Parkinson’s Disease

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Parkinson’s Disease: An Overview

Parkinson’s Disease (PD) is characterized as a chronic, progressive neurological disorder that affects muscle reflexes and the extrapyramidal nervous system (Durstine et al., 2009). Decreased amounts of dopamine, a neurotransmitter produced by the substantia nigra, in the basal ganglia, is attributed to the symptoms of the disorder. Gibb & Lees (1988) suggested loss of neurons in the substantia nigra and an approximated 80% depletion in striatal dopamine begins substantially prior to motor symptom development. Parkinsonism is the term used to describe the four cardinal symptoms of PD: tremor, rigidity, bradykinesia, and postural instability. Idiopathic PD is the most common form of parkinsonism. Lewy bodies are defined neuronal inclusions that are markers for neuronal degeneration and determines the percentage of risk in the development of Parkinson’s Disease. Thus, the population size exhibiting causes of Parkinson’s Disease is directly correlated with the size of Lewy bodies.

Demographics of Parkinson’s Disease

Of the three main neurogenerative diseases in the elderly, by 2040 Parkinson’s Disease is expected to surpass cancer as the second most common cause of death (Lang & Lozano, 1998). The size of PD population worldwide is significant, with more than 1 in 10 of persons over 80 years old affected. Hughes et al. (1992) estimated the mean range for Parkinson’s Disease is 31-85 years of age, although disease onset has a mean age of 64-65 years. The duration of Parkinson’s Disease is ranged from 2-35 years. Patients with degenerative diseases affecting the central nervous systems and the elderly have a higher percentage of Lewy bodies and are thus the populations most likely to have Parkinson’s Disease. According to Gibb & Lees (1988), the pervasiveness of Lewy bodies increased from 3-8% to 12-8% from the 6th to the 9th decade of life; this confirms both age and time are highly correlated with the progression of Parkinson’s Disease. Idiopathic PD is found more often in men and is less frequent in African blacks and Asians. In the United States, PD affects an estimated 1.5 million people (Durstine et al., 2009).

History of Parkinson’s Disease

Lang & Lozano (1998) document Parkinson's Disease symptoms were found in Ayurvedic texts in India from 4500 to 1000 B.C., termed 'kampavata' which was composed of tremor and akinesia. James Parkinson gave the first description of “paralysis agitans” characteristic of Parkinson’s Disease in 1817, in An Essay on the Shaking Palsy. From 1868-1881, Jean-Martin Charcot made distinctions in the disease conditions, including rigidity, weakness and bradykinesia and coined the term Parkinson’s Disease. Parkinson's Disease was discussed during the Industrial Revolution in America which links it to the causative role of exogenous toxins (Lang & Lozano, 1998).

PD Diagnosis & Clinical Features

Diagnosis of Parkinson’s Disease is through neuropathological examination, including brain imaging. Magnetic resonance imaging (MRI) will demonstrate a mix of low and high signal intensities with multi-system atrophy such as: striatonigral degeneration; olivopontocerebellar atrophy; midbrain atrophy; asymmetric cortical atrophy; striatal infarcts, subcortical and
periventricular white-matter variations. Tremor, rigidity and akinesia form the classic triad of the physical manifestations in Parkinson’s Disease. Resting tremor has a classic 4-to-6 Hz frequency (Lang & Lozano, 1998).

Stages of PD: The Hoehn & Yahr Scale

Durstine et al. (2009) outlines the stages of PD according to the severity of the cardinal symptoms. Early stages of PD are marked by minor tremor and infrequent stiffness. The moderate form of PD occurs when the patient feels limitation in movement and mild to intermediate intensity tremor. The advanced stage of PD occurs when the patient is significantly limited in movement despite medical treatment. The symptoms of the progression of PD are given a 0 to 5 relative level of disability measure in the Hoehn and Yahr scale. Symptoms on only one side of the body are characteristic of stage one. Stage two involves bilateral symptoms without impairment of balance. In stage three, the patient is physically independent despite balance impairments. In stage four, or severe disability, the patient is still capable of standing and walking unassisted. Stage five is evident when the patient is wheelchair bound or bedridden and needs constant assistance.

Classification of PD

Parkinson’s Diseases is classified through various categories (Durstine et al., 2009). The age classification differentiates patients into: juvenile PD (diagnosed under 40 years of age); PD between the ages of 40-70; and PD after age 70. Clinical symptom classification is: tremor predominant; akinetic-rigidity predominant; or postural instability-gait predominant. Patients are categorized into dementia absent or present mental-status. The course of PD is subdivided into: benign, progressive and malignant.

PD Motor Symptoms

The four cardinal symptoms of PD are attributed to weaker dopaminergic projections from the substantia nigra. Specifically, the projection to the internal pallidum has a weaker dopaminergic excitatory effect and the projection to the external pallidum has a weaker dopaminergic inhibitory effect. The brain’s cortex receives a lessened amount of excitatory input from the basal ganglia from these functional changes in the direct and indirect pathways (Latash, 2008). Tremor occurs at rest and sometimes during voluntary movement; it has a characteristic frequency of 5-6 Hz in the antoagonist muscles controlling a joint. Rigidity is often found in the neck, shoulders, trunk and extremities; it is defined as a constant resistance to joint movements. Bradykinesia is often evident in the slow movements of the fingers, hands, arms and/or legs. Postural instability is observed as kyphosis, adducted shoulders, flexed knees and elbows; it is associated with greater pre-programmed corrections in postural muscle activities (Latash, 2008). According to Durstine et al. (2009), gait in PD patients exhibits: festination (short, hasty and shuffling steps); retropulsion (walking backwards); akinesia (freezing or difficulty starting movement); and reduced arm swing. Akinesia occurs most often in doorways, narrow passages and turns. PD patients are at a very high risk for falls. Activities of daily living that are affected include: handwriting (micrographia); difficulty using utensils and swallowing food; and a
hindered ability to dress, bathe, and move around the house. Speech impediments and communication disorders are common due to hypomimia (loss of facial expression).

**PD & Voluntary Motion**

Bradykinesia is the primary movement pathology in persons with Parkinson’s Disease. A greater reaction time is evident especially when movements are more complex. An exaggerated movement time is correlated with acceleration and deceleration phases that have asymmetry. Problems with inter-joint coordination in multi-joint motions is common in PD. Movements have high jerk indexes that are associated with compensation in interaction torques. Variability, both temporal and spatial, to a target is greater in PD. In PD, this variability is linked to a compensatory mechanism in the brain and not a primary deficit. Targets are generally under-shot in PD patients; these hypometric motions are particularly evident during fast movement (Latash, 2008).

In PD, persons have difficulty creating one motor action. A motion is often segmented into discernable pieces, with prolonged intervals between movement sections. This is observed on EMG recordings that show cycles of agonist and antagonist bursts of activity. A typical EMG recording for isometric contraction shows a slow accumulation of muscle activity followed by unusual co-contraction of antagonist muscle groups. In PD, kinematic patterns are disrupted during voluntary movement. In general, PD patients have a non-precise number and frequency of motor units which undergo activation. This has been connected to a failure to energize the muscles even though the motor program is in-tact. The rate of force in muscle activity is altered in PD and it is challenging for the patient to control force levels. This change is attributed to the time patterning of muscle activity which is regulated by reflex feedback loops and the segmental spinal apparatus (Latash, 2008).

Posture is controlled via two corrective reactions in non-PD persons. In the first reaction, the central nervous system uses *anticipatory postural adjustments* (APAs) to control postural stability before a predictable perturbation. In the second reaction, the central nervous system pre-programs reactions; information is sent to the CNS from a peripheral stimulus when perturbations are unexpected. Pre-programmed reactions are faulty in PD and postural perturbations are not voluntarily controlled. A PD patient demonstrates greater feedback-triggered reactions to postural perturbation that are poorly controlled. In PD, a muscle that is stretched will have a long-latency muscle response and high amplitude; this is true for active and non-active muscles. APAs prior to voluntary motion have smaller amplitudes in PD patients due to the antagonist muscle’s anticipatory co-contraction. APAs are inappropriately generated in PD and can affect movement initiation and programming. Therefore, motor deficits in PD are associated with adaptive reactions (Latash, 2008).

Reasons for pre-programmed motor correction problems in PD are unknown although many possible mechanisms are speculated. Walking and sustaining a vertical posture requires the CNS to perform constant corrections. Memory stores these pre-programmed reactions therefore the ability to make corrections is not lost. The mechanism that triggers these reactions is impaired. To overcome the impairment, the CNS overcompensates by either: lowering the pre-programmed corrections’ triggering threshold or increasing the correction response. In the later, the result of
overcompensation is a pre-programmed reaction in the opposite direction. Rigidity is expected from overcompensation as the system’s stiffness is increased. Latency periods can double in the pre-programmed response since peripheral receptors take more time to react to perturbations caused by an earlier pre-programmed reaction. The oscillations of this period have the characteristic PD tremor frequency of 5-6 Hz (Latash, 2008).

Causes of Parkinson’s Disease: Hypotheses

In general, the cause of Parkinson's Disease is accepted as multi-factorial with genetic and non-genetic components (Lang & Lozano, 1998). The fields of science and medicine have not been successful in finding the exact causes of Parkinson’s Disease albeit likely generalized causes of Parkinson’s Disease are environmental and genetic.

Hughes et al. (1992) suggests causes for Parkinson’s Disease may relate to Lewy body distribution. This distribution may indicate a “field change” in various neuronal types; specifically, they call attention to the cells that synthesize tyrosine hydroxylase, found in the cerebral cortex, and their plausible roll in Lewy body genesis. Leroy et al. (1998) found irregular UCH-L1 protein aggregation, one of the most common proteins found in the brain, in Lewy bodies. The dominance of this protein was associated with reductions in the the proteolytic pathway. The UCH-L1 protein has a natural substrate that is still not discovered, more research is needed to understand the role it takes at different stages in the pathogenesis of Parkinson's disease.

The greatest risk factor for Parkinson’s Disease is age. The pathogenetic factors are not known for Lewy ody disease, although research demonstrates an increase in Lewy body disease with age. Despite the age-specificity of Lewy body disease, normal aging cannot be isolated as a cause, only a risk factor, in Parkinson’s Disease. Other risk factors associated with Parkinson's Disease include: herbicide and pesticide exposure; smoking; deficient intake of antioxidants; and low vitamin E intake. The largest risk factor, besides age, is a family history of Parkinson's Disease.

Lang & Lozano (1998) spotlight the importance of the preclinical period in Parkinson's Disease, termed the prodromal period. This time frame starts at the occurrence of the pathological changes in the substantia nigra with associated loss of striatal dopamine. Therefore, research is needed on the duration of the preclinical period and is implied to help with the understanding of the causal factors in Parkinson's Disease. Current research is controversial and the preclinical period can range from 5 years to several decades.

Environmental toxicity is correlated with the many possible causal factors in Parkinson's disease: mitochondrial dysfunction; high rates of oxidative and metabolic stress in proteins; activity of excitotoxins; inadequate neurotrophic support; and immune factors such as high levels of cytokines (Lang & Lozano 1998). Schapira et al. (1990) studied mitochondrial respiratory-chain enzyme proteins' structure and function in patients with Parkinson’s Disease to support the theory that environmental toxins may cause Parkinson's Disease. Their study suggests the activity of NADH-ubiquinone reductase (Complex I) and NADH cytochrome c reductase were abnormally low. Since this unknown biochemical defect of Complex I activity was also found in
animal models with parkinsonism, an environmental toxin was implicated as a causal factor in Parkinson's Disease. Parkinsonism in both man and primates is associated with the selective toxicity of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). MPTP's derivation (MPP) inhibits Complex I which leads to mitochondrial myopathies and produces nigrostriatal cell death. Schapira et al. (1990) concluded an unknown toxin could be incorporated into this process and prompt Complex I deficiency. This implies that Parkinson’s Disease is not specific to any one geographic location and may or may not be attributed to a widespread environmental factor. The hypothesis of an early life origin, antenatal period, as a possible cause of Parkinson’s Disease warrants more examination including researching endogenous neurotoxins and growth factors.

The supraspinal origin of Parkinson’s Disease leads to the hypothesis that motor disorders are caused by alterations in the descending motor commands. Latash (2008) gave support for this hypothesis through tendon jerk reflexes that were not changed in PD patients and Ia-muscle afferents with normal short latency actions. Changes in the segmental apparatus is found in PD patients in a deficit known as deficit in reciprocal inhibition. This deficit results in; a significant co-contraction of antagonist muscle groups during voluntary motions; tracking phases with heightened reflex activity causing the muscle to lengthen; and the Westphal phenomenon. In the Westphal phenomenon, there is a sudden reflex excitation of the muscle and shortening reaction, the opposite of the stretch reflex (Latash, 2008).

Lewy Body: An Overview

Lewy bodies are highly specific. Lewy bodies of the substantia nigra and locus coeruleus have customary structural features. The central body of the Lewy body is recognizable by a pale-staining halo and are eosinophilic at the core; such characteristics are from the high packing density and unambiguous filament degradation. Lewy bodies are round (5-25 pm diameter) and intracytoplasmic. The outside the body structure is composed of radially orientated, aggregated filament (7-15 nm in diameter). Other Lewy body compositions include: a dark core with or without dark laminae. The laminae are rendered by immunostaining and look like peripheral rings. Laminae are produced from the reaction between: polyclonal antibodies and neurofilament proteins; and/or monoclonal antibodies and neurofilament polypeptides.

Idiopathic Lewy Body Disease

Consistent evidence suggests Lewy bodies to be inclusive in Parkinson’s Disease, leading to the term ‘idiopathic Lewy body Disease’ as a synonym for Parkinson’s Disease. The loss of neurons in the brain alters the structure of its neurofilaments, yet many of the surviving neurons have Lewy bodies (Gibb & Lees, 1988). The specific arrangement of the loss of cells acts as a diagnostic marker in the pathology of Parkinson’s Disease. Gibbs & Lees (1988) found Parkinson’s Disease can be excluded when a specific section of the substantia nigra is lacking the presence of Lewy bodies (one of the two bilateral 7pm areas). Parkinson’s Disease is associated with cell loss in extranigral sites which is common in both dementia and autonomic failure. These disease states are comorbidities with Parkinson’s Disease. When Lewy bodies are manifested in persons without Parkinson’s Disease, this is called ‘incidental Lewy body Disease, and is a pre-symptomatic form of Parkinson’s Disease.
Location of Lewy Bodies

Parkinson’s disease can be diagnosed with the presence of Lewy bodies. Within the broader definition of Parkinson’s Disease, Lewy bodies are generalized to be evident in the substantia nigra. Other common locations for Lewy bodies in other brain regions include: the locus coeruleus; dorsal vagal nucleus; nucleus basalis of Meynert; and hypothalamus. The autonomic nervous system in Parkinson’s patients is considerably affected by Lewy bodies in the following areas: the Edinger-Westphal nucleus; the salivatory nuclei; the dorsal vagal nucleus; the intermediolateral nucleus; the sympathetic ganglia; and the parasympathetic ganglia. Lewy bodies are less often found in the cerebral cortex, thalamus and autonomic ganglia. The cells with the greatest distribution of Lewy bodies are mono-aminergic and cholinergic neurons that are medium to large in scale (Gibb & Lees, 1988).

Literature Review: Lewy Bodies

In the studies of over 140 brains by Gibb & Lees (1988), Lewy body predominance in persons in their fourth decade of life is less than 07%. This finding gives merit to the hypothesis that Parkinson’s Disease is a progressive pathology linked to idiopathic Lewy body disease. Case studies suggest the fundamental pathogenetic mechanism in Parkinson’s disease is neuron destruction which happens at a consistent and moderate rate. The pervasiveness of Lewy body disease in persons with Parkinson’s Disease over 85 years old increased to 19%. The onset of Parkinson’s Disease symptoms is related to an 80% loss of striatal dopamine. With this data, Gibb & Lees (1988) point to the ages of 25-35 years as when histological changes start to occur, with the average onset of Parkinson’s Disease symptoms occurring at age 60. Therefore, histological changes could start before or after this benchmark, depending on the case-specific age on symptom onset. Hughes et al. (1992) screened 100 patients with idiopathic Parkinson’s disease. This study utilized resources from the Parkinson’s Disease Society Brain Bank (PDSBB) in London which has donor tissue, received and examined annually, from Parkinson’s patients. Within 5 minutes neuroscientists found 76 had nigral Lewy bodies in the cerebral cortex, mostly in the frontal anterior cingulate gyrus and some in the parahippocampal gyrus. Loss of neurons was also evident in the substantia nigra with Lewy bodies in the locus coeruleus and dorsal vagal nucleus. Off the 100 patients, loss in the substantia nigra due to Lewy bodies was calculated as mild in 1%, mild-moderate in 30%, moderate-severe in 39%, and severe in 20% of the cases.

Pathological Findings

Lang & Loazano (1998) specify the neurological degeneration which occurs with Parkinson’s Disease. Brain regions affected include: the pars compacta of the substantia nigra (the neuromelanin-laden dopaminergic neurons); aminergic brain-stem nuclei (catecholaminergic and serotoninergic); the cholinergic nucleus basalis of Meynert; hypothalamic neurons; the cingulate gyrus and entorhinal cortex; the olfactory bulb; and sympathetic ganglia. Their study found some dopaminergic projection areas to be more susceptible than others. Parkinson’s Disease has the characteristic of greater neuronal cell loss, 60-70% at symptom onset, in the ventrolateral tier of the pars compacta. This is followed by neuron degradation in the medial ventral tier and the dorsal tier, respectively. Loss of striatal dopamine the resultant feature and is likely attributable
to the development of akinesia and rigidity. The proposed mechanism deals with dopamine transporter and the degree of expression of messenger RNA. The neurochemical pathway linked to Parkinson’s Disease is associated with ubiquitin-positive processes (Lewy neurites) and their degenerating qualities on cells (Lang & Lozano, 1998).

**Treatment**

According to Lang & Lozano (1998), treatment for Parkinson's Disease is three-fold: preventative, symptomatic, and regenerative treatments. Preventative treatment involves drugs that protect neurons while influencing oxidative phosphorylation. These drugs aim to stop: free radical damage; disproportionate iron deposition; calcium homeostasis imbalances; cytokines; excitotoxicity; nitric oxide; and apoptosis. Treatment of Parkinson’s Disease requires pharmacological intervention. Medications change the neurochemical imbalances in the brain and aim to lower epinephrine and norepinephrine levels while increasing levels of acetylcholine. Dopamine production is modified through: dopaminergics (levadopa, levadopa/carbidopa, amantadine, pergolide, bromocriptine, pramipexole, ropinirole); anticholinergics (benztropine, trihexyphenidyl); and monoamine oxidase type B inhibitors (MAO-B such as deprenyl and selegiline). Early symptomatic therapy is sometimes linked with Levodopa but has been proven to cause numerous harmful side-effects including: motor fluctuations such as loss of mobility and dexterity due to the dependency and loss of response to the drug; dyskinesias; and a possible neurotrophic effect. This dopamine metabolic-precursor passes through the blood-brain barrier and augments the quantity of dopamine available to the basal ganglia. Levadopa is also metabolized in peripheral muscles although research as to its effects is sparse. These medications have been documented to produce the following responses: bradycardia and tachycardia (levadopa/carbidopa); dyskinesia (levadopa/carbidopa, pergolide, pramipexole, ropinirole, selegiline); and mood elevation (selegiline). Others side effects include: insomnia, hallucination, confusion, and mental activity swings. Drug use for greater than 5 years results in a reduced response to its effectiveness. Later stages of Parkinson's Disease manifest as dementia, one of the most challenging handicaps to therapeutically manage. This field requires additional research. Restorative therapy may or may not require surgery. Neurosurgical procedures attempt to alter activities in the following parts of the brain: motor thalamus, the internal segment of the globus pallidus, or the subthalamic nucleus. The thalamus is specifically targeted for tremor symptoms. Lang & Lozano (1998) documented the potential for deep brain stimulation, either disruptive or inhibitive in function, via implanted electrodes as a treatment strategy. Gene therapy combined with surgical interventions is utilized to: increase the expression of enzymes specifically involved in the biosynthesis of dopamine; prevent apoptosis; enhance mitochondrial function; scavenge free radicals; and clean-up toxic metabolites. More clinical studies are warranted to effectively treat and halt the progressive course of Parkinson's Disease.

**Exercise As Medicine for PD**

Outcome to exercise training is variable and individualized. Exercise has assisted in the halt or reverse of PD symptoms and increased the quality of life in PD patients (Durstine et al., 2009). Improvements from exercise training include increases in: motor performance; trunk rotation; hand-eye coordination; stability and balance (especially during walking); non-motor symptoms; and muscle strength and volume. The exercise therapist must consider the following responses to
exercise in the PD patient due autonomic nervous system dysfunctions: thermal regulation; heart rate and blood pressure changes; and oxygen consumption alterations. According to Durstine et al. (2009), a comprehensive exercise program has aerobic, endurance, resistance, flexibility and functional components. The exercise program’s aerobic training component: seeks to maintain or increase aerobic capacity; is practiced three days a week at 60 minutes per session, with a peak heart rate of 60-80%; and can use leg and arm ergometry or rowing machines. The endurance component of the exercise plan includes supervised, short walking sessions that are 30 minutes in length with an optimal frequency of five sessions per day. The strength aspect of the exercise training can use weight machines or therabands to build muscle tone in the arms, shoulders, legs and hips; one set of 8-12 repetitions is advised three times a week. The flexibility component involves stretching through modes such as yoga, tai-chi and Pilates to increase ROM and is practiced three days a week. The functional aspect of the exercise program includes walking and posture exercises. An example of a functional exercise involves placing a scotch tape on the exercise floor at stride-length intervals. The patient walks with an even pace on the tape and the tape is moved periodically to train new walking patterns.

Physical Therapy Treatment

The main goals in physical therapy treatment for PD include: maintaining functional capacity at a high level such as ADLs via functional re-training; enhancing coordinated movement such as movement initiation, trunk mobilization, and general ROM; decreasing tremor, rigidity, muscle atrophy and postural changes; and preventing secondary complications from the disease or its medication. The physical therapist’s overarching theme is to integrate the bodily systems. Since the 1950s, The Guide to Physical Therapist Practice (Cedarbaum et al., 1992) indicated PD patients need targeted interventions to help cope with decrements in functional tasks. Early physical therapy treatment aims to keep the patient’s body conditioned; whereas later stages of PD warrant preventing contractures, immobilization, pain and drug-dependency.

Deficits which need to be accounted for are not limited to: balance; endurance; motor and sensory function. Mitchell (2003) states rehabilitation techniques may be useful to the PD population. Visual and auditory cueing, chaining (de-constructing a complex motion into several simple movements), proprioceptive feedback and biofeedback have been documented to assist in gait improvements. Coordination exercises are implemented to assist with activities of daily living can where axial structures should be emphasized to increase ROM and improve functional reach (Schenkman et al., 1998). Intervention for strength building is essential since PD patients are less capable of isometric and isoinertial force production (i.e. when the patient moves against a pre-determined resistance throughout a ROM).

Osteopathic manipulation was investigated to examine its effects on rigidity, flexibility and muscle length. Improved gait stride length and cadence were reported in the treatment group versus control groups (Wells et al. 1999).

Intervention strategies that were therapy-based were reviewed to study SES, summary effect sizes (De Goede et al., 2001). The Q-statistic for each set of SES were examined. In a fixed model, the meta-analysis gave the following results: homogeneous SES for ADLs with 0.40 confidence interval of 0.17-0.64; homogeneous SES for stride length with 0.46 confidence
interval of 0.12-0.82; and heterogeneous SES for walking speed using a random effects model with 0.49 confidence interval of 0.21-0.77.

Acknowledgements

This research project was chosen so that I may better understand Parkinson’s Disease. Throughout this semester I have volunteered my time at Piedmont Yoga (3966 Piedmont Ave, Oakland, CA., 94611) volunteering with PD Active. PD Active began in 2007, sponsored by the Mark Morris Dance Group and obtained 501(c)3 federally exempt non-profit status in 2009. The class is funded by a grant from the Yoga Dana Foundation. PD Active stands for: People impacted by Parkinson’s disease (PD); Discovering that we can improve our own quality of life; Advocating on our own behalf; Connecting and building PD communities; Transforming existing programs & services to better serve our needs; Increasing awareness about PD; Voicing our opinions with dignity and tenacity; Energizing each other to stay involved and ACTIVE!

Dr. Barry Gustin introduced me to David Leventhal, the Program Manager, Dance for PD & Mark Morris Dance Group (david@mmdg.org & www.danceforpd.org). I met with the director of PD Active's Oakland/Berkeley chapter, Herb Heinz, who introduced me to Vickie Russell Bell. I assisted Vickie as she taught yoga to individuals with Parkinson's Disease. The class meets every Thursday from 1:30-2:45 pm although I would arrive early and stay after-class to set-up and put-away all the yoga props. Other assistants also helped with this and other tasks.

I observed as Vickie taught the students how to stretch and strengthen their bodies. Every class centered around a different theme including: stretching target areas of the body (chest, shoulders, upper back); breathing and moving in synchrony (arm and pelvis motions); balance exercises (and falling practices); and relaxation techniques. Props were often utilized to explore a pose, as several students had flexibility and rigidity limitations. Vickie based each lesson on what the group needed; some days required a more active practice and others, more restorative. I saw the community that was built around this class and how much fun the students have with the yoga practice, Vickie and each other. As a certified yoga teacher of 10 years, I felt confident in this setting and was able to explore 'adjusting' students in poses. I learned how to access when to actively assist a student in, into or out-of a yoga pose versus when to let them perform these transitions independently. I enjoyed observing how each individual adapted the yoga practice to fit their particular needs and the subsequent variations that occurred.

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References


