removal. The principle of *structure governs function* theorizes that the cranial bones as the container for the central nervous system, and thus the function of the central nervous system, are dependent on the mobility of the cranium (Treganza & Frymann, 1998). "Compression or trauma can create impairment of physiologic motion and it is the

disturbance of the inherent motility that leads to the manifestation of progressive neurologic dysfunction within the central nervous system" (Frymann, 1998, p. 203).

If the structure of the Parkinson's patient is compromised the function will be adversely impacted. The spinal cord lies in the central canal of the spinal vertebrae and the structure and movement of the head, neck, vertebrae, and sacrum affect the protective soft tissue layer of the brain and spinal cord, the dura. If there is compromise in structure in any of these areas, this osteopathic principle states its function will be compromised.

The posture or *structure* of the spine progressively becomes more stooped and moves farther from the ideal posture with the progression of Parkinson's disease (Clarke, 2007; Fargel, Grobe, Oesterle, Hastedt, & Rupp, 2007; Lang & Lozano, 1998; Smithson, Morris, & Iansek, 1998). The typical late-stage Parkinsonian posture is represented by an increased thoracic kyphosis with a resultant forward head posture. This leads to craniovertebral extension (Clarke, 2007; Lang & Lozano, 1998; Marsden, 1994; Smithson, Morris, & Iansek, 1998) and compression of the posterior structures located at the base of the cranium. Rivera-Martineza (2002) found lesions at the occipital condyles (C0) and the first cervical vertebra (C1) in later stage Parkinson's patients consistent with this late stage posture. The compression of the vessels, nerves and foramen at the base of the cranium as a result of the late stage Parkinsonian posture could contribute to a more rapid progression of symptoms in the later stages of the disease. Along with the structural change of the vertebra there is also an increase in muscular rigidity that coincides with disease progression. The greater rigidity of that occurs in the muscular system as a result of Parkinson's disease will affect the muscles of the sub-occipital region that is responsible to maintaining the forward head posture and compressed craniovertebral

region. This rigidity in the peripheral muscular system can contribute to the somatic dysfunctions seen in the spine. As a result, the worsening of posture and increasing rigidity can further alter the function of the spinal segments and affect the neurological function to and from those vertebrae affecting autonomic functions including vascular supply and the overall health of the end organs to which those nerves travel (Korr, 1975a, 1975b). In the case of Parkinson's disease the supply of the nerves from the cervical spine impact the supply to the cranium and basal ganglia, from these upper cervical nerves and the sympathetic ganglia, in particular the superior cervical ganglia located at the level of C1. Owens (1999) was able to show the affects of somatic dysfunction and structural asymmetry and their effects on systemic function of the body and the endocrine system.

It is difficult to ascertain whether a C0 lesion was present for many years prior to Parkinson's symptom presentation and possibly contributed to the pathological process associated with Parkinson's, or whether it contributed directly to the altered *stooped* posture seen in later stage Parkinson's. It is also difficult to determine whether the C0–C1 lesion occurred as a result of the altered posture and progressive stiffness seen in the later stages of Parkinson's disease. The increased rigidity and postural instability may lead to the posture and this may result in the C0–C1 lesions. Sustained tension of the cervical musculature as a result of muscular rigidity and tremor occurring in Parkinson's disease could lead to tension on the occiput and temporal bones at the cranial base. The rectus capitis posterior minor, ligamentum flavum, ligamentum nuchae, and cervical fascia have attachments to the dura (Mitchell, Humphreys, & O'Sullivan, 1998). A pull on the fascia from a cranial lesion of the base can alter the reciprocal tension membrane in the cranium

and the dural mechanism down the spine and can cause changes to the position of the sacrum (Magoun, 1976).

Compromised structure of the cranial base will affect the functions associated with the structure; *structure governs function*. The progression of Parkinson's disease increases in the later stages of the disease. As structure governs function it may also be said that function governs structure. It is possible that the Parkinsonian posture could contribute to a more rapid progression of the disease.

A stooped Parkinsonian posture alters muscle tension and changes joint compression, which over time has been shown to cause joint deterioration. Changes in the functional activity of a joint can lead to development of osteoarthritis (O'Connor & Brandt, 1993).

The cranial bones are related to vital structures of the brain responsible for coordinating the movements that are primarily affected in Parkinson's disease. The basal ganglia are paired structures just superior to the cranial base. The basal ganglia are related to the transverse axis of the occipital bone and are therefore thought to be more susceptible to injury by cranial dysfunctions. Limitation of movement of these bones may compromise the health and function of the basal ganglia. The primary area of motor is the sensory motor cortex, which may be compromised by a parietal lesion. Although these areas are not directly implicated in Parkinson's disease, compromise of these cortexes would influence motor function.

The function of the venous drainage of the basal ganglia and cranium are dependent on the motion of their corresponding sutures. The jugular vein draining the majority of the brain exits the cranium through the jugular foramen. The jugular foramen

is formed by the petrous portion of the temporal bone and the occiput (Gray, Williams, & Bannister, 1995). Along with the jugular vein, other structures that exit the jugular foramen can be compromised. The glossopharyngeal nerve (CN IX), responsible for tongue motion and relating to jaw position and posture (Sakaguchi et al., 2007), could be affected. The vagus nerve (CN X) and accessory (CN XI) nerve also exit through the jugular foramen. Compromise of the vagus nerve as it exits the jugular foramen may adversely affect the gastrointestinal system whose primary parasympathetic innervation is the vagus nerve. This may explain why Parkinson's patients frequently report alterations of the digestive system (Eliassi, Aleali, & Ghasemi, 2008; Natale, Pasquali, Ruggieri, Paparelli, & Fornai, 2008). The vagus nerve is also the parasympathetic innervation to the liver and pancreas, responsible for the digestive enzymes. Cranial lesions involving the occiput or temporal bones forming the jugular foramen at the base of the cranium could affect the vagus nerve and influence the digestive system. There is also evidence of Lewy body formation in the vagus nucleus (Alegre-Abarrategui, Ansorge, Esiri, & Wade-Martins, 2008; Jager, Hartog, & Bethlem, 1960; Natale, Pasquali, Ruggieri, Paparelli, & Fornai, 2008). The function of the CNS can be affected by alteration of the foramen magnum where the vertebral-basilar artery system is located as well as the spinal cord and brainstem itself (Goel, Sharma, Dange, & Kulkarni, 2005).

Although the basal ganglia lie deep in the grey matter of the telencephalon, they are anatomically related to the cranial base and therefore may be affected by cranial base dysfunctions. Another important structure for general homeostasis is the hypothalamus and its connections to the pituitary and endocrine system. The hypothalamus is in anatomical relation to the basal ganglia. The overall health of people, including

Parkinsons's patients, is dependent on homeostasis of the endocrine system. The hypothalamus is also thought to be subject to distortion by occipital malpositions (Magoun, 1968). If an occipital cranial lesion compromised the function it would also affect the cellular health of the Parkinson's patient.

3.3 THE BODY AS A FUNCTIONAL UNIT

When treating one aspect of a person, the osteopath is accessing all of the systems of the body, not just the musculoskeletal system. The human body is intimately connected fascially, and systemically via the neural, endorcrine and fascial system. Still (1910) believe that in order to allow the osteopathic client to maximize their health the whole body the osteopath cannot just treat the location of the symptoms. Parkinosn's disease results from a loss of function of the dopaminergic neurons in the substantia nigra. However, the primary symptoms are found in the reduced function of the peripheral tissues in the form of tremor, rigidity and bradykinesia. The osteopathi must consider the treatment of the brain in order to effectively treat the Parkinson's patient. Still (1910) considered the brain the storehouse of the body. "I [Dr. Still] begin with the head as I consider it the organ or division of the body in which most of the fluid and force for the use of the entire body is generated and stored" (p.35).

The classic Parkinsonian posture is an uncompensated anterior typology with a forward flexed spine (Figure 12).

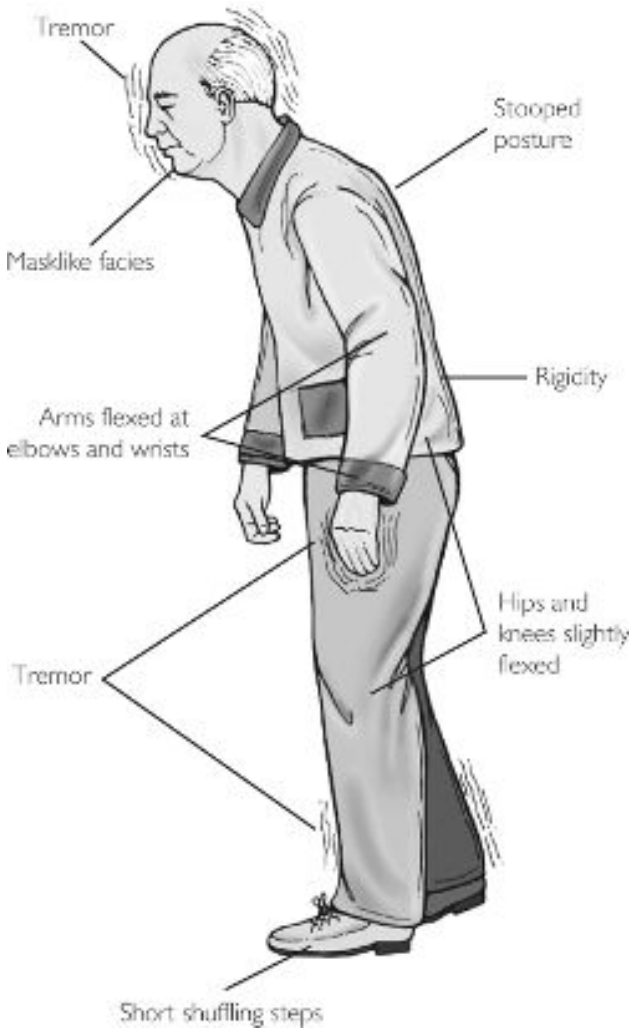

Figure 14. Parkinsonian Posture (Joseph, 2000, pg. 46)

Flexion occurs at the hips, knees, ankles, and thoracic spine, and at the occiput on the atlas. A correction at one location will have an effect on the other areas affected by each specific line of gravity (Jacobs, Dimitrova, Nutt, & Horak, 2005). The postural imbalance seen in people living with Parkinson's may contribute to dysfunction at the cranium, or conversely, dysfunctions in the cranium may lead to full body postural alterations. Cranial osteopathic treatment may improve both the primary and postural compensatory lesions in those with Parkinson's disease.

The sympathetic division of the nervous system is an important part of the regulatory system responsible for maintaining blood flow and general homeostasis. The superior cervical ganglia (SCG) sit anterior to the upper two cervical vertebrae. They are intimately related to the cranial base and are responsible for the control of the arterial supply to the brain; they also influence the heart (Gray, Williams, & Bannister, 1995). If an osteopathic lesion in this area affects the SCG it could affect the blood supply to the brain and basal ganglia. Still (1910) and subsequently supported in the literature that sympathetic inhibition or stimulation would have far reaching believed that

The cranium and upper cervical segments have strong attachments to the dura, the covering of the central nervous system. Freedom of the dura is required for the involuntary movement of the sacrum (Sutherland & Wales, 1990). The straight sinus was discussed in the rule of the artery section with the other venous sinuses. Importantly, it lies within the dura. The tentorium cerebelli portion of the dura is considered to be part of the three diaphragms osteopathically. Frymann (1968) described the concept and the coordinated movement of the three diaphragms, and noted the importance of treating these diaphragms in relation to the global health of patients. This provides support for additional possible global effects of cranial osteopathic treatment in the treatment of Parkinson's disease, although not all three diaphragms were directly treated in this study.

The tentorium cerebelli, one of the three diaphragms, provides a connection between the cranium and the thoracic diaphragm and therefore cranial treatment may result in improved diaphragmatic function and breathing for Parkinson's patients. The thoracic diaphragm is the primary muscle of respiration. A reduction in respiration results in a reduction of supplied oxygen. Respiration is a key element in cellular health, as

oxygen is required by all tissues, and in particular, the brain and brainstem, which require a disproportionate amount of oxygen delivery. Respiration in those with Parkinson's disease has been connected to postural alterations. The stooped posture typical of late-stage Parkinson's patients also limits thoracic cage expansion and has been shown to reduce lumen diameter of the airway (Bacon, Berreur, Krieger, Hildwein, & Stierle, 1992). A second factor in limiting respiration in the Parkinson's patient is through the fascial connections to the pharynx from the cranial base. An increase in tension may result in an increased tension in the airway. The inter-relationship between arterial flow and respiration, both needed for cellular respiration, is thought to be behind the primary respiratory rhythm (PRM) palpated in cranial osteopathy. The PRM and vitality depend on proper functioning of both of these systems.

The sphenobasilar symphysis (SBS) is the primary articulation controlling the expression of the primary respiratory mechanism (Magoun, 1976). The SBS is the articulation of the basi-sphenoid and basi-occiput. It has direct fascial attachments to the pharyngobasilar fascia (PBF) below and the dura above (Magoun, 1976). Its mechanical movement is a component of the action of the reciprocal tension membrane and is associated with the action of the whole craniosacral mechanism and the fluctuation of the cerebral spinal fluid. Influencing the fluid and fascial movement of the brain as a whole and the sacrum caudally, the reciprocal tension membrane (RTM), especially in a fluid protocol, can have a powerful effect on the syntonisation of the autonomic nervous system (Druelle, 2006). The SBS is considered part of the central chain described by Druelle (2006) and is in close proximity to the pituitary gland (Gray, Williams, & Bannister, 1995). Magoun (1976) believed the SBS is essential for understanding the

entire cranium since it is related to some of the most vital parts of the nervous system. The SBS is located in the cranial base and was a primary area addressed by treatment in this study. The dura and SBS are intimately related to the sella turcica and the pituitary gland, responsible for control of the human endocrine system with the pituitary.

3.4 THE PRINCIPLE OF AUTOREGULATION

Auto-regulation is the body's inherent ability to heal itself. When the human body is functioning optimally, its tissues are the healthiest and in the best position to heal injured or damaged tissues. Maintaining homeostasis maximizes health, and improving the physiological function is part and parcel of the osteopathic approach (Kuchera & Kuchera, 1994; Wright, 1960). Of particular importance is the cranium. Magoun (1976) believes "no system of treatment involves a wider potential of influence, with full reciprocity in the interrelation of organ systems, as controlled by the all-important nervous system; with biochemical, bioelectrical and hydro-lymph stream and the tissue juices" (p. 106). If the appropriate environment exists for the tissues of the body, the body's own healing systems are in the best position to combat disease and heal injured tissue, and are in the best position to compensate any compromise or deficit in other areas of the body (Druelle & Forget, 2000).

In Parkinson's disease, symptoms do not appear until 60 to 80 percent of the dopamine-producing neurons are destroyed (Adler & Ahlskog, 2000). This may indicate that the Parkinson's patient has an impaired autoregulation mechanism. The body normally has back-up mechanisms for dopamine production. This is evidenced by the secretion of dopamine in the anterior cingulate of the frontal lobe and even peripherally in the adrenal glands (Rakshi, et al., 1999). Improving the body's compensatory mechanisms may limit the progression of the disease through collateral dopamine

production. It would enhance the body's own ability to deal with the dopamine deficiency. Perhaps through improved autoregulation the Parkinsonian patient would be better able to utilize the available dopamine and maximize central nervous system function.

Druelle and Forget (2000) discussed the systemic influences that many osteopathic techniques can have on the health of the autoregulatory systems of the body. Osteopathic treatment is thought to enhance the autoregulation in a number of ways. The proposed therapeutic effects are listed in Table 1.

Table 1. Desired Effects for Autoregulation (Druelle & Forget, 2000)

Therapeutic Effects described by Druelle and Forget (Druelle & Forget, 2000)	**Techniques used to achieve these changes**
Increased vitality	Venous Sinus Bilateral Temporal Rocking Lateral Ventricles
Increased venous drainage	Venous Sinus Bilateral Temporal Rocking
Increased fluid drive	CV4
Balanced membranes – thought to improve function of cranial mechanism and improve homeostasis	EV4 Posterior Fossa CV4 Parietal Lift Bilateral Temporal Rocking Lateral Ventricles
Balanced fluids	Parietal Lift Lateral Ventricles
Harmonized membranes and fluids	Parietal Lift Bilateral Temporal Rocking
Decreased inflammatory process	CV4
Influence the autonomic nervous system	CV4
Restore ventricular pumping function	Lateral Ventricles
Increased production of cerebrospinal fluid	Lateral Ventricles
Increased resiliency of the encephalon	Parietal Lift – primarily sensorimotor cortex Bilateral Temporal Rocking
Decompress the encephalon against the base of the cranium	Parietal Lift
Stimulate the thalamus and central nuclei	Bilateral Temporal Rocking
Increased systemic activity of the brain	Lateral Ventricles

A common symptom in Parkinson's disease is orthostatic hypotension (Fisher, Davis, Srikusalanukul, & Budge, 2005; Jankovic & Stacy, 2007; Stryjer, Klein, Treves, & Rabey, 2005). This is important because it results in a temporary reduction in blood supply to the brain. It is also an indication of a person who is not in functional homeostasis, as they are unable to make rapid circulatory adaptations. This leads to poor perfusion of the brain as a result of changing body positions. If cranial osteopathic treatment can improve the CNS and autonomic nervous system function, it will help these systems in the Parkinson's patient, and should improve global function, evidenced by an improvement in functional mobility. Autonomic involvement, common with Parkinson's and orthostatic hypotension, is being postulated as a contributing factor to Parkinson's disease as a result of a reduction in blood flow to the dopamine-producing cells of the basal ganglia.

The goal of this study is to show improvement in gait and functional mobility in those with Parkinson's disease because of their relevance and close correlation with falls and quality of life. Autoregulation of the overall brain and neurological function of those with Parkinson's disease appears to correlate well with focusing treatment in the hope of maximizing cerebral perfusion (the rule of the artery), maximizing the upper cervical and cranial base (structure governs function), and understanding and addressing the interconnections of the Parkinson's patient (the functional unit). Osteopathic cranial therapy is believed to improve somatic dysfunctions of the craniosacral mechanism, thus maximizing the autoregulatory system of the Parkinson's patient.

3.4.1 THE ROLE OF INFLAMMATION

Another prevalent theory of Parkinson's disease etiology is the role of inflammation. It was discovered inadvertently when drug users began presenting with

Parkinson's symptoms. It was found that exposure to MPTP created dopaminergic cell death in the substantia nigra. Studies using MPTP to induce Parkinson's disease in rats have shown that exposure to this toxin produces an inflammatory response in the basal ganglia. This is evidenced by a glial cell reaction. Hirsch (2007) was able to show that the inflammatory response and glial cell activity continues long after the exposure to the toxin has been removed. The authors hypothesized that the continued cell damage may occur long after the initial toxic insult. Although this inflammatory mechanism has been recognized, no studies using non-steroidal anti-inflammatory medications (NSAIDs) have shown benefits in the treatment of Parkinson's disease (Rees, et al., 2011).

Other successful pharmacological treatment, directed at the possible oxidative or inflammatory contribution, which has been used in the treatment of Parkinson's disease, is a class called monoamine oxidative (MAO) inhibitors. Selegiline is a MAO inhibitor and believed to reduce the oxidative stress in the basal ganglia. It is hypothesized that, given in combination with levodopa, it would provide a greater improvement for those with Parkinson's disease (Jankovic & Stacy, 2007). There are refuting studies in this regard. The gains resulting in reduced oxidative stress may be negated by reduced blood flow in others (Olanow & Jankovic, 2005). Unfortunately, studies have shown the prolonged levodopa in combination increases the likelihood of developing orthostatic hypotension(Stryjer, Klein, Treves, & Rabey, 2005).

Cranial osteopathic treatment does not address symptoms in isolation, but rather hopes to achieve better homeostasis in the patient. Homeostasis by means of cranial osteopathic treatment should not only increase blood supply to the basal ganglia, but also

improve orthostatic hypotension and general perfusion to the basal ganglia, substantia nigra, and the neighbouring nervous tissue.

This exciting time in medical research continues to support the tenets of osteopathy. Osteopathy addresses the whole body and the interconnections between systems. By treating the primary lesions, and viewing the Parkinson's patient as the best person to promote healing, the treating osteopathic practitioner may promote the autoregulation system and improve health.

3.4.2 NEUROPLASTICITY

Neuroplasticity, or cortical 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. Recent preliminary research suggests that physical exercise effects neuroplasticity in Parkinson's disease (Hirsch & Farley, 2009).

3.4.3 COMBINED CRANIAL THERAPY AND EXERCISE

There is evidence of the brain's ability to change, repair, and alter itself. General physical exercise and brain-specific exercises such as visualization and cognitive behavioural therapy have been shown to improve neuronal function (Will, Galani, Kelche, & Rosenzweig, 2004). Will et al. (2004) have been able to show increased neuronal size and increased brain mass with thought and brain exercises. Imaging, using PET (Leenders et al., 1990), SPECT (Paschali, et al., 2010; Velakoulis & Lloyd, 1998), and fMRI (Shine, Ward, Naismith, Pearson, & Lewis, 2011; Thomason et al., 2011) provide evidence that brain activity results in an increase in blood flow to meet the

demands needed for cellular perfusion. Like a muscle, the brain requires an increase in vascular supply when there is increased demand. If the demands of the tissue are not met, ischemia results. The brain can be thought of as the master muscle. If adequate perfusion is not met, the brain cannot perform optimally.

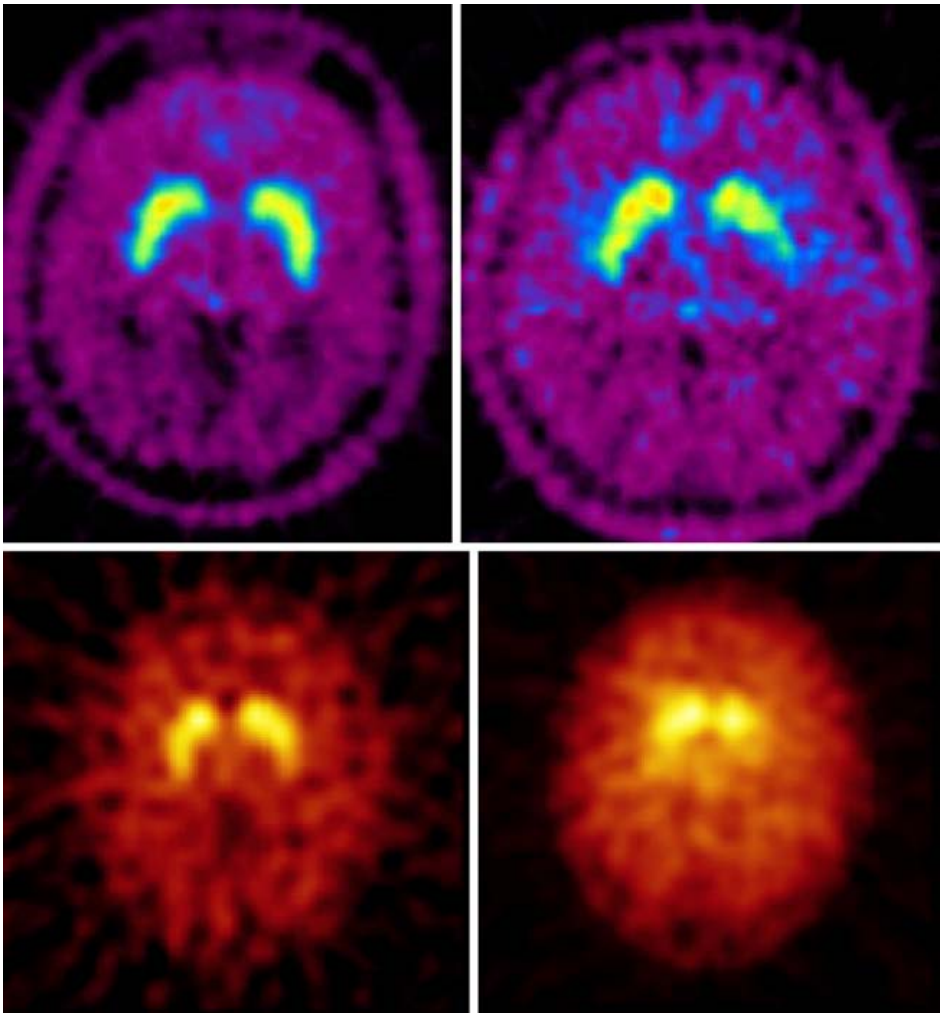

Figure 15. F-DOPA PET scans in (upper left image) healthy controls and (upper right) patients with early-stage PD, and FP-CIT SPECT scans in (lower left) healthy controls and (lower right) patients with early-stage PD (Eshuis, Jager, Maguire, Jonkman, Dierckx, & Leenders, 2009, pg.202)

Figure 15 shows the perfusion changes in the brain seen in early stages of Parkinson's disease on PET and SPECT scans versus normals. Of note is the loss of

perfusion in the central brainstem ganglia. Figure 16 illustrates perfusion differences between the depressed brain and a normal brain. Again, the alteration in perfusion, in particular the central brainstem ganglia may provide insight to the connection between perfusion and neurodegenerative pathophysiology seen in neurodegenerative disease. Physical and intellectual exercise altar perfusion to the brain.

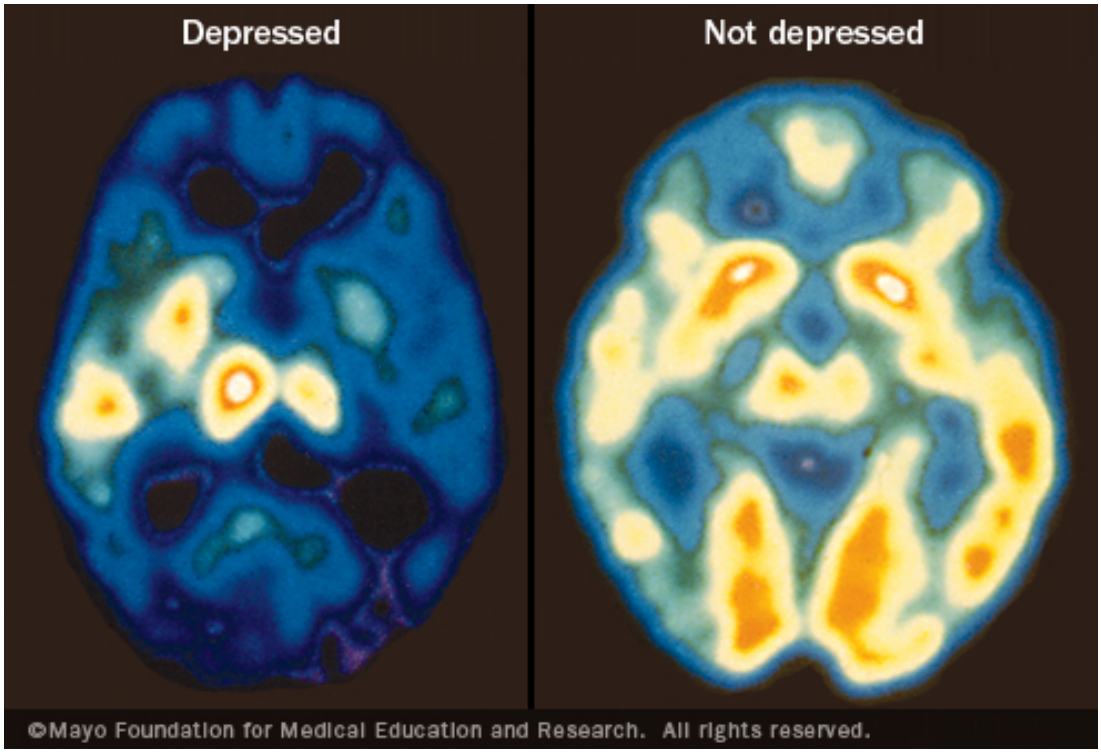

Figure 16. PET Scan showing the difference in perfusion in those with depression vs. normals (Mayo foundation for medical education and research, 2011)

Lancee (2005) showed that through stimulation of certain areas of the brain by cognitive exercises, sub-functioning areas of the brain are able to restore optimal function. This is similar to the findings made by Frymann (Frymann, 1976a), who was able to show improvements in academic scores of all children receiving cranial osteopathic treatment. Yue and Cole (1992) were able to show that imagining using one's muscles actually strengthens them.

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 midbrain to the striatum (Fix, 2009). These imbalances reduce the activation of the motor cortex, producing the cardinal symptoms of Parkinson's disease. Parkinson's disease brains, along with other neurological brain diseases, such as depression (Velakoulis & Lloyd, 1998), fibromyalgia (Guedj et al., 2007), and learning disabilities (Thomason, et al., 2011) also seem to be a result of an imbalance in neuronal firing and all show alterations of blood flow to different areas of the brain (Eshuis, Jager, Maguire, Jonkman, Dierckx, & Leenders, 2009; Lee, et al., 2009). This may help explain the inter-connection between Parkinson's disease and depression. Research has shown that smiling increases the release of serotonin, the neurotransmitter responsible for happiness. Parkinson's patients frequently suffer from depression. Perhaps the mask-like appearance of a Parkinsonian face as a result of motor dysfunction, leads to a reduction in smiling and may contribute to the prevalence of depression seen in Parkinson's patients. If so, it is a further argument in favour of the use of a global treatment approach in the treatment of Parkinson's disease.

3.4.4 NEUROPROTECTION

The theory of neuroprotection suggests that the nerve cells can be made more resilient and thus are in a better position to withstand a suboptimal environment.. Likewise, by improving the environment the nerve cell, it is able to become more robust. Neuroprotection is an essential component for consideration in the treatment of a degenerative disease such as Parkinson's diesease. Unfortunately, the clinical symptoms of Parkinson's disease often do not appear until 60 to 80 percent of the substantia nigra neurons are destroyed (Adler & Ahlskog, 2000). This statistic demonstrates the

importance of the neuroprotection of the remaining neurons within the basal ganglia once the disease becomes symptomatic and even more important if early detection were possible. The oxidative stress model (Jenner & Olanow, 1996) hypothesizes that the destruction of the striatal pathways is somehow caused by either exposure to too many toxins, or the inability to remove the toxins, which may increase the tissues' duration of exposure. If osteopathic cranial treatment is able to improve homeostasis, increase perfusion, and address the body globally, it is reasoned that this type of treatment will be neuroprotective.

The lesions of the Parkinson's brain, Lewy bodies, are produced by the increase in iron content that results when there is inadequate perfusion to an area of the brain. This increase in iron content has also been linked to elevated levels in homocysteine. An increase in homocysteine has been shown in the Parkinsonian brain (Levin, Blatzel, Giese, Vogeser, & Lorenzl, 2010) and is connected to other neurodegenerative disorders such as dementia and Alzheimer's (Seshadri, et al., 2002). Improved perfusion would result in less iron deposition and improved homocysteine levels. The reduced levels of homocysteine are thought to protect against the development of Lewy bodies seen in Parkinson's disease (Belcastro et al., 2010; Levin, Blatzel, Giese, Vogeser, & Lorenzl, 2010).

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 in elimination of toxins (McGeer & McGeer, 2004). This further shows that optimal vascularization to

the basal ganglia and surrounding tissues may contribute to the removal of potentially damaging toxins.

3.4.5 NEURORESTORATION

The preservation of nerve cells should help those with Parkinson's disease. A cure of Parkinson's disease and all neurological disease lies in the ability of nerve cells to regrow. The existence and discovery of neurogenesis began with the discovery of neuronal stem cells. These cells can divide and differentiate to become neurons or glial cells and from there can specialize their actions (Eriksson et al., 1998). They are also shown to exist in those with advanced age and even just before death (Eriksson, et al., 1998). Interestingly, these neuronal stem cells have been found in the olfactory bulb and striatum, areas that are similarly affected by Parkinson's disease (Eriksson, et al., 1998). There may be a connection between the loss of these neuronal stem cells and loss of regeneration in the pathogenesis of Parkinson's disease.

Nerve regeneration is possible. First, neural re-growth by exposure to a stimulating environment can increase neuronal genesis. Mice showed an increase in brain volume and 40,000 new neurons when placed in a stimulating cage environment compared to mice raised in ordinary cages (Kempermann, Gast, & Gage, 2002). Secondly, increased blood supply induced by exercise has also been shown to increase cell proliferation in the brain (van Praag, Kempermann, & Gage, 1999). If cranial osteopathy is shown to increase blood supply, by maximizing the mechanical and neurological pathways, one could hypothesize that neuronal cell proliferation may be possible. Increased cell proliferation should improve neuronal function and help in the fight against the degenerative processes of Parkinson's disease. Exercise also contributes to an increase in the neuronal growth factor BDNF, which affects neuroplastic changes in

the brain (Vaynman & Gomez-Pinilla, 2005). This increased cell proliferation brought about by exercise, in combination with enhanced cell survival resulting from learning new tasks and skills, can behave in a neurorestorative manner. Physical exercise and learning seem to work together to optimize neuronal health in normal individuals. The author hypothesizes that the same could occur in the Parkinson's brain.

3.5 SUMMARY OF OSTEOPATHIC JUSTIFICATION

Parkinson's disease is a complicated neurodegenerative disease with no known cure. Cranial osteopathic treatment is a non-invasive, well-received, comfortable treatment intervention with no known complications. The principles of osteopathy, and in particular cranial osteopathic treatment, may provide a treatment method to minimize somatic dysfunction that may impede homeostasis. This thesis 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. By improving homeostasis, the Parkinson's patient's brain is better able to repair itself, remove inflammation, and maximize collateral function.

4 CHAPTER FOUR: RESEARCH METHODOLOGY

4.1 TYPE OF RESEARCH

This study was a randomized single-blinded between-group design that examined the effect of cranial osteopathy on the functional mobility of subjects with Parkinson's disease. It consisted of a control group that performed exercise only and an experimental group that received both exercise and four one-hour cranial osteopathic treatments. A pre-intervention and post-intervention assessment using the Timed Up and Go (TUG) test was administered to both groups. A pre- and post-intervention osteopathic evaluation was completed to identify and classify the osteopathic lesions and their vitality. This was performed for both the control and the experimental groups.

4.2 TARGET POPULATION

Prior to the outset of this study a power analysis determined a sample of 16 (eight per group) was needed to show statistical power. A number of papers employing TUG measures were reviewed for information that could be used for sample size calculation (Hackney, Kantorovich, Levin, & Earhart, 2007; Hain, Fuller, Weil, & Kotsias, 1999). Assuming four equally spaced treatment occasions, a power of 80 percent, and an alpha = 0.05, PASS2000 produced a sample size of eight per group. A sample size of only 11 subjects was achieved despite rigorous recruitment. The target population was composed of 11 volunteers (five male and six female) between the ages of 55 and 90 who had a neurologist diagnosis of idiopathic Parkinson's disease.

4.3 INCLUSION CRITERIA

- Primary diagnosis of idiopathic Parkinson's disease by a physician
- Can ambulate with or without an assistive device
- Between the ages of 55 and 90 years old
- Signed consent form (Appendix A)

- Completed medical questionnaire
- Rating between stage II and stage IV on the Hoehn and Yahr scale (Appendix B)

4.4 EXCLUSION CRITERIA

- 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
- Diagnosis of a balance disorder other than Parkinson's disease
- 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
- Any diagnosis of dementia or significant cognitive impairments limiting the subject's ability to understand instructions

4.5 INDEPENDENT VARIABLE

The independent variable was the cranial osteopathic treatment. Both groups received the exercise protocol, but only the experimental group received the osteopathic cranial intervention. Each osteopathic treatment followed the guidelines described in section 4.5.2. The treatment protocol was created to allow some individualization based on individual findings but also rigid as possible to ensure each subject in the experimental group received maximal uniformity within each treatment. Each subject presented differently and likely had different disease progressions and therefore each subject received treatments appropriate for that person.

In order to maximize inter-rater and intra-rater reliability a CCO faculty member validated the hands of Stacey Hauserman and Thomas Hein, prior to commencement of

this study. On September 8, 2009, at the Canadian College of Osteopathy, Toronto campus, the researchers were asked to evaluate four structures and write down the lesions they perceived. Brad McCutcheon, D.O.M.P, Principal of the Canadian College of Osteopathy, Toronto School, had previously evaluated the volunteer and recorded his findings. The findings were compared. The findings were consistent among evaluators and Brad McCutcheon determined both the researchers' hands to be validated.

4.5.1 EXERCISE INTERVENTION: CONTROL AND EXPERIMENTAL GROUPS

The exercise intervention was the standard exercise protocol described by the Parkinson's Society of Canada, supervised by registered physiotherapist Hermina Vas (Appendix C). The exercise protocol was one hour in duration each week for four consecutive weeks. It was included to provide all participants with some type of therapy. It also provided an intervention to assist in blinding the subjects from which group they were part of.

The physiotherapist was trained by the researchers to teach all the exercises in the exercise protocol to each participant at their current level of function, as long as they could be performed safely. The first sessions acted as an introductory session where each exercise was described, demonstrated, and attempted by each participant. Postural education and exercise in lying, sitting, and standing were described.

Stretching was the second section of the exercise protocol. The physiotherapist was trained to read the instructions to each subject, demonstrate, and then correct the subject to ensure that the stretches were completed as accurately as possible. The general stretches targeted the spinal rotators, latissimus dorsi, pectoralis major, deltoids, the hip flexor group, quadriceps, soleus, and gastrocnemius. The subjects were instructed to hold the stretches for 30 seconds, holding gently with no pain.

The next section included strengthening exercises. Isometric and isotonic exercises were included. Both upper and lower body strengthening exercise were part of the program. The physiotherapist determined the amount of weight so that there was subjective fatigue on the third set of ten repetitions. If no fatigue was noted, the weight was increased by one pound the following week. A series of functional movements, such as marching and sidestepping, were also included. Exercises for the facial muscles, such as smiling and lifting the eyebrows, were reviewed at the end of each exercise intervention. Two subjects did not complete the sidestepping exercise due to safety concerns.

4.5.2 CRANIAL INTERVENTION: EXPERIMENTAL GROUP ONLY

The subjects assigned to the experimental group received a cranial osteopathic intervention one time per week in weeks two through five of the study. The treating thesis-level osteopathic student (Treator) performed the one-hour cranial intervention prior to the exercise protocol in weeks two through four and prior to the final testing in week five of the study.

The goal of the first osteopathic treatment, given in week two, was to clear non-physiological lesions that could impede the craniosacral mechanism. This included an evaluation and treatment of all spinal segments and pelvis. The first treatment session was concluded with a venous sinus technique to increase vitality and drainage, thereby increasing circulation. A more detailed description of each technique is available in Appendix D.

The second treatment focused on the craniovertebral spine and the cranium itself. The choice of techniques was based on the severity of each lesion. The classification and severity of each lesion was based on the clinical methodology of the CCO (Appendix E).

Compactions were addressed first, followed by non-physiological lesions without respect to the axis within the cranial base, the atlas (C1), and the axis (C2). The second treatment was concluded with the posterior fossa technique, an expansion of the fourth ventricle (EV4) and compression of the fourth ventricle (CV4). The goal of this treatment was to increase the fluidic drive.

The third treatment focused on restoring the cranial axis and increasing vascular flow to the brain. A parietal lift concluded the third treatment to balance the dural membranes. Under the parietal bones lies the sensorimotor cortex. An endocranial pumping technique to the encephalon concluded the third treatment.

The fourth and final cranial osteopathic treatment in the fifth week focused primarily on the encephalon, in particular those areas chiefly responsible for the motor disturbances in Parkinson's disease. A bilateral rocking technique is thought to stimulate the central nuclei and the thalamus and was performed on all the subjects in the experimental group. The endocranial techniques were followed by the lateral ventricle technique. Druelle and Forget (2000) hypothesize that this technique increases the systemic activity of the brain.

The Treator performed a local and regional integration balancing the posterior fossa, and performed a global integration with a corelink after completing the goals of each treatment and the specified specific techniques described above.

On the fifth and final visit, after the osteopathic treatment, each subject was re-assessed using the TUG test and osteopathic evaluation. A summary of the goals and specific techniques are found in Table 3.

Table 3: Summary of Osteopathic Treatment Interventions for Experimental Group

First Osteopathic Treatment Week 2	• Goal: Clearing of serious lesions impeding the craniosacral mechanism • Venous Sinus
Second Treatment Week 3	• Goal: Clear cranial base • Posterior Fossa • CV4 • EV4
Third Treatment Week 4	• Goal: Increase vascular flow and restore cranial axis • Parietal lift
Fourth & Final Treatment Week 5	• Goal: Treatment of endocranium • Bilateral temporal rocking • Lateral ventricles

4.6 DEPENDENT VARIABLES

The dependent variable was the TUG test scores for the subjects' pre- and post-assessment.

4.7 MEASURING TOOL

4.7.1 THE TIMED UP AND GO (TUG) TEST

The TUG test is an objective functional mobility assessment tool. Independent reviews (Brusse, Zimdars, Zalewski, & Steffen, 2005; Morris & Morris, 2001; Smithson, Morris, & Iansek, 1998) concluded that the TUG test is an easily administered, cost-effective tool to measure functional mobility and a falls risk tool that both is reliable and shows high levels of validity. TUG best related to activities of daily living (ADL) of Parkinson's disease patients (Hachiya, Murata, Kumano, Maeda, Nozumi, & Mizokami, 2012)

Test-retest reliability of the TUG has been examined over short intervals and is considered excellent (Brusse, Zimdars, Zalewski, & Steffen, 2005). Testing functional performance in people with Parkinson disease was reported as moderate to excellent. The TUG test is commonly used with the elderly population for a falls risk assessment and also used in studies of people with Parkinson's disease, stroke, and other various balance conditions (Morris & Morris, 2001). It is an inexpensive outcome measure incorporating functional tasks and is easily applied in a clinical setting. It also correlated well with the Berg Balance Scale (BBS) and with the risk in falls (Brusse, Zimdars, Zalewski, & Steffen, 2005). The TUG test measures, in seconds, the time taken by the individual to stand up from a standard armchair with a seat height of 46 centimetres, walk 3 metres, turn around, walk back to the chair, and sit down. A practice trial was given, and then three timed trials were averaged. The TUG test has been used in similar designs looking for improvement in function in persons with Parkinson's disease (Hackney & Earhart, 2008; Hackney, Kantorovich, Levin, & Earhart, 2007). The times were measured using a *PT Fitness* stopwatch, product number 84-0794-0, 1/100th second accuracy (Figure 17).

Ellis et al. (2011) performed a cross-sectional study of 263 people with Parkinson's disease. He concluded that functional tests, like TUG, which test functional actions like sit to stand and walking are the best at determining quality of life. Our goal in this study is to show improvement in mobility in the hope that this will improve quality of life. A description of the TUG test can be found in Appendix F.

Figure 17. PT Fitness stopwatch, product number 84-0794-0, 1/100th second accuracy.

4.7.2 OSTEOPATHIC ASSESSMENT MEASURES

4.7.2.1 SOMATIC DYSFUNCTION SEVERITY SCALE

Each structure was rated on a scale of zero to four with four being the most severe, representing a compaction of the structure being evaluated. Three was reserved for dysfunctions classified as non-physiological lesion without respect to the axis, a rating of two for non-physiological lesions with respect to their axis, one was used for physiological lesions and zero was reserved for structures that present with normal movement. This order of severity was based on the CCO methodology (Appendix E) of lesion severity.

4.7.2.2 VITALITY RATING SCALE

A zero to three vitality rating scale was used to measure the vitality of each of the thirty-eight structures listed in the Osteopathic Assessment Form (Appendix F). The assessing osteopath used palpatory feel to rate all thirty-eight structures. A rating of *zero* indicated no vitality or a complete blockage, and a *three* represented full or normal vitality. The rating of one was given to structures with a major restriction that presented

with very low, but not absent vitality. The rating of two was reserved for structures with only a minor restriction. These structures had some vitality and were closer to full vitality than to absent vitality. This rating scale of vitality has been used in a number of CCO theses with success(Chasczewski, 2010; Marchand, 2008).

4.8 RECRUITMENT METHODOLOGY

Recruitment occurred within the greater Toronto area focused on the York Region area and in particular Thornhill, the location of the research. Advertisements were posted in movement disorder clinics, and physician letters (Appendix G) were sent to local family doctors and movement disorders specialists. Personal contact by email and phone was made with Dr. Gutmann and Dr. Adams, Neurologists specializing in movement disorders at Markham Stouffville Hospital and at Baycrest Hospital. Personal contact was made possible through mutual patient connections, family, and friends who have seen these doctors for their Parkinson's either currently or in the past. After two emails and a brief conversation with Dr. Adams, he informed the researchers that their clinic refers to another physiotherapy clinic, and without an ethics review they did not feel comfortable sending their patients to this study. No potential or actual participants were successfully recruited for this study using this resource.

The physician letters and postings in local medical offices yielded five phone calls but did not result in any qualifying participants. The primary reason given was difficulty getting to the clinic. Doctor offices, hospitals (York Central Hospital, Markham Stouffville Hospital, and North York General and Branson Hospital), seniors' centres, and long-term care facilities were contacted. Business cards (Appendix H) were left for reference and recruitment notices (Appendix I) were posted. No inquiries or participants were obtained from these methods. However, two inquiries were recruited through word

of mouth from discussions with employees at North York General Hospital. One subject was excluded due to dementia and the other was excluded because they were diagnosed with concomitant progressive supranuclear palsy (PSP).

Personal contact was made with the Sutherland-Chan Massage School Parkinson's outreach program, which did not yield any results. Contacting the local chapters of the Parkinson's Association provided email contacts for potential clients, and three participants were recruited in this manner. Both researchers emailed their respective databases of consented patients, friends, and family, to inform them about the study. The patients were asked to pass on the information to anyone that they believed might benefit from this research. Seven inquiries and four subjects were attained from this source.

The majority of inquiries were from direct referral initially, and subsequently from direct referrals from the participants themselves. Feedback from both the control and the experimental groups was positive. A total of 24 patients inquired about the study. Three were excluded due to ambulatory issues that prevented them from completing the TUG test. Three subjects were excluded due to due to a Parkinson's plus diagnosis. One potential participant who met all the criteria did not show up for her initial appointment. Upon follow-up she stated only that she had changed her mind. The remaining five potential subjects were not able to commit to the five consecutive weeks required to complete this study.

The summary chart on the following page lists both the successful and the unsuccessful recruitment methods and the most likely reason for their success or failure.

Table 2: Summary of Recruitment Methods and Their Degree of Success

Recruitment Method	Number of Subjects	Number of Subjects	Possible Reason for Success or Failure

	Inquiring	Recruited	
Movement disorder clinics at Baycrest, MSH	0	0	Unknown
Seniors' centres and hospitals (NYGH, NYBH, YCH, HRRH)	0	0	Lack of knowledge regarding osteopathy, lack of ethics review
Personal contact with Dr. Adams and Dr. Guttman through the author's mother – phone calls and emails	0	0	Possibly due to lack of ethics board or close affiliation with another physical therapy clinic
Personal contact with the Sutherland-Chan Massage School Parkinson's outreach clinic	0	0	Distance to travel to Thornhill
Physician Letters & Posters	2	0	Lack of knowledge regarding cranial therapy and distance to travel, lack of ethics review
Word of Mouth/Direct referral from family, friends	7	5	
Talking to all current patients to ask if they knew anyone with PD that might participate in the study	8	2	Were excluded based on exclusion criteria.
Email to all contacts	6	4	Exclusion criteria
Attrition related to exclusion criteria	0	0	

4.9 TELEPHONE SCREENING

Interested subjects called the clinic number and received a semi-scripted interview by the clinic receptionist (Appendix J), or in some cases the interview was performed in person at Physioactive. The responses to the interview were recorded directly on the same form and reviewed by the researchers to determine whether the potential subject met the initial criteria for the study. The potential candidate was told that their answers would be

evaluated by the researchers and they would be contacted within two days and told whether they qualified for the study or not.

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.

4.10 RANDOMIZATION

The participants were placed in either the control group or experimental group during the initial appointment after the TUG testing and before the osteopathic assessment (See 1.11 Procedure for when this process occurred). Each patient was asked to randomly choose an envelope from a tabletop where the envelopes had been placed an equal distance apart. Each envelope contained either a red or a black card. There was eight of each colour. The cards were placed facing down in envelopes and sealed at the commencement of this research. The black cards represented the experimental group and the red cards the control group. The envelopes were shuffled and placed on the table each time a new subject entered the study.

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 (Treator) and Thomas Hein was to be the Assessor.

After the subject selected the envelope they handed it to the clinic receptionist. The receptionist passed the envelope to the Treator, who then removed the card from the envelope and recorded the participant's data in the data log (Appendix K).

Figure 18. Envelopes for subject randomization

4.11 PROCEDURE

If the participants met the inclusion and exclusion criteria they were deemed appropriate to participate in the study. Once accepted, the qualifying participants in the study were told they would need to commit to five consecutive weeks and should plan clinic visits for five consecutive weeks during a time in which they knew they would not be required to change their medication. They were given the clinic address, told to wear comfortable clothing and walking shoes, and given a date and time for their initial appointment which would last approximately two hours.

The candidate would register with the administrative assistant at Physioactive upon arrival at the clinic and they were asked to fill out an informed consent form (Appendix A) and a medical history questionnaire (Appendix L). Subjects were informed

of the privacy policy (Appendix M), their right to withdraw from the study at any time, and the forfeit of participation if there were any changes that would exclude them from the study.

Once the participant was registered, the administrative assistant introduced the candidate to both researchers of this study by their full names. The study participant was not privy to who the assessing Osteopath was and who was the treating Osteopath. Thomas Hein (Assessor) led the candidate to the gym where the two were seated in equal chairs facing each other to review the paperwork. The privacy policy and the subject's right to withdraw were reviewed again. Any further questions regarding any of the forms were clarified at that time.

The Assessor administered the TUG test. The results of the TUG test were recorded on the TUG recording form (Appendix F).

After the completion of the TUG test the subject was re-introduced to the Treator. The Assessor was sequestered to a treatment room so that there was no way the Treator could witness the selection of the playing card. The subject was asked to randomly pick an envelope from a tabletop, where the envelopes had been placed an equal distance apart. Each envelope contained either a red or a black card, both of which occurred in equal numbers for the total number of subjects. Black represented the experimental group and red represented the control group. The Treator opened the envelope at that time and entered the participant's data into the log (Appendix K). The treating osteopath accompanied the participant to the reception desk, instructed the reception staff to book their follow-up appointments, and gave the subject an appointment form (Appendix N).

The Assessor was not privy to the group assignment information and thus remained blinded.

Regardless of which group was selected by the subject, the Treator led the subject to the private treatment room where the Assessor had been sequestered. The Assessor completed the osteopathic assessment form (Appendix O). This part of the assessment was done after the selection processes to act as a subject-perceived placebo treatment, making it more difficult for the subject to determine which group they were in. In the treatment room the Assessor completed the osteopathic evaluation and then led the subject back to the gym for an introduction to the exercise program. The exercise program was taught and supervised by a licensed physiotherapist. This physiotherapist was also not privy to which group the subjects were part of.

If the subject was assigned to the experimental group, he or she returned in one week for the first osteopathic cranial treatment and also participated in the exercise protocol at that time. All the participants in the experimental group attended three additional osteopathic treatments, one week apart, each approximately 60 minutes in length. These osteopathic treatments occurred in weeks two through five. The experimental group attended a supervised exercise group, once a week during the first four weeks of the study, on the same day as their osteopathic treatment. The treatments were scheduled at the same time of day each week and subjects were instructed to make their best effort to take their medication at the same time each day. The subjects were instructed to let the treating researchers know if there was any alteration in time or dosage of their medication. No changes were reported.

The final assessment for the experimental group occurred following the last osteopathic treatment. The testing process followed the same order as the baseline testing done at week one. The TUG test was first administered by the Assessor, then followed by the osteopathic assessment.

The control group's first appointment was identical to that of the experimental group. After completion of the TUG test, the subject selected a card identifying the participant as a member of the control group. The participant would then be led to the treatment room for their osteopathic assessment. After the assessment the Assessor led the participants back to the gym for their physiotherapy-supervised exercise program. The following three weeks consisted of the same supervised exercise program as the experimental group, but the control group received no osteopathic treatment. The appointments of the exercise sessions were scheduled at one-week intervals after the initial visit at the same time as the initial assessment. On the final visit, the fifth week, the participant was led to the gym by the Physioactive administrator to meet with the Assessor. The TUG test was completed, followed by the osteopathic assessment. The participants were offered a rest and a drink of water if necessary.

It is important to note that the hypothesis under consideration was focused on the benefits of cranial osteopathic treatment, not global osteopathic treatment, in improving mobility in Parkinson's patients. The exercise regimen was included as a publicly recognized treatment and provided a perceived benefit for all participants. The exercise was restricted to a basic and appropriate level for all subjects in the study

Table 3: Summary of intervention during the five week study

	Control	Experimental

Week 1	Initial Assessment and Introduction to Exercise Program	
Week 2	Exercise Program	Exercise Program and Cranial Treatment
Week 3	Exercise Program	Exercise Program and Cranial Treatment
Week 4	Exercise Program	Exercise Program and Cranial Treatment
Week 5	Final Assessment	Cranial Treatment and Final Assessment

4.12 DESCRIPTION OF CLINICAL ENVIRONMENT AND TREATMENT ROOM

This research was held at Physioactive Clinic, 1450 Clark Ave., Thornhill, Ontario.

Figure 19. 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 performed the initial and final TUG test as well as the exercise program.

Figure 20. Gym where exercise protocol was administered

All osteopathic treatments were performed 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.

Figure 21. Treatment room

4.13 ETHICS

In this study, each subject was required to read and sign an informed consent form (Appendix A). This form indicated that the subject was able to terminate their participation at any time, for any reason, without consequence. The subjects were told that they were randomly placed into either a control or an 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 policy (Appendix O) that was compliant with the PIPEDA guidelines. The interventions used were low risk to health, and the inclusion and exclusion criteria were designed to reduce 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

Peter Lewycky P. Eng prepared the statistics for this research. Peter has extensive experience in the preparation of statistics and has prepared statistics for osteopathic research in the past. He 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. The data analyzing the effects of cranial osteopathy on functional mobility in Parkinson's subjects were analyzed using the Kruskal Wallis (KW) Exact. A non-parametric KW was needed because of the outliers that existed in the data. A parametric test such as a t-test would have been preferred as they are more powerful but could not be used in this instance as a result of the statistical outliers. The non-parametric KW test is less powerful and thus typically renders a larger p value.

The results were analyzed to determine the effect of cranial osteopathic treatment using a Timed Up and Go (TUG) test in subjects in the control group versus the experimental group. Both groups saw improvement in the TUG score whether treatment was exercise or exercise plus cranial osteopathic treatment. Improvement was greater in the experimental group receiving osteopathic treatment in conjunction with exercise versus exercise alone. The statistical analysis did not yield a significant result. P=.23 > .05.

Table 3: TUG Scores Pre- and Post-Intervention

				Count	Mean	Std. Dev.	Min.	Max.
Treatment	Control	Period	Pre- TUG	4	11.66	3.94	7.53	16.39
			Post-TUG	4	11.43	3.66	7.82	15.76
	Experimental	Period	Pre- TUG	7	12.85	5.43	9.28	24.70
			Post-TUG	7	11.31	3.91	8.72	19.29

Table 4: Categorical Variable Information

			N	Percent
Factor	Treatment	Control	4	36.4%
		Experimental	7	63.6%
		Total	11	100%
	HoehnYahr	Stage 2	6	54.5%
		Stage 3	5	45.5%
		Total	11	100%

5.1 OSTEOPATHIC EVALUATION ANALYSIS

For information purposes, the stimulation of thought and discussion, and to provide osteopathic insight, the research included a pre- and post-intervention osteopathic assessment looking at the types of lesions and the relative vitality of each structure. The assessor remained blinded to which group, control or experimental, the subjects were part of. The study did not assess the sixteen participants the researchers required to reach statistical significance. This resulted in uneven groups, with the control n=4 and the experimental n=7. The statistical observations are meant to add guidance to the clinical practitioner and to lay the foundation for future study in this area. The tables below show the results of measures of both vitality and cranial lesion classifications.

5.1.1 DATA ANALYSIS OF LESION CLASSIFICATION

Thirty-eight anatomical structures in the craniosacral mechanism were evaluated pre-intervention and post-intervention for both groups. The rating scale for the types of somatic dysfunctions or *lesions* is based on the methodology described at the Canadian College of Osteopathy (Appendix O: Clinical Methodology of the CCO). Each structure was assigned a number from zero to four representing with zero being least severe and four being the most severe. On this scale, zero represents no lesion, one represents a physiological lesion, two represents a non-physiological lesion with respect to the axis,

three represents a non-physiological lesion without respect to the axis, and four represents a compaction. The mean provides an average of severity of lesion per structure within each group.

Due to the unequal sample sizes and subjective measure of cranial assessment the researchers caution the reader not to draw definitive conclusions from this data. Of interest is consistent improvement seen in the lesions primarily in the experimental group. Improvement in the mean classification corresponded well with the improvements seen in the TUG scores.

Table 4: 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 Somers'd test was used to evaluate change in the pre-classification and post-classification lesion scores of both groups. No anatomical structures in the control group showed statistically significant change from pre- to post-intervention scores. This data supports the hypothesis that exercise alone is not enough to make a change to palpable cranial lesions. Conversely, the experimental group had statistically significant

improvement $p<0.05$ in 18 out of thirty-eight structures. Osteopathic treatment and exercise in this study showed a significant improvement in almost half the lesions tested compared to no changes in the control group. Of the twenty remaining that did not reach statistical significance, eight were a result of having a lesion severity rating too low (a pre-test starting value of one or less) to make a significant change. Seven of the 38 structures that didn't reach statistical significance did reach their maximum improvement. Another two structures had a standard deviation greater than one and thus, with their increased variance, those structures were unable to reach statistical significance. Only two of the 38 structures did not improve in lesion severity.

The following table (Table 5) shows the p-value for each structure in both the control and experimental group. The structures that had a statistically significant ($p<0.05$) improvement are highlighted in yellow and marked with an asterisk.

A more detailed analysis is included in Appendix Q.

Table 5: 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*
Petro-basilar Left	P=1.000	P=0.016*
Petro-basilar 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*

* yellow represents statistically significant change $p<.05$

5.1.2 DATA ANALYSIS OF STRUCTURES IN RELATION TO VITALITY

Each of the 38 anatomical structures pre- and post-intervention as measured on a scale of vitality. The vitality scale was measured using scores ranging from zero to three, with zero representing no vitality, one representing a major restriction, two indicating a minor restriction, and three representing normal vitality. The table below shows descriptive statistics for each of the 38 variables on the vitality measure.

Table 6: 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

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 anatomical structures evaluated.

Twenty-nine of the 38 structures showed statistically significant improvements between the pre-vitality and post-vitality scores in the experimental group. Similarly to

the lesion analysis, the control group showed no statistical significance in any structure between the pre-vitality and post-vitality scores. Four structures in the experimental group had a standard deviation greater than one indicating a large starting variance and thus made it more difficult for these structures to reach statistically significant improvement. All thirty-eight structures improved in vitality scoring in the experimental group.

The following table (Table 7) shows is a summary of the p-value for the vitality measure for each structure in both the control and experimental group. The statistical significant changes are highlighted in yellow and an asterisk. More detail is found in Appendix R.

Table 7: Summary of 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

In this pilot study the effects of cranial osteopathy and exercise compared to the effects of exercise alone on Parkinson's patients were measured using the TUG test. In addition, an osteopathic evaluation of anatomical somatic dysfunctions and their respective vitality were measured pre- and post-treatment. The results provide some promising findings, which will help guide future research into the treatment of Parkinson's disease.

6.1 DISCUSSION OF TUG RESULTS

The null hypothesis: *The experimental group with four cranial osteopathy treatments and exercise will show a faster time during the TUG test of function and mobility than the control group using exercise only, with statistical significance of p<.05 or better.* The TUG results indicate that the null hypothesis can be rejected due to a result of p=.23, which is greater than .05. Although these results are not conclusive, there was a trend to improvement that was greater in the experimental group that included both exercise and osteopathic cranial treatment versus the control group that received exercise only. As well, both groups showed improvement in what is considered a progressively deteriorating disease, Parkinson's.

6.1.1 POSSIBLE REASONS FOR NOT REACHING STATISTICAL SIGNIFICANCE

There are a number of possible reasons contributing to the TUG scores not reaching statistical significance. Statistical power is achieved by a combination of change in effect and size of the sample. During the proposal process a power analysis determined that a minimum of 16 participants would be needed, based on an estimated degree of change expected. The size of sample and degree of change are inversely proportional to each other. As a result, a greater degree of improvement was needed to achieve statistical

significance due to the recruiting of only 11 participants, five less than was desired. The degree of change in the TUG scores compared the exercise-only control group with the exercise and cranial osteopathy experimental group. The literature has shown that exercise itself, used in the control group, can result in an improvement in TUG scores. Using a successful intervention in the control group likely diluted the comparative effects of osteopathic treatment. Perhaps a non-exercise group using a sham would have shown a greater between-group difference. Unfortunately a sham is not without effect, particularly in osteopathy. "In the process of providing sham treatment, however, researchers must acknowledge that some therapeutic benefit may occur, thereby reducing the comparative effect of OMT" (Licciardone & Russo, 2006, p.462). The methodology chosen in this study was to offer all groups some beneficial treatments; it was felt that a sham-only treatment group might impact the participation and recruitment and it was thus not included. The inclusion of exercise in the control group provided a blinding to the participants, as they were not aware whether they were in the experimental or the control group. The proven treatment effect of exercise as well as a possible placebo effect could be responsible for eroding the potential effect of the osteopathic cranial therapy and could have resulted in a smaller difference between groups.

Another possible reason for a lack of statistical significance could be related to the uneven distribution of the two groups. Had the study achieved full recruitment of the 16 participants intended, a full randomization of eight in each group would have occurred. This study also contained one outlier in the experimental group that had a significantly higher TUG result than the other subjects in the study. This outlier negatively impacted the statistics, as a less powerful non-parametric method was needed

to interpret the results. This outlier, in the experimental group, did provide an interesting case study: the study participant with the worst functional mobility as measured by the slowest TUG scores improved the most between the pre- and post-intervention. Lastly, the inclusion criteria related to the severity of the disease was either a stage II or stage III on the Hoehn and Yahr scale. Stage II participants included those with more minor or moderate symptoms of the disease. Perhaps inclusion criteria limiting the study to only stage III participants, or those with a more progressed form of the disease, would better support the hypothesis. It is possible that osteopathic cranial treatment could have a greater effect on participants in different stages of Parkinson's disease, measured by the TUG test, as evidenced by the outlier described above. Limiting the stages of Parkinson's disease may limit recruitment, an important factor when considering a full study.

6.1.2 RELEVANCE OF FINDINGS

Though not statistically significant, the improved TUG test results in both the experimental group and the control group are of interest. Both groups showed improvement, and it is the hope that a full study would result in statistically significant gains between groups. The gains seen in the experimental group could also be attributed to the neurological influence, both proprioceptively and posturally, by osteopathic treatment to the craniovertebral region of the neck. This area contains a high concentration of proprioceptors (Richmond & Bakker, 1982; Richmond, Singh, & Corneil, 1999) and has a close interaction between the neck and the vestibular (Mergner, Huber, & Becker, 1997) and visual systems (Mergner, Nasios, Maurer, & Becker, 2001), supporting the theoretical assumption that cervical sensory information is important to maintain postural control during movement. Sensorimotor integration makes it possible to interpret body orientation in space and to maintain postural control during movement

(Mergner et al. 2001; Peterka and Loughlin 2004; Wolpert et al. 1995). This may be another explanation for the results seen in the outlier in the experimental group.

This study contained one outlier from the experimental group that proves to be an interesting case study. This patient was a level III on the Hoehn and Yahr scale, and took the longest to complete the TUG test both pre- and post-intervention. They showed the most improvement in TUG times of all the patients in the study, with an improvement of 5.4 seconds. Although a case study is not level one randomized controlled trial research, this case example of the outlier in the experimental group provides an example of level four literature (Phillips, et al., 2009) that can provide insight into an area of limited literature. Another possibility for the outlier's improvement could be the effect of the exercise alone. This subject could have been more sensitive and better targeted by the control intervention. It is hoped that future research may differentiate the possible cause of this improvement.

With the permission of the CCO Research Committee, this study was changed from a full study to a pilot study. It is important to not minimize the usefulness of a pilot study by focusing purely on the statistical outcomes (Thabane et al., 2010). The primary purpose of a pilot study is to guide future literature (Thabane, et al., 2010). This discussion, in conjunction with the self-critique section, provides useful guidance to complete a full study. It is a justifiable and useful study when the treatment intervention being studied has not been previously supported nor negated in the current body of literature.

6.2 DISCUSSION OF OSTEOPATHIC RESULTS AND JUSTIFICATION

In addition to evaluating the TUG test, important findings were discovered regarding the somatic dysfunctions commonly found in persons with Parkinson's disease.

An osteopathic evaluation of somatic dysfunctions and their respective vitality was measured pre- and post- treatment. Interestingly, the combination of osteopathic treatment and exercise in this study resulted in a statically significant improvement in almost half of the lesions tested, compared to no changes in the control group. In the experimental group, 17 out of 38 structures showed improvement in lesion classification following exercise and osteopathic treatment, and 29 out of 38 structures had statistical improvement in vitality, compared to none in the control. It was not just the statistically significant lesions that improved in the experimental group.

While the focus of the pilot study was primarily on the objective TUG test measure, these subjective results are of particular interest because they help support the osteopathic theory that manual manipulation can improve movement of structures. Within osteopathic philosophy, this should correspond to improved health. It also adds to the osteopathic body of knowledge of cranial osteopathy and Parkinson's disease.

The most common somatic dysfunctions present in the participants were the left nasal bone, the sphenobasilar symphysis, the left petro-basilar suture, and the left C0-C1. All but the nasal bone are found in the cranial base. The cranial base is important to the osteopathic practitioner because it houses the foramina where most nerves and blood vessels enter and exit the cranium. C0-C1, petro-basilar suture, and the sphenobasilar symphysis have direct relationships to the vagus nerve and thus may impact its function. Vagus involvement and possible autonomic dysfunction may relate to orthostatic hypotension (Pearson, Gatti, Sahibzada, Massari, & Gillis, 2011), common in Parkinson's disease (Alegre-Abarrategui, Ansorge, Esiri, & Wade-Martins, 2008), and possibly contribute to the perfusion loss seen in the damaged areas of the Parkinsonian brain. The

jugular foramen and foramen magnum are located in the cranial base. The venous drainage of the brain and spinal cord exit the foramen magnum. The arterial supply to the basal ganglia and brainstem base enter through the foramen magnum (the vertebral system) and the carotid arteries enter through the temporal bone. The circulation of the cerebrospinal fluid enters and exits at the foramen magnum.

The most common structures affected were primarily on the left side of the body. A possible reason for these disproportionate left-sided findings may be related to the lymphatic drainage channels of the body. The thoracic duct, which drains the legs, trunk, left arm, and head, may be more susceptible to venous insufficiency, and any impact on the body's lymphatic function would have a greater impact on the left side of the head than the right arm and head, which are drained solely into the right subclavian vein (Gray, Williams, & Bannister, 1995).

One possibility as to how the improvement in lesion classification and vitality was obtained could be via normalizing autonomic function through osteopathic treatment. This would provide increased circulation and venous drainage. This, in turn, would provide a more optimal environment for cellular health, reduction of inflammation, and removal of toxins from the basal ganglia and other areas of the brain. Interestingly, Hirsch (2007) found that poor venous return or lack of toxin removal may contribute to the ongoing destruction of the basal ganglia in Parkinson's disease. These findings suggest that improving circulation or venous return may slow down the destruction of the basal ganglia. Hirsch's (2007) findings, in conjunction with the improved lesion classification and vitality results in this study, open the door for future research on the osteopathic treatment of Parkinson's disease.

While the results of the osteopathic assessment are interesting and contribute to a body of osteopathic literature related to Parkinson's disease that is currently lacking, there are limits to the conclusions that can be drawn from these findings. First, there is insufficient literature to strongly support the reliability of a palpation exam, and second, the results are based on one Assessor's interpretation and therefore would require repeated studies to determine validity and inter-rater reliability. It is hoped that these findings can help guide future research.

6.3 DISCUSSION OF INTERVENTIONS

Exercise is considered to be the gold standard in non-pharmacological treatment of Parkinson's disease. The exercise protocol used in this study was recommended by the Parkinson's Society of Canada and was easily accessible for the study and the subjects. The Parkinson's exercise protocol was placed in this study both as a reasonable control and as an incentive for subject recruitment. The study design allowed for all subjects to have some intervention without confounding the results of cranial osteopathic treatment.

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 an exercise regime in conjunction with some type of manual intervention, in the case of this study an osteopathic evaluation at the start and end of the study, provided a means of preventing the subjects from knowing to which group they were assigned.

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

One participant in the study complained of increased neck pain as the study progressed and was fearful of having their neck evaluated in the final osteopathic assessment. This subject was in the control group and the researchers were unaware of this complaint until the final osteopathic evaluation in week five.

6.4 DISCUSSION RELATED TO LITERATURE REVIEW

There is insufficient literature to support cranial osteopathy in the treatment of Parkinson's disease in relation to gait or mobility. This lack of literature at the proposal time meant there was little guidance for developing a methodological plan based on successful past studies. One purpose of a pilot study such as this is to refine the methodology and ensure the study is safe and effective (Thabane, et al., 2010). 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, such as multiple sclerosis and Alzheimer's, and childhood neurodevelopment, with the intention of using these studies to help explain possible outcomes to the interventions in this study.

In order to substantiate the need for this pilot study, research in the validity of cranial osteopathy was discussed. There appears to be conflicting evidence (Green, Martin, Bassett, & Kazanjian, 1999) to support the therapeutic claims of cranial therapy, but there is enough good research to support the mechanics involved in cranial osteopathy. This includes the movement of the cranial bones, the fluctuation of CSF, and the fact that manual intervention can have an impact on cranial structure and its the underlying tissue.

Evidence-based practice dictates that the combination of the best available literature with clinical expertise and experience will result in the best available treatment. While the literature was very limited, it is hoped that this review of cranial osteopathy and Parkinson's disease stimulates discussion, prompts new research, and provides some insight on which future research can be built, especially since Parkinson's disease is a complex disease and current available interventions are insufficient.

6.5 POSSIBLE CONFOUNDS

Several subjects had additional comorbid diseases that may have impacted their mobility as measured by the TUG test. Two subjects, both in the experimental group, self-reported osteoarthritis of the hip, and one reported having bilateral knee replacements for prior severe osteoarthritis. In the control group one subject reported suffering from pain and stiffness as a result of lumbar degenerative disc disease. Another subject within the control group had a previous pelvic fracture causing periodic and recurrent pelvic pain.

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

One of the subjects within the control group was fearful of having anyone touch his neck as a result of a negative treatment experience he had in the past. He refused to have the post-osteopathic intervention unless it was completed without touching his neck.

6.6 THE VALUE OF A PILOT STUDY

Unfortunately the initial intent to produce a full study on cranial osteopathy and Parkinson's disease was unattainable due to lack of recruitment. With the support of the

thesis advisor and the permission of the CCO Research Committee, the study and title were changed to become a pilot study (Appendix S). Permission was granted on March 31, 2011. Two inquiries were made after this date. One potential participant did not qualify as a result of a Parkinson's plus diagnosis and the second potential participant did not call back after their initial inquiry.

Pilot studies are frequently used in the healthcare research (Lancaster, Dodd, & Williamson, 2004), and in osteopathy (Duncan, 2007; Duval, Lafontaine, Hebert, Leroux, Panisset, & Boucher, 2002; Halma, Degenhardt, Snider, Johnson, Flaim, & Bradshaw, 2008; Hundscheid, Pepels, Engels, & Loffeld, 2007; Noll, Degenhardt, Stuart, Werden, McGovern, & Johnson, 2004; Sandhouse, et al., 2010), and are often used as a precursor to a larger study (Lancaster, Dodd, & Williamson, 2004). They help determine whether a larger study is merited. The results of this study support the need for a full study and can provide a solid basis upon which a future study can be built.

Specifically, the improvements measured using the TUG test suggest that the intervention of cranial osteopathy and exercise used in this study may be useful. A larger sample size may show a statistically significant result if repeated. The success of the blinding, the null attrition, and overall satisfaction of the subject suggest that the study set-up would be worth repeating in a larger scale study. The positive changes of the lesions and vitality scores from the experimental group also provide some evidence of the validity of palpatory findings made by the hands of the therapist. Although subjective, a future study showing similar findings would continue to add to the significance of these subjective osteopathic findings. The changes in pre- and post- intervention assessments are also suggestive of positive mobility and vitality changes possible with cranial

treatment. The researchers received positive feedback regarding this study from all the participants. This, and the fact that participants referred other Parkinson's patients from their support groups to the study, suggests that the client satisfaction was well received and suggests that future studies based on this protocol should also be well received by future subjects. Leveraging this pilot study, the self-critique section, and recommendations provided, a future researcher has a good foundation from which to create a useful study of cranial osteopathic treatment for those with Parkinson's disease.

7 SELF –CRITIQUE

7.1 CRITIQUE OF METHODOLOGY

The methodology of this study was both well organized and easy for a future researcher to implement. The Assessor and participants were kept blinded. The participants were efficiently and accurately screened for their inclusion. The study was executed seamlessly and the positive feedback and null attrition is an indication of how positively it was perceived by the participating subjects. A future study would do well to follow the general outline of this pilot study and include the modifications suggested in this section to organize a full study. The hypothesis seems to be worthy of a full study and preliminary evidence requires a full sample study to be conclusive. Improvements in a future study can be made from the experience gained in this pilot study.

The randomization process itself was satisfactory. The inequality in group sizes which occurred in this study were not a result of poor randomization, but rather a result of not achieving full recruitment. The full recruitment of 16 participants was not achieved despite rigorous attempts and this caused a late change to a pilot study with the approval of the CCO Research Committee (Appendix S). This late change resulted in a sudden change in required recruitment and led to this inequality.

The inclusion and exclusion criteria provided appropriate subjects for our study. The pre-screening process eliminated those inappropriate for the study in a timely and cost-effective way. Small changes to expand the inclusion and exclusion criteria of this study could have helped with subject recruitment. In this study the subject pool was limited to those with a diagnosis of idiopathic Parkinson's disease. This does not appear necessary. Including all Parkinson's diagnosis could potentially have helped in recruitment, as it would increase the available number of eligible participants. The greater

the sample size the smaller measureable improvement needed to reach statistical significance. Secondly, using inclusion criteria of a Hoehn and Yahr scale score of II to IV did not always limit the subject pool to those with significant mobility and balance deficits. Many of the participants in our study completed the TUG test close to normal values. A newer revised version of the Hoehn and Yahr Scale (Goetz et al., 2004) with more stage categories would be recommended for future research. The Hoehn and Yahr is the most commonly used scale to describe the severity of Parkinson's disease (Goetz, et al., 2004) and all the studies reviewed during the proposal process used the standard Hoehn and Yahr. This newer version is currently being designed (Goetz, et al., 2004).

Table 8: Hoehn and Yahr scale (Goetz et al., 2004, p. 1021)

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

In the modified Hoehn and Yahr scale below, the addition of two, extra half stages between stages one and three would more precisely identify the pre-testing capabilities of the subjects. However, another possible study in the treatment of Parkinson's disease could include the early Hoehn and Yahr stages, but a more sensitive outcome measure would be required to detect a significant change.

Table 9: Modified Hoehn and Yahr scale (Goetz et al., 2004, p. 1021)

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

It is recommended that for future research the modified version of the Hoehn and Yahr be employed.

7.2 RECRUITMENT

During the study design process the researchers believed that with a good recruitment plan in place and a network of contacts, prior patients treated with Parkinson's, the recruitment of the sixteen subjects needed for a statistical outcome would be achieved.

Unfortunately, only eleven subjects were recruited and the study did not reach statistical power and was changed to a pilot study. All of the clients recruited were from personal contact or a direct referral from a participant. No subjects were recruited by advertising, contact with family physicians, posters or business card distribution.

This author would recommend having the support from a movement disorder clinic or a neurologist specializing in Parkinson's disease. The prevalence of Parkinson's disease was not the limiting factor but rather the access or direct support from health

professionals was needed for population-targeted recruitment. Some movement disorder clinics contacted stated a lack ethics review limited their support while others did not give a reason for not referring to the study. Other movement disorder clinics already referred to physiotherapy clinics in the area for therapy and felt the study was designed for marketing reasons or financial gain.

If the authors had set out to perform a pilot study the experimental versus control group sizes could have been better allocated. This area has such little research perhaps a pilot study is a good starting point that future research can build from. There were no such pilot studies available prior to the commencement of this paper. Only 11 subjects were recruited and thus no statistical conclusions can be drawn from this study. However, the trends showed improvement in both groups but a greater improvement was shown in the experimental group.

7.3 CRITIQUE OF INCLUSION/EXCLUSION CRITERIA

It is the opinion of this author that a diverse disease such as Parkinson's disease has a large variance of underlying pathological cause. The literature review revealed many possible-contributing factors to the development of the Parkinson's disease. It makes the most sense to use a treatment most likely directed to individual etiologies in order to have the most profound treatment effect.

This pilot study was assessing the effects of cranial osteopathic treatment specifically. Thus, it makes sense that to really assess the effects of treatment to the craniosacral mechanism, the subjects should have a significant cranial lesion to be included in the study. A Parkinson's patient may present early in the disease with few symptomatic Parkinsonian findings but could have severe cranial lesions. It could be hypothesized that this patient may benefit more from this treatment intervention when

compared to a Parkinson's patient with no or minor cranial lesions. A recommendation for future research would be to include cranial base lesions classified as a compaction or non – physiological lesions within the inclusion criteria. Secondly it seems unnecessary to include age limits. The symptoms and progression of early onset Parkinson's disease do not differ from that of later onset Parkinson's disease (Martin, Suchowersky, Kovacs Burns, & Jonsson, 2010). Removal of age limits should improve recruitment.

With the complexity of Parkinson's disease, and the still unknown cause for the neuronal cell destruction, cranial osteopathy may prove to be one piece of the large puzzle in the treatment of Parkinson's disease. The osteopathic principles do seem to be applicable and it is the hope of this author that future time and energy are put into research in this area.

7.4 CRITIQUE OF OUTCOME MEASURES

During the initial literature review during the proposal stage to determine an valid and reliable measure of mobility in Parkinson's disease it was determined that the TUG was an appropriate and sensitive test for use in a Parkinson's study measuring functional mobility. Upon completion of the study, it became evident that that some of the participants were in very early stages of the disease. However, a more recent study by Zampieri et al. (2010) found that the TUG test was not sensitive enough an outcome measure to discern a change in higher functioning subjects with Parkinson's disease. It is possible that a similar result occurred in this study as many of the subjects in this study were very high functioning. This 2010 study was not available at the proposal period of this study. A recommendation for future studies studying functional mobility in Parkinson's disease would be to use a more sensitive test such as the instrumented timed up and go (iTUG) test. It looks at other characteristics such as arm swing, turning

velocity, and cadence, rather than only the timed value done in the traditional TUG test. Zampieri et al. (2010) determined that the constructs in the iTUG test were more correlative with the unified Parkinson's disease rating scale (UPDRS) than the TUG test in mild Parkinson's disease. This suggests that the iTUG test is a better measure of functional mobility. It is the suggestion of this author that if the TUG test is used as an outcome measure only later stage Hoehn and Yahr scale patients be included in the study. A more sensitive outcome measure such as the iTUG should be used in early stage participants if a specific Parkinson's disease population is studied.

If resources were unlimited, assessing the perfusion changes to the Parkinson's brain would be an ideal physiological measure and could possible have a greater impact in the earlier stages of the disease. Symptom change at this stage would be more difficult to measure due to the lack of symptomatology. However, by measuring perfusion an assessment of Parkinson's prevention could be objectively measured.

7.5 RECOMMENDATIONS FOR FUTURE RESEARCH

Suggestions for future research based on the current pilot study.

1. Repeat the current study with minor modifications and a full sample size.
2. Consider using seasoned osteopaths rather than student practitioners.
3. Consider increasing treatments from four to six or more visits.
4. Obtain prior support from a movement disorders clinic or neurologist that assesses and treats Parkinson's patients
5. Future researchers could go through the process of a formal ethics review, which could assist physicians and Parkinson's society based subject recruitment
6. Adding a quality of life questionnaire to gather information regarding the effects of treatment on depression, sleep and the general well-being of the subjects.

7. Increase the number of cranial treatments from four to six or eight to determine whether this would increase the effect of treatment.
8. Add a one and/or three month follow-up assessment to assess the long-term effects or retention of treatment effects of cranial osteopathic treatment in Parkinson's subjects.
9. Possibly adding a sham treatment for the control group to discern possible placebo effect.
10. If possible, get access to susceptibility weighted imaging (SWI) MRI. If not, other measuring devices such as functional MRI, PET scan, SPECT scans or other devices able to detect small changes in perfusion in the Parkinson's patient would provide definitive, measurable changes as a result of cranial osteopathic treatment Perfusion changes could be measured in both in the basal ganglia and other areas of the brain. Due to cost this type of study would need collaboration with a facility that could perform and help fund these tests.
11. Having a third group receiving only a sham with no exercise or cranial osteopathic intervention. In this study both groups improved. The improvements seen in the control group may have eroded the differences between groups, and thus, made if more difficult to attain a statistically significant difference.
12. Complete in an inpatient basis. The researchers found that for a Parkinson's patient to commit to coming to an outpatient clinic on 5 consecutive weeks was difficult. This produces a sample bias as it was more likely for a higher functioning Parkinson's patient to participate than a more functionally limited subject.

8 CONCLUSIONS

Parkinson's disease afflicts primarily seniors, of which two-thirds are likely to fall, resulting in a hospital admission. Parkinson's disease will become a growing concern both financially and socially in the coming years as the population of Canada ages. Parkinson's disease is an important public health issue in Canada. It is estimated that one in every one hundred people over the age of 65 (De Lau & Breteler, 2006) are currently diagnosed with this condition. The number of Canadians over the age of 65 is expected to double in the next 25 years (Statistics Canada, 2010). There is no known cure for Parkinson's disease and the etiology is still not fully understood.

The gold standard of treatment for Parkinson's disease is the drug levodopa. Unfortunately, there are side effects of long-term use, and levodopa does not slow or stop the progression of the disease, but rather minimizes the symptoms of dopamine loss. Traditional paramedical treatment of Parkinson's disease has been directed at combating the symptoms of loss of balance, progressing rigidity and slowness of movement, and altered posture, rather than the underlying cause of these symptoms. This study attempted to approach the treatment of Parkinson's disease from its neurodegenerative cause.

Cranial osteopathy is a commonly practised treatment modality. It is a safe and non-invasive therapy that is well received by patients. By applying the four principles of osteopathy, the author hoped to show that cranial osteopathic treatment would improve the neurological function and mobility of those with Parkinson's disease. Current literature using cranial osteopathy for the treatment of Parkinson's disease is limited, but the results of this pilot study and a review of current theories of Parkinson's etiology demonstrate that it is an area where further study is warranted. Best practices guidelines

for cranial osteopathic treatment in the literature provide only case study support, at best, for its use.

This study was designed to assess the effects of cranial osteopathic treatment on mobility in subjects diagnosed with idiopathic Parkinson's disease. It was changed to a pilot study at the eleventh hour due to insufficient sample size, despite rigorous recruitment. 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.

A power analysis based on four equally spaced cranial osteopathic treatments determined that a power of n=16 was needed to show statistical significance ($p<0.05$). The lack of recruitment affected the statistical power and resulted in seven subjects randomly assigned to the experimental group and four subjects randomly assigned to the control group.

The experimental group receiving cranial therapy and exercise improved by a mean of 1.5 seconds measured by the TUG test, compared to .22 seconds in the control group receiving exercise alone. Although the TUG measures did not reach statistical significance ($p=.23$), the trend to improvement and sound research methodology used in this study merits a full study. In addition to the TUG scores, an osteopathic evaluation determined that 17 of 38 somatic structures measured pre-treatment and post-treatment on the lesion severity scale improved. In the vitality measures, 29 of 38 structures showed statistically significant improvement in the experimental group $p<.05$). There was no

change in the control group for either measure. It should be noted that the osteopathic evaluation was a subjective measure and the reader should recognize its limitations.

Cranial osteopathy is a widely used modality within osteopathy for the treatment of many illnesses, but the available research relating to cranial osteopathy and Parkinson's disease is very limited. More research is needed to demonstrate the clinical efficacy of cranial osteopathic treatment with specific focus in neurological disorders. Advancements in imaging may become more economically feasible in the future to help support the use of cranial osteopathy and its physiological effects on cerebral perfusion and the central nervous system.

It is the hope of this author that a full study is completed based on this pilot study. The research methodology and recommendations outlined herein provide a foundation from which a full study can be designed. Although no statistical conclusions can be drawn from this pilot study, the trend toward improvement in the TUG test and the thought-provoking results from the osteopathic evaluation suggest a full study is merited.

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APPENDICES

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

Contact Information for the Canadian College of Osteopathy:

Phone: 416-597-0367

APPENDIX B: HOEHN AND YAHR SCALE

Hoehn and Yahr staging of Parkinson's disease (Goetz, et al., 2004):

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 PROTOCOL (PARKINSON SOCIETY CANADA, 2008)

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.

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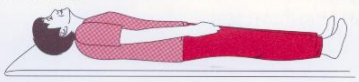

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2) When you wake each morning, lie flat on your back, with just enough support to keep your head and neck from tipping back for 5 minutes. Do not press your shoulders or head back into the bed. RELAX! Allow gravity to stretch you as straight as possible.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

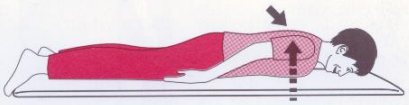
3) Every time you sit in a chair, make your shoulder blades touch the back of the chair. Hold for a few seconds. Do this three times, each time you sit down.

✔ correct

✘ incorrect

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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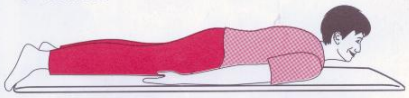

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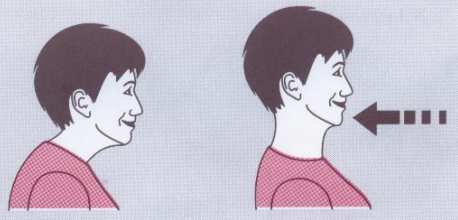
5) Lie face down on the bed or on a mat on the floor with your arms beside you. Gently pull your shoulder blades together. Keeping your head and neck in a straight line lift slightly. Hold for a few seconds. DO NOT LIFT WITH YOUR LOWER BACK.

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

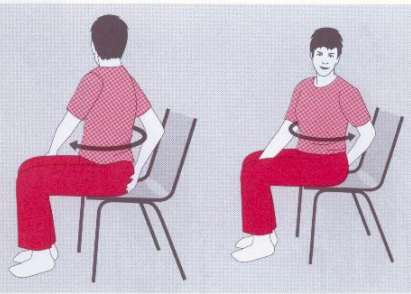
FLEXIBILITY OR STRETCHING

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.

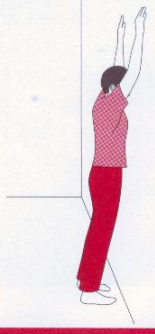
1) Sit tall on the edge of your seat. Turn your shoulders to the right. Reach your right hand behind you and stretch it towards your left hip. Turn your head and body as well. Relax your muscles and hold that position for at least ten seconds. STAY TALL! Repeat the other way.



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.

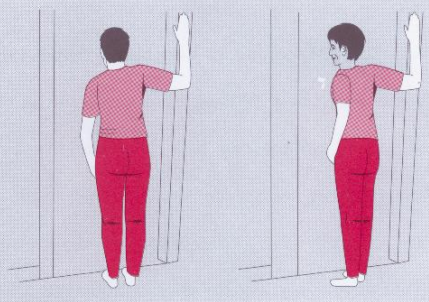
OR

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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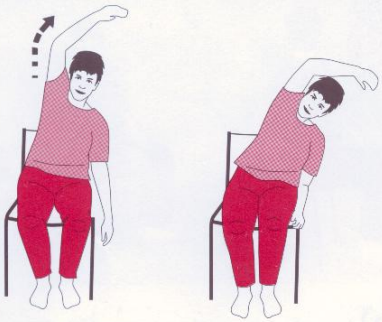

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4) Sit tall in a chair. With your right arm curved over your head, slowly reach for the opposite wall. Do not tip your body. DO NOT BEND FORWARD. Feel a stretch in your right side muscles. Hold for at least ten seconds. Repeat the other way.

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.

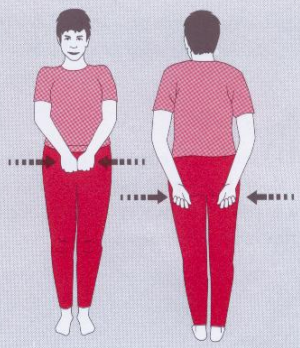
All of the following exercises may not be right for you. Choose the ones you feel safe doing.

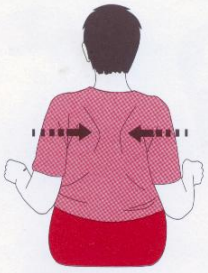
Some people like to do all of their exercises at one time while others prefer doing their exercises for short periods at different times during the day. Either way is correct.

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.

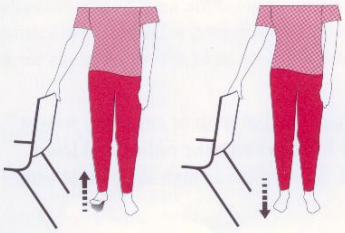




2) Sit or stand tall. Keep elbows at ninety degrees. Pull shoulder blades together in back.

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: TECHNIQUE DESCRIPTIONS

The specific techniques used on each participant were dependent on need. The goals of each of the four treatment sessions are outlined all the techniques used during the treatment of the participants are listed below. Five specific techniques were performed on all subjects of the experimental group. They were

1. The Venous Sinus Technique (Druelle & Forget, 2000)
2. Compression of the Fourth Ventricle (CV4) (Colford & Pelletier, 2000)
3. Parietal Lift (Colford & Pelletier, 2000)
4. Bilateral Rocking of the Temporal Bones (Druelle & Forget, 2000)
5. Lateral Ventricles (Druelle & Forget, 2000)

All subjects received these five treatment techniques but no subject in the experimental group received all the treatments listed below. This study primarily used osteopathic techniques taught at the Toronto location of the Canadian College of Osteopathy. Treatments were individualized for each subject with the exception of one or two techniques based on the goals of each of the four cranial osteopathic treatments described in the methodology.

Techniques were considered contraindicated if they were unacceptably uncomfortable to subjects, they were medically contraindicated, or the subject did not give verbal or nonverbal consent. Nonverbal lack of consent included obvious physical or psychological discomfort with the technique or positioning despite verbal consent. For example, one client was very apprehensive if any pressure contact was made with their neck on the post-assessment. The Treator in this study, Stacey Hauserman, provided the following list.

General explanations of the styles of the osteopathic techniques used and how they are believed to work are well described in the literature. Texts by Magoun (Magoun, 1976), and Upledger and Vredegoogd (Upledger & Vredegoogd, 1983) describe techniques used in cranial osteopathy extensively.

The references for these techniques are primarily unpublished course materials from the Toronto campus of the CCO.

Cranial Techniques

- Normalization of the frontal and greater wings of the sphenoid (LaFlamme & Lanthier, 2000)
- Normalization of the anterior and posterior sphere (Druelle, Evans, & Forget, 2000)
- Normalization of the endocranial membranes (Druelle & Forget, 2000)
- Normalization of the posterior fossa (Druelle, Evans, & Forget, 2000)
- Opposite physiological motion of the occipito-mastoid suture (Colford, Lulic, & Muzzi, 2000)
- Normalization of spheno-petrous (Druelle, Evans, & Forget, 2000)
- Temporal normalization (Forget, Laett, Lanthier, & Van Vliet, 2004-5)
- Bilateral rocking of the temporal bones (Druelle & Forget, 2000)
- Sphenoid vertical strain release (Magoun, 1976)
- Sphenoid lateral strain release (Magoun, 1976)
- Lateral ventricles (Druelle & Forget, 2000)
- Normalization of petro-basilar (Druelle, Evans, & Forget, 2000)
- Sphenobasilar normalizations (Beaulieu, Muzzi, & LaFlamme, 2000)
- Sphenobasilar decompaction (Beaulieu, Muzzi, & LaFlamme, 2000)
- Muscle Energy C0/C1 (Druelle, Evans, & Forget, 2000)

- C0 - C1 - C2 normalization (Druelle, Evans, & Forget, 2000)
- Decompaction of C0 C1(Druelle, Evans, & Forget, 2000)
- Decompression of base (Druelle, Evans, & Forget, 2000)
- Normalization of posterior fossa (EV4) (Druelle & Forget, 2000)
- Normalization (indirect technique) for parietal bone (Colford & Pelletier, 2000)
- Anterior-posterior cranial sphere normalization (Colford, Forget, Laett, Lanthier, & Van Vliet, 2000)
- Normalization for compaction of ethmoid/sphenoid (LaFlamme & Lanthier, 2000)
- Normalization for compaction of ethmoid/frontal (LaFlamme & Lanthier, 2000)
- Normalization for compaction of vomer/sphenoid (LaFlamme & Lanthier, 2000)
- Normalization for compaction of vomer/ethmoid (LaFlamme & Lanthier, 2000)
- Normalization for compaction of the bones of the nasion (LaFlamme & Lanthier, 2000)
- Normalization for compaction of zygoma/temporal process of temporal bone (LaFlamme & Lanthier, 2000)
- Zygoma lift technique (LaFlamme & Lanthier, 2000)
- Temporal normalization (Forget, Laett, Lanthier, & Van Vliet, 2004-5)
- SBS decompaction technique (Beaulieu, Muzzi, & LaFlamme, 2000)
- Basal Expansion (Laflamme, 2004)

Cervical Techniques

- Muscle energy techniques for C/S ARS lesions (C2 to C7) (Colford, Lulic, & Muzzi, 2000)
- Normalization of RS lesion cervical spine (Colford, Lulic, & Muzzi, 2000)

Pelvic Techniques

- Release of the ilia and sacroiliac joint (Beaulieu, Muzzi, & LaFlamme, 2000)
- Functional correction of sacral physiological sacral torsions (LaFlamme & Lanthier, 2000)
- Functional correction of non-physiological sacral torsions (LaFlamme & Lanthier, 2000)
- External decompaction of the sacrum/coccyx (Beaulieu, Muzzi, & LaFlamme, 2000)
- Decompaction technique of the sacrum and L5 (LaFlamme & Lanthier, 2000)

Other Techniques

- Three diaphragm technique (Colford, Forget, Laett, Lanthier, & Van Vliet, 2000)
- Carotid Artery release (Druelle, Evans, & Forget, 2000)
- Release of Jugular Vein (Druelle, Evans, & Forget, 2000)
- Corelink (Beaulieu, Muzzi, & LaFlamme, 2000)
- Venous sinus technique (Colford & Pelletier, 2000)

APPENDIX E: CANADIAN COLLEGE OF OSTEOPATHY METHODOLOGY

The Canadian College of Osteopathy classifies lesions based on severity their likelihood of having a greater impact on the patient. It is used to guide treatment and are listed below in their order of priority.

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. Physiological lesions

The priority can be further determined by the vitality of each tissue assessed, which often correlates with the severity of classification. Vitality is a term used in osteopathy to describe the volume of life within the tissue . A living cell undergoes cellular respiration where each individual cell takes in nutrition, produces energy in the mitochondria, and then excretes its waste. This fluidic and mechanical respiration of each cell and combined for each tissue can be subjectively quantified. This expansion and contraction is consistent with the health of the tissue and is its vitality. The vitality of a local tissue may or may not be expressed throughout the rest of the body.

The order of priority does not always determine the order of treatment. The osteopath may choose a different order of treatment based on their clinical judgment to prepare, integrate or more effectively access

APPENDIX F: TIMED UP AND GO TEST RECORDING FORM

The test was performed in the hallway of the clinic. A standard chair 46 centimetres in height with armrests was placed a set distance of three meters away from a mark on the floor made with tape.

The researcher explained the test to the participant. The participant will have one practice session and then will repeat the test 3 more times. The timing starts once the researcher says "Start". The stopwatch is stopped once the participant is once again sitting on the chair on which they began. The subject walks alone but may use assistive devices (cane, walker, etc. if they normally use one) if necessary. The interval is timed using a stopwatch/interval timer. The Timer does not talk to or walk with the subject during the test. Only one subject is tested at a time. Measurement is taken in seconds with a stopwatch.

Results are recorded on a coded form (a different form for each assessment) and sealed in an envelope until the study period is over.

Simple, standardized instructions were used:

"When I say 'Start' you are to get up off the chair, walk to the tape, turn around, return to the seat and sit back down. The clock will start when I say "start" and the test ends when you have retuned to a seated position."

Sample recording form:

Participant #	Practice	Trial 1	Trial 2	Trial 3	Average

Subject Code: _____ Test: Pre-study _____ Post Study _____

Time to Complete: _____

APPENDIX G: 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 H: BUSINESS CARD

APPENDIX I: 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 J: 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.

APPENDIX K: DATA LOG

Patient Number	Control	Experimental
1		x
2	x	
3	x	
4		x
5		x
6		x
7		x
8	x	
9	x	
10		x
11		x
12	Unable to recruit	
13	Unable to recruit	
14	Unable to recruit	
15	Unable to recruit	
16		Unable to recruit

APPENDIX L: 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 M: 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 416-597-0367

Thank you,

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

APPENDIX N: 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 Thomas Hein at 905-695-0371 or tomphysio@gmail.com.

Thank you for your participation.

APPENDIX O: 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 P: STATISTICIAN LETTER

February 5, 2012

To Whom It May Concern,

I was the Statistician for the research thesis: 'Osteopathic Treatment for Mobility in Parkinson's Disease Patients' authored by Thomas Hein.

All work was done in a professional manner using appropriate statistical techniques.

Regards,

Peter Lewycky

B.Sc., M.Eng., P.Eng. (ret.)

APPENDIX Q: LESION CLASSIFICATION CORRELATION TABLES

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 the control group there is no statistically significant relationship between pre and post and the distribution of sphenobasilar symphysis (SBS) scores; Exact significance, p= 0.771. For the experimental group there was 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 the control group there is no statistically significant relationship between pre and post and the distribution of left occipito-mastoid suture (OM) scores; Exact significance, p= 0.821. For the experimental group there was a statistically significant difference in the distribution of left OM 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 the control group there is no statistically significant relationship between pre and post and the distribution of right occipito-mastoid (OM); Exact significance, p= 0.771. For the experimental group there was a statistically significant difference in the distribution of right OM scores between pre and post; Exact significance, p= 0.003.

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 the control group there is no statistically significant relationship between pre and post and the distribution of the left petrous basilar junction (Pet – Bas) scores; Exact significance, p= 1.000. For the experimental group there was a statistically significant difference in the distribution of left Pet-Bas 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 the control group there is no statistically significant relationship between pre and post and the distribution of right petrous basilar symphysis (pet-bas) scores; Exact significance, p= 1.000. For the experimental group there was a statistically significant difference in the distribution of right pet-bas scores between pre and post; Exact significance, 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 the control group there is no statistically significant relationship between pre and post left C0-C1 scores; Exact significance, p= 0.829. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post right C0-C1 scores; Exact significance, p= 0.743. For the experimental group there was no statistically significant difference between pre and post; Exact significance, p= 0462.

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 the control group there is no statistically significant relationship between pre and post left C0-C1 on C2 scores; Exact significance, p= 1.000. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post ethmoid scores; Exact significance, p= 0.657. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post left jugular foramen scores; Exact significance, p= 0.657. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post right jugular foramen scores; Exact significance, p= 1.000. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post foramen magnum scores; Exact significance, p= 0.829. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post left parietal bone scores; Exact significance, p= 1.000. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post right parietal bone scores; Exact significance, p= 0.486. For the experimental group there was no statistically significant difference 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
			T L 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
			T 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 the control group there is no statistically significant relationship between pre and post left temporal bone scores; Exact significance, p= 1.000. For the experimental group there was no statistically significant difference between pre and post; Exact significance, p= 0.315.

Crosstab

Count

Treatment			TB R 0	TB R 1	TB R 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 the control group there is no statistically significant relationship between pre and post right temporal bone scores; Exact significance, p= 0.771. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post left frontal bone scores; Exact significance, p= 1.000. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post right frontal bone scores; Exact significance, p= 0.657. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post occipital squama scores; Exact significance, p= 0.771. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post greater wing of the sphenoid scores; Exact significance, p= 1.000. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post left zygoma scores; Exact significance, p= 1.000. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post right zygoma scores; Exact significance, p= 1.000. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post left maxilla scores; Exact significance, p= 0.200. For the experimental group there was no statistically significant difference between pre and post; Exact significance, p= 0.478. This was one of only a few scores that seemed to show greater improvement with the control group, though both were not statistically significant.

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 the control group there is no statistically significant relationship between pre and post right maxilla scores; Exact significance, p= 0.314. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post left mandibular scores; Exact significance, p= 0.486. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post right mandibular scores; Exact significance, p= 0.829. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post left palantine scores; Exact significance, p= 0.429. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post right palantine scores; Exact significance, p= 1.000. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post vomer scores; Exact significance, p= 0.571. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post left lacrimal scores; Exact significance, p= 0.400. For the experimental group there was no statistically significant difference between pre and post; Exact significance, p= 0.592. After completion of the study and the treatment reviewed of the subjects, it was determined that the treating osteopathic practitioner did not treat the lacrimals.

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 the control group there is no statistically significant relationship between pre and post right lacrimal scores; Exact significance, p= 1.000. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post left nasion scores; Exact significance, p= 0.429. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post right nasion scores; Exact significance, p= 1.000. For the experimental group there was no statistically significant difference between pre and post; Exact significance, p= 0.192.

Crosstab

Count

Treatment			Sacrum			
			0	1	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 the control group there is no statistically significant relationship between pre and post sacrum scores; Exact significance, p= 0.771. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post L5-S1 scores; Exact significance, p= 0.971. For the experimental group there was also no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post left innominate scores; Exact significance, p= 1.000. For the experimental group there was no statistically significant difference 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 the control group there is no statistically significant relationship between pre and post right innominate scores; Exact significance, p= 0.686. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre and post C3 scores; Exact significance, p= 0.400. For the experimental group there was a statistically significant difference 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the sphenobasilar symphysis, p= 1.000. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left occipito-mastoid suture p= 0.429. For the experimental group there was a statistically significant difference between pre- and post- scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right occipito-mastoid suture p= 0.771. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left petrous basilar suture p= 0.143. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right petrous basilar suture p= 0.486. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left C0-C1 p= 0.829. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right C0-C1 p= 0.486. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the C0-C1 on C2 p= 0.686. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the ethmoid p= 0.400. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left jugular foramen p= 0.429. For the experimental group there was a statistically significant difference between pre- and post-scores, p= 0.009.

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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right jugular foramen p= 1.000. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the foramen magnum p= 0.257. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left parietal bones p= 0.486. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right parietal bones p= 0.486. For the experimental group there was a statistically significant difference between pre- and post-scores, p= 0.048.

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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left temporal bone p= 0.143. For the experimental group there was no statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right temporal bone p= 0.486. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the frontal bone p= 0.657. For the experimental group there was a statistically significant difference between pre- and post-scores, p= 0.009.

Crosstab

Count

Treatment			FB R 0	FB R 1	FB R 2	FB R 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right frontal bone p= 1.000. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the occipital squama p= 0.571. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the greater wings of the sphenoid p= 0.657. For the experimental group there was a statistically significant difference between pre- and post- scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left zygoma p= 1.000. For the experimental group there was no statistically significant difference between pre- and post-scores, 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 the control group there was missing data so no statistically significant testing was possible for the right zygoma with respect to vitality. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left maxilla p= 1.000. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right maxilla p= 1.000. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left maxilla p= 1.000. For the experimental group there was no statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right mandible p= 1.000. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left palantine bone p= 0.771. For the experimental group there was no statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right palantine bone p= 0.771. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the vomer p= 0.686. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left lacrimal p= 0.543. For the experimental group there was no statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right lacrimal p= 1.000. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left nasion p= 1.000. For the experimental group there was no statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right nasion p= 0.571. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the sacrum p= 1.000. For the experimental group there was no statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the L5-S1 p= 0.657. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the left innominate p= 0.371. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for the right innominate p= 1.000. For the experimental group there was a statistically significant difference between pre- and post-scores, 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 the control group there is no statistically significant relationship between pre- and post- vitality scores for C3 p= 0.571. For the experimental group there was no statistically significant difference between pre- and post-scores, 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.

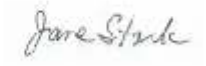
If you take this offer, and don't find the 16 subjects that you require, you just might find enough to give your study the power it needs so that it can be completed as a full study.
If it turns out that you fall short of subjects then you will be required to provide some compelling scientific reasons (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

cc. Brad McCutcheon
cc. Ryan Marciniak

APPENDIX T: PROPOSAL