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THINK BIG TO BEAT PARKINSON’S DISEASE – A PRESENTATION FOR PHYSIOTHERAPY STUDENTS

Degree Programme in Physiotherapy
2017
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Degree Programme in Physiotherapy
November 2017
Number of pages: 28

Keywords: Parkinson’s disease, amplitude training, presentation

Parkinson’s disease is the second most common neurodegenerative disorder that causes slowness of movement (bradykinesia), tremors and rigidity. Bradykinesia can present itself in multiple ways from reduced facial expression to disturbances in the normal gait cycle.

Recent advances in neuroscience have shown that the brain is able to restructure itself in response to physical activity. This concept has driven the development of a new treatment protocol, LSVT BIG (Lee Silverman Voice Therapy BIG) that aims to tackle the cardinal symptom of Parkinson’s disease: bradykinesia.

The protocol is very focused on one key area of the disease. This focus is distilled into one verbal cue, “BIG” that is applicable to all activities of daily living. Having a specific aim and a target simplifies the process of physical therapy so that more time is spent on activities and relearning the proper use of one’s body and less time is spent cluttering the mind with words.

The objective of this thesis was to design a presentation for physiotherapy students that introduces the basic concepts and principles and the rationale behind the LSVT BIG protocol. The presentation also showed what a physiotherapy student could learn from the LSVT BIG protocol regardless of their chosen field. The presentation was considered a success according to the feedback from the audience.
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INTRODUCTION

Parkinson’s disease is a condition where the dopamine producing neural pathways in the brain die resulting in tremors, rigidity, bradykinesia and hypokinesia. A shuffling gait or freeze attacks are also common characteristics of Parkinson’s disease. Neuropsychiatric symptoms may also be present as the disease progresses. Patients diagnosed with Parkinson’s disease are at a higher risk of falls and their level of activity and participation may suffer in the later stages.

The treatment protocol of Parkinson’s disease always includes medical intervention, mainly Levodopa treatment. Physiotherapy is largely aimed at improving physical capacity, mobility, balance, manual activities and transfers. Bradykinesia, meaning slow movement, is rarely the focus of treatment.

Recent advances in the field of neuroscience has given rise to the concept of neuroplasticity. It is known that exercise can affect brain function in a positive way. This concept serves as a cornerstone of the LSVT BIG protocol. The acronym “LSVT” comes from the words “Lee Silverman Voice Therapy”.

The LSVT BIG protocol is a high intensity intervention aimed specifically at treating Parkinson’s disease, focusing on hypo- and bradykinesia. It is derived from the LSVT LOUD method used worldwide by speech therapists. The BIG protocol aims at using high intensity exercise daily to able the clients to modify their brain resulting in a well ingrained internal cue that benefits all activities of daily living. The results of this treatment are shown to last over three months post intervention.

The author of this thesis aims to disseminate this information in Finland in order to raise awareness of a new tool that can be used in treating people with Parkinson’s disease.
2 PARKINSON’S DISEASE

2.1 Definition

Parkinson’s disease is a progressive neurodegenerative disease that is characterized by slowness of movement (bradykinesia), abnormal muscle rigidity and resting tremors. The motor symptoms of Parkinson’s disease can be explained by the loss of dopamine producing cells in the midbrain. The disease also causes changes in autonomic nervous system function and non-motor symptoms such as dysfunctions in communication, mood and cognition. (Stokes & Stack, 2012, 117.) Neurons are also affected in the peripheral nervous system, other neurotransmitter systems, the brainstem, the autonomic nervous system and the cortical and subcortical structures which causes the non-motor symptoms (Käypä hoito 2017).

Primary Parkinson’s disease is idiopathic, meaning its causes are unknown. It is most likely caused by multiple different factors such as aging, environmental toxins and genes that cause neurophysiological changes in cells that produce dopamine, causing them to die. (Stokes & Stack, 2012, 118.) An imbalance in the secretion of dopamine and acetylcholine is also hypothesized to cause most of the symptoms (Tortora & Derrickson 2011, 630). Twelve genes have been identified and linked to Parkinson’s disease, with an earlier onset indicating the likelihood of a familial cause (Blochberger & Jones 2011, 361). However, a family history of Parkinson’s disease is present in only 5%-20% of the cases and it is rarely hereditary (Tortora & Derrickson 2011, 630; Atula 2016). Parkinson’s disease seems to be one of the very few conditions from which smoking seems to protect, although no means of actually preventing the disease have been identified (Atula 2016).

Approximately 5.2 million people worldwide suffer from Parkinson’s disease. It is more prevalent in North America and Europe than in Africa, most likely explained by the difference in the average life expectancy. (Blochberger & Jones 2011, 361.) Parkinson’s disease is one of the most prevalent degenerative neurological conditions, right after Alzheimer’s disease (Yang, Tang & Guo 2016). As of 2014, approximately 14,000 people in Finland suffer from Parkinson’s disease. Of the whole population, 1-
2 out of a thousand have a diagnosis of Parkinson’s disease increasing up to 2 in a hundred among people aged 70 years or older. The disease is most prevalent among people 50 to 80 years of age, with men having a higher risk of getting a diagnosis. (Website of the Finnish Parkinson’s Association 2017).

Initial symptoms of Parkinson’s disease are often unspecific such as fatigue, pain, weakened voice and micrographia, small handwriting. Symptoms are usually unilateral at first and become bilateral as the disease progresses. Primary findings like rigidity, hypokinesia and postural instability present around the moment of diagnosis. (Soinila, Kaste & Somer 2012, 218-219.) Parkinson’s disease also negatively affects the subject’s ability to swallow and control their balance, gait and limb function (Fox, Ebersbach, Ramig & Sapis 2012). Most common cognitive impairment is bradyphrenia, slowness of speech and difficulties initiating speech, and the most common neuropsychiatric symptom is depression, occurring in approximately half of the cases (Soinila, Kaste & Somer 2012, 219). A correlation has been found between the cognitive and motor impairments in Parkinson’s disease that may lead to earlier identification of rehabilitative strategies, though further research is needed (Varalta et Al 2015). Both motor and non-motor symptoms have been shown to reduce the quality of life of people with Parkinson’s disease (Santos-García & de la Fuente-Fernández 2013; Chapuis et Al 2004).

2.2 Motor Symptoms

Bradykinesia, akinesia and hypokinesia are the most common symptoms of Parkinson’s disease as they manifest themselves in virtually every patient and at the early stages of the disease. These terms are used interchangeably, but generally refer to slower, smaller movements and reduced spontaneous movements such as blinking and smiling. This reduction in movement speed is caused by abnormal activity in many parallel circuitries in the basal ganglia. (Mazzoni, Shabbott & Cortés 2012). The slowness and smallness of movement can appear in a multitude of different ways. Stride length reduces, getting up from a chair becomes more difficult and the reduced or lack of facial expression entirely can lead to difficulties in social interaction, for example. (Website of Parkinsoninfo.fi 2017).
Tremor is low in frequency and is present mostly in the upper limb at rest and is diminished during activity. The so called pill-rolling tremor is a typical form of tremor in the fingers. (Atula 2016). Tremor is the first symptom for every three out of four patients. It is one of the most common and easy to identify symptoms of Parkinson’s disease. Anxiety, stress and lack of sleep increase the tremor. However, a portion of the affected populace never experience tremors, or it can be very mild and infrequent. The tremor can also appear in the jaw and tongue which also affects speech. (Website of Parkinsoninfo.fi 2017).

Rigidity can present itself as slowness in movements and it is also apparent during assisted movements in which the subject resists passive mobilization of the joint (Atula 2016). Rigidity can be tested by passively moving one limb and combining it with a voluntary movement on the contralateral side. The test may even show signs of latent rigidity not apparent to the patient, who often do not complain about rigidity, but the ensuing functional limitations such as difficulties getting out of bed. It is suggested that rigidity is a result of a widespread pathology and is also correlated with a reduced overshoot in tracking tasks. (Baradaran et Al 2013). During active movement, in addition to the agonistic muscle the antagonistic muscles contract as well, resisting the wanted movement. Rigidity is often associated with muscle pain. (Website of Parkinsoninfo.fi 2017).

Postural instability is present in the later stages of the disease and is mainly caused by the degeneration of the systems that control balance and positioning (Atula 2016). Roughly half of people with Parkinson’s disease fall more than once a year (Käypähoito 2017). People with Parkinson’s disease have a tendency to sway backwards, which is called retropulsion. This may provide difficulties in turning, standing up from a chair or just remaining upright. Postural instability is often tested with the pull test, where the subject is tugged backwards with moderate force and then their recovery is observed. (Website of Parkinson’s Disease Foundation 2017). The subject’s posture may also become deformed, especially in the neck and trunk, where both become increasingly flexed. The knees and elbows may also become flexed. Postural control difficulties during movement may be present in the early stages of the disease and later, the balance reactions themselves may become impaired. (Keus et al. 2014).
Freezing is one of the secondary motor symptoms of Parkinson’s disease that may contribute to the heightened risk of falling. It is not explained by bradykinesia or rigidity. The subject may have trouble stepping forward in numerous situations, such as starting to walk, crossing a threshold or turning. The freezing is temporary and can be helped with verbal cues or other strategies. (Website of Parkinson’s Disease Foundation 2017).

2.3 Non-motor Symptoms

Parkinson’s disease is not only a motor disorder as it affects a wide variety of other systems such as the autonomic nervous system, cognition and sleep (Yang, Tang & Guo 2016). It was considered a pure motor disorder but is now considered a neurodegenerative multisystem disorder (Magrinelli et Al, 2016). These symptoms can often be unnoticed even though they pose a considerable threat to the subject’s quality of life and may actually appear more than a decade before the cardinal motor signs (Keus et al. 2014). As the disease progresses, dysfunctions of the autonomic nervous system can become apparent. These symptoms include sudden shifts in blood pressure, constipation or incontinence, excessive sweating or lack thereof, olfactory dysfunction or impotence. Sleep disturbances, drowsiness and severe exhaustion can also be present. Cognitive impairments, especially memory problems, slowness of thinking or depression can manifest. It is seldom that one person presents with all of these symptoms. (Atula 2016.) People with Parkinson’s disease may be embarrassed to discuss these symptoms with professionals or they may be unaware that these symptoms are related to the disease (Keus et al. 2014).

Executive dysfunction impairments result in the inability of the subject to function in an efficient manner in everyday life. This ailment is characterized by deficits in attention control, changing the target of attention, planning towards a goal, concentration, dual tasking, social interaction and resolving conflicts and making a decision. Executive functions are the core of normal, goal-oriented human behavior and encompasses all cognitive functions related to information processing including memorizing, planning, organizing and manipulating abstractions of time and space and inhibiting improper responses to external stimulus. These ailments may affect the subject’s ability
to follow a rehabilitation program or the ability to take medication as prescribed. (Keus et al. 2014).

2.4 Diagnosis

Diagnosis of Parkinson’s disease is based on a clinical neurological examination where at least two cardinal symptoms must be identified and abnormal findings are excluded. The United Kingdom Parkinson’s Disease Society’s Brain Bank criteria have resulted in an accuracy of over 90% in a specialized facility. Symmetrical motor symptoms, lack of tremor and early autonomic symptoms and falling could refer to some other type of parkinsonism. (Käypä Hoito 2017). Zero response to Levodopa treatment is also a sign of atypical parkinsonism and not Parkinson’s disease itself (Keus et al. 2014). Brain imaging technology cannot be used to diagnose Parkinson’s disease, but can help exclude other diagnoses. A good response to a month-long trial of dopaminergic medicine supports diagnosis. Differential diagnosis is focused on excluding parkinsonism caused by psychosis medication, vascular parkinsonism, multiple system atrophy, progressive supranuclear palsy, corticobasal syndrome and Lewy body disease. (Käypä Hoito 2017).

The probability of a misdiagnosis is 10-29% and it is especially inaccurate in the very early stages of the disease. Typical misdiagnosis in the early stages is due to essential tremor. (Käypä Hoito 2017.) People with a probable Parkinson’s disease are referred to a specialized neurologist for a better diagnostic accuracy (Keus et al. 2014).

2.5 Treatment

Medication is started as soon as the subject feels the symptoms affecting their quality of life. MAO-B inhibitors or dopamine agonists are usually the first choice of medication. Levodopa is considered the most effective medication for treating Parkinson’s disease. The aim is to find the minimal dose possible as Levodopa does not come without side effects. (Käypä Hoito 2017). Parkinson’s disease often requires multiple doses of different drugs that aim to manipulate the neurotransmitter imbalances in the basal ganglia, thus the adherence to medication is often quite low (Keus et al. 2014).
Dopamine agonists are weaker in alleviating the motor symptoms of Parkinson’s disease and will eventually require the addition of Levodopa. There is no evidence that dopamine agonists would be better in their treatment effect in the early stages of the disease than Levodopa and after two years, 40% of people with dopamine agonist as the first line of treatment required the addition of another type of medication, either MAO-B inhibitor or Levodopa. The aim is to increase the dose gradually to receive the most effective clinical impact, but if side effects do not enable this, another dopamine agonist should be used. The most common side effects of monotherapy is drowsiness, nausea, impulsive-compulsive behavior, orthostatic hypotension, dizziness, lower extremity swelling, sleep disturbances and constipation. (Käypä Hoito 2017).

MAO-B inhibitors selegiline and rasagilin are a good choice for subjects in the early stages of Parkinson’s disease, but their treatment effect is relatively weak and limited in duration. People do however tolerate these inhibitors well. If combined with selegiline, the Levodopa dose can be kept low. It is also associated with less shifts in function than Levodopa, but it is equal with dopamine agonists. Selegilin treatment results in less side effects than dopamine agonists and the treatment is seldom terminated because of that. Rasagilin is not yet shown to have an effect on the motor complications caused by Levodopa or the initiation of Levodopa treatment. (Käypä Hoito 2017.)

Levodopa is the most effective drug for Parkinson’s disease when compared to any other medication and over 40 years of clinical results have proven its effectiveness. It can cause nausea, loss of appetite and dizziness in the early stages of treatment, and increased shifts in functionality and psychological symptoms in long term treatment. Levodopa results in quite few side effects in addition to shifts in functionality and dyskinesias. In order to counter the effects the dosage is kept to the smallest possible one. If a subject presents with a history of psychosis, Levodopa is the safest form of medication. The modification of Levodopa treatment aims at reducing the daily “off” stages and their duration and also to minimize the eventual effect of dyskinesia, unwanted chorea. This is done by splitting the medication into multiple smaller doses throughout the day or with the addition of a dopamine agonist for example. (Käypä Hoito 2017.)
Deep brain stimulation is a form of neurosurgery that has almost completely replaced any other type of surgical treatment of Parkinson’s disease. Deep brain stimulation is very effective in reducing shifts in motor function and dyskinesia, and improves the subject’s quality of life. The stimulation of the substantia nigra and the globus pallidus interna effects last for at least five years. It benefits most those subjects with whom even the optimal medication suffer from shifts in function, dyskinesias such as tremor or have a short duration of response to Levodopa. In general the subjects have had Parkinson’s disease for ten years. Substantia nigra and the globus pallidus internus are the most common targets of deep brain stimulation. (Käypä Hoito 2017.)

The requirements are that the subject has a positive response to Levodopa. They must also have realistic expectations of the surgery as it does not cure the disease. They should be under 70 years of age, but it is not absolute. They must be able to cooperate during the surgery and also to learn to use the remote that comes with the device. No co-morbidities that would increase the level of risk for the actual operation and that they have had the disease for at least five years. (Käypä Hoito 2017.)

Deep brain stimulation may have a negative effect on the fluency of speech, speed of processing information and working memory. Given the electrodes do not run through the nucleus caudatus, the cognitive and psychiatric effects are not exacerbated. There’s no remarkable difference in cognitive changes post-surgery between the globus pallidus internus and substantia nigra stimulation. Usual complications include infections, stroke, breaking of the device’s cords, dislocation of either the generator, electrodes or wires and erosion of the skin at the site of the generator. Dysarthria and falls are also a complication. Paresthesia or diplopia may occur but they usually fade after the programming is finished. (Käypä Hoito 2017.)

2.6 Rehabilitation

There are five core areas that are to be assessed in a physiotherapy program for people with Parkinson’s disease according to the European Physiotherapy Guideline for Parkinson’s disease. These core areas are physical capacity, transfers, manual activities, balance and gait. (Keus et al. 2014).
Physical capacity consists of both the muscular and respiratory systems and physiotherapy aims to affect these. Sufficient muscle strength, endurance, coordination and mobility are essential in daily life. People with Parkinson’s disease also tend to be more inactive than the general population. Improvements in physical capacity is suggested to reduce the chance of early mortality and provide a plethora of other health benefits. (Keus et al. 2014).

Transfers are relatively complex chains of motor functions and may become increasingly difficult as the disease progresses, especially getting in and out of bed and rising from a chair. As fluency of movement combinations, accuracy and coordination becomes increasingly deficient, the ability to perform manual activities is also reduced. Tremor and regulation of muscle force may also affect the ability to do manual activities (Keus et al. 2014).

Falls are quite common in Parkinson’s disease and may present even in the early stages so the training of balance becomes a key factor. They can be caused by improper postural reflexes, proprioception or range of motion for example. As a consequence, people with Parkinson’s disease are at a higher risk of hip fractures and the rehabilitation takes longer and is less successful in general. Those who fall have a higher risk of falling again which may contribute to inactivity as the subject becomes increasingly afraid of moving. (Keus et al. 2014).

Gait disorders are considered either continuous or episodic. Continuous impairments include slower and smaller strides, absence of arm swing, a stooped posture and difficulties in turning. Reduced walking speed is correlated with limitations in activities of daily living and it is also a risk factor for mortality. Episodic impairments include festination of gait and freezing episodes. In festination, the feet are behind the center of their gravity and the subject takes multiple small steps, and it also refers to retropulsion. Freezing means the momentary inability to move the feet at all. (Keus et al. 2014).

The general guideline for conventional physiotherapy is to first educate the patient about their condition and also physical exercise in general and the benefits thereof.
Motivating people with Parkinson’s disease to maintain an active lifestyle is very important. Supervised exercise is considered more effective than unsupervised exercise. The physiotherapy program should encompass all the core areas with the focus on high amplitude movement aided by audible, visual and verbal cues. In the later stages of the program, dual tasking and complexity should be added. Other forms of exercise such as dance and Tai Chi can be effective in improving certain key areas. (Keus et al. 2014).

3 PATHOPHYSIOLOGY AND THE BASAL GANGLIA

The basal ganglia is a set of nuclei in the midbrain that initiate and stop movement and regulate unwanted actions and muscle tone (Tortora & Derrickson 2011, 550). They are also responsible for motor learning, behavior and emotions. They communicate with the cortex via two parallel pathways with opposite functions. The basal ganglia is associated with many movement disorders. (Lanciego, Luquin & Obeso 2012). The different circuitries between the basal ganglia nuclei have a wide range of purposes from selecting motor programs and shutting down unwanted ones and also help prepare for and execute movements. They are also used for other functions such as learning, planning and executing functions. (Magrinelli et Al 2016). The major components of the basal ganglia are depicted in Figure 1.

The basal ganglia can be divided into three different subsystems. The first one consists of the input nuclei that receive information from cortical, thalamic and nigral sources, for example. They are the caudate nucleus, putamen and accumbens nucleus. The second one consists of the output nuclei that project information to the thalamus. They are the globus pallidus internus and substantia nigra pars reticulata. The last system resides between the first two subsystems and it consists of the globus pallidus externus and substantia nigra pars compacta. The system forms a loop that receives information from the thalamus and the cortex into the striatum, then further into the basal ganglia and finally back to the origin, the thalamus and the cortex. (Lanciego, Luquin & Obeso 2012). The thalamus emits sensory and motor information to the cortex, which in turn initiates voluntary movements (Tortora & Derrickson 2011, 555).
In the midbrain lies a neuromelanin pigmented area, the substantia nigra. It is the source of dopamine in the midbrain. (Lanciego, Luquin & Obeso 2012). Reduced output of dopamine from the substantia nigra pars compacta to the striatum reduces the output level of the basal ganglia system and thus hinders the activity of the motor cortex (Mazzoni, Shabbott & Cortés 2012). The loss of these neurons in the substantia nigra leads to Parkinson’s disease and also bradykinesia, the central disorder. (Keus et al. 2014). Damage to the substantia nigra in animal models produced only bradykinesia, suggesting that other symptoms such as tremor and rigidity would require additional lesions. (Hallett & Khoshbin 1980). Because the projection of dopamine to the frontal lobes, striatum and limbic circuitries is reduced and also over time, non-dopamine related brain areas are damaged, the disease has a very complex way to manifest in a person (Keus et al. 2014).

4 BRADYKINESIA

4.1 Definition

Diagnosis of Parkinson’s disease requires the subject to present with bradykinesia (Website of the National Parkinson Foundation 2017). Bradykinesia is a motor symptom that describes the slowness of movement. It is sometimes used as a synonymous
term to describe akinesia, poverty of movement and hypokinesia, smallness of movement. (Berardelli, Rothwell, Thompson & Hallett 2001). It can cause difficulties with repetitive movements and everyday activities such as walking and buttoning a shirt and also decrease facial expression and the volume of speech (Website of the Parkinson’s Disease Foundation 2017). Daily activities take longer for people with Parkinson’s disease and in the later stages become more and more difficult. (Tortora & Derrickson 2011, 630). People with Parkinson’s disease often undershoot the initial movement and follow it with additional shorter movements to reach their target (Mazzoni, Shabbott & Cortés 2012). The number of these additional movement trials correlates with increased movement distance (Mazzoni, Hristova & Krakauer 2007). People with Parkinson’s disease also require additional firing of normal neuronal bursts to complete larger ballistic movements, for example elbow flexion in different ranges (Hallett & Khoshbin 1980).

Even though other symptoms of Parkinson’s disease, such as rigidity may contribute to bradykinesia, the main deficit is insufficient utilization of muscle force at the start of a given movement (Berardelli, Rothwell, Thompson & Hallett 2001). The lack of evidence of co-occurrence suggests that for example, rigidity and bradykinesia are manifestations of two different deficits (Mazzoni, Shabbott & Cortés 2012; Brumlik & Boshes 1966). Motor motivation can also contribute as people with Parkinson’s disease subconsciously opt for lower than normal speed of movement despite being able to reach the same accuracy and speed as healthy subjects (Mazzoni, Hristova & Krakauer 2007). Due to the way the basal ganglia are organized, it is possible that only one area of the subject body is affected, until all of the basal ganglia is affected (Hallett & Khoshbin 1980).

4.2 Exercise and bradykinesia

Progressive strength training has been shown to positively affect bradykinesia, improve upper limb movement speed and restore parts of the normal neuronal firing in people with Parkinson’s disease (David et Al 2016). Low load, high velocity training was also shown to improve levels of bradykinesia significantly in people with Parkin-
son’s disease during a three-month power training program (Ni, Signorile, Balachandran & Potiaumpai 2016). When lifting relatively high loads, people with Parkinson’s disease did not exhibit bradykinesia, compared to light and medium loads, which calls for more research on this topic (Allen, Canning, Sherrington & Fung 2009). High intensity, eccentric strength training also improved not only bradykinesia, but muscle force and quality of life in people with Parkinson’s disease during a 12-week study (Dibble, Hale, Marcus, Gerber & LaStayo 2009).

5 LSVT BIG

5.1 Definition

LSVT BIG is an amplitude based treatment protocol that is used to target the motor symptom bradykinesia in people with Parkinson’s disease. It is founded on the LSVT LOUD protocol, an amplitude based program for the speech motor system. The protocol is based on intense daily exercise for at least a month with the goal of making the movements of the patients bigger and calibrate them so that they learn a simple strategy for movement applicable to a multitude of different situations in their daily lives. With calibration, the physiotherapist also aims to re-educate the sensorimotor system of their patient. (Website of LSVT Global 2017.)

5.2 Principles of LSVT BIG

The principles of the LSVT BIG protocol are aligned with some of the concepts that drive activity dependent neuroplasticity, structural changes in the brain due to physical movement. These concepts include specificity, training focus on a single target that is increased movement amplitude, intensity, which means using an increased dosage of treatment, repetition, using an increased number of repetitions both in therapy and at home and salience, referring to using carryover and hierarchy tasks that are meaningful for each individual client (Fox, Ebersbach, Ramig & Sapir 2012).
5.2.1 Mode of treatment

The LSVT BIG treatment is a standardized exercise program. The dosage is four consecutive training sessions every week for four consecutive weeks with a total of sixteen sessions in a month. Repetitions for the daily exercises vary from eight to sixteen depending on the task. The level of effort must be anywhere from eight to nine out of ten, with ten being the absolute maximum intensity. (Website of LSVT Global 2017). It is hypothesized that a focus on a single verbal cue and motor goal, the cognitive load is reduced and focusing attention on movement and movement perception would become easier. (Fox, Ebersbach, Ramig & Sapir 2012).

5.2.2 Target of treatment

The goal of the LSVT BIG treatment is increased movement amplitude across the limb motor system to produce normal movement, including gait. The premise is that training amplitude as a main target would affect the mechanism that causes bradykinesia, insufficient muscle force when initiating movement (Berardelli, Rothwell, Thompson & Hallett 2001; Fox, Ebersbach, Ramig & Sapir 2012). Even though the goal is to increase the amplitude of movement, it is by the help of the therapist that the end result is not over-exaggerated movement, but movement within the normal range (Fox, Ebersbach, Ramig & Sapir 2012).

5.2.3 Calibration

People with Parkinson’s disease manifest problems in sensorimotor perception and may have issues recognizing their reduced movement amplitude and speed. In LSVT BIG, the goal is to recalibrate the client in a manner that the movements they may perceive as too big are in fact within the normal range of movement. This is done by constant feedback on the client’s movement and asking them how they feel about a given movement. The use of a video camera is also advised to record the client walking for example. The goal of the protocol is to have the client recognize the required level of movement amplitude to produce movements within the normal range and then have
them apply that amplitude habitually in every single activity of daily living. (Fox, Ebersbach, Ramig & Sapir 2012; Website of LSVT Global 2017).

5.3 The protocol

First half of the session consists of daily exercises that are high effort, intense movements that are repeated every day. As the treatment progresses, these exercises may be modified to challenge the subject more. Most of the early sessions will be focused on the daily tasks. However, as treatment progresses, less time is spent on the daily tasks and the hierarchy tasks will take more of a priority. (Website of LSVT Global 2017.)

The first tasks are maximum sustained movements that are done seated. Focus is on making a big effort movement that ends in a stretched position that is sustained for as long as possible. The stretch position is held for ten seconds, counted aloud by the client. (Website of LSVT Global 2017.)

The second tasks are repetitive and directional movements done standing. They include stepping forward, sideways and backward with large upper limb movements included. Final daily exercises are rocking movements, the forward rock and reach and the sideways rock and reach. (Website of LSVT Global 2017.)

The third daily tasks are functional component movements. These are up to five exercises that are derived from daily activities. Sit-to-Stand is always the first one for every single client. With the help of the therapist the client identifies simple components of these tasks which are then done with the same amount of effort and intensity as the aforementioned daily exercises. Each exercise is done for five repetitions. (Website of LSVT Global 2017.)

The fourth task is called BIG walking, where the client practices using the newly formed big amplitude for gait training. The purpose is to take bigger steps with bigger arm swings, with the therapist providing first a model and then shaping the client’s movement so that it will big enough, but not too big. (Website of LSVT Global 2017.)
Second half of the treatment session is spent on Hierarchy tasks. Hierarchy tasks are increasingly complex tasks or sequences of tasks that are specific to the client’s goals and needs. The aim is to transfer the increased, rescaled level of amplitude from the daily exercises into an increasingly complex activity that is context specific and variable. The tasks become more and more difficult week by week and the client is challenged to multi-task. Difficulty can be increased by increasing amplitude and complexity, duration or with the addition of a cognitive task. (Website of LSVT Global 2017.) All the exercises and a typical session are depicted in Figure 2.

The client is also given homework which should be done once on treatment days and twice on non-treatment days. These sessions take approximately 15 to 20 minutes and include all the daily exercises for half the volume, the functional component tasks, BIG walking and a carryover assignment. A carryover assignment is a daily task where the client uses big amplitude to override slow movements so that they may complete this task in the future independently. (Website of LSVT Global 2017.)

Table 1: Outline of a typical therapy session in LSVT BIG.

<table>
<thead>
<tr>
<th>Name of ex.</th>
<th>Position</th>
<th>Aim</th>
<th>Reps &amp; Sets</th>
<th>Time spent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max sustained</td>
<td>Seated</td>
<td>Use BIG amplitude</td>
<td>16 per exercise (2)*</td>
<td></td>
</tr>
<tr>
<td>Repetitive &amp; Directional</td>
<td>Standing</td>
<td>Use BIG amplitude</td>
<td>20 per exercise (5)*</td>
<td></td>
</tr>
<tr>
<td>Functional components</td>
<td>Varies</td>
<td>Apply BIG to small daily tasks</td>
<td>5 per exercise</td>
<td>30-45 minutes for all daily tasks</td>
</tr>
<tr>
<td>BIG walking</td>
<td>Walking</td>
<td>Apply BIG to gait training</td>
<td></td>
<td>5-10 minutes</td>
</tr>
<tr>
<td>Hierarchy tasks</td>
<td>Varies</td>
<td>Apply BIG to complex movement sequence</td>
<td></td>
<td>15-30 minutes</td>
</tr>
</tbody>
</table>
6 AIM AND OBJECTIVE OF THE THESIS

The objective of this thesis is to prepare a presentation to physiotherapy students that introduces the LSVT BIG protocol using the most recent evidence based knowledge of Parkinson’s disease and the LSVT principle within the context of brady- and hypokinesia.

The aim is to disseminate this information around Finland and raise awareness of a new type of intervention that would improve the physical capabilities of people with Parkinson’s disease.

7 THE PRESENTATION

The presentation was included in the classes’ timetables and it was held in the new SAMK campus in Pori the 12th of October 2017, which their teacher had reserved and scheduled for the author. An hour and a half was reserved for the entire presentation. There was no time reserved for questions, instead, the students were able to ask questions whenever they wanted. The presentation began with the students showing whether or not they were especially interested in neurological physiotherapy. This was done by a show of hand and there were no students specifically interested in the topic.

The key points of the lecture were in order the importance of having multiple perspectives, the pathology and clinical manifestations of bradykinesia and finally the LSVT BIG protocol. The LSVT BIG protocol was not the focus of the lecture, instead, the author’s view on the perspective of LSVT, how the patient is seen in LSVT BIG and how this differs from the conventional method in physiotherapy in Finland today and why it is important to handle multiple perspectives was the central idea.

7.1 Feedback

No formal feedback was asked. Instead, at the end of the lecture, after all the questions, the author asked the audience to provide some form of feedback on the presentation.
Most of the feedback was very positive. Most people said it was the author’s enthusiasm, vivid language and multiple examples from real life that made the lecture interesting even though they were not interested in the topic in the first place. Also the depth of knowledge on the author’s part was apparent and one student felt inspired to change their own thesis topic. It was also mentioned that the lecture pace was relatively slow, and the topic was handled incredibly thoroughly which, given the audience, could have been made slightly faster, for certain groups most likely will know the importance of perspective and the anatomy of the central nervous system to name a few.

8 THESIS PROCESS

8.1 Choosing a thesis type

This thesis is focused on the operational aspects. Such a thesis aims to respond to a practical need or problem by designing for example events, exhibitions, fairs and related planning and organization, marketing campaigns or computer programs to name a few. (Hakala 2004, 23-27; Vilkka & Airaksinen 2004, 9-15.) The initial method was writing a journal article. However, this proved to be an unpleasant endeavor and it was concluded that the author is more competent in presenting the topic to a live audience verbally instead of through text.

It is good to start planning a thesis by getting to know the subject matter and reading previously written text to formulate an individual perspective and an opinion on the subject. It is also important to determine the objective of the thesis so that it is consistent with the demand. (Hakala 2004, 23-27.) A well-designed plan eases the writing process and the progression of a thesis. A thorough examination of a subject enables the author to design a good project (Vilkka & Airaksinen 2004, 9-15).

The author noticed a demand for such a lecture in 2015 when they learned about the LSVT BIG protocol. At the time there were no LSVT certified physiotherapists in Finland. This was surprising as there is a lot of evidence supporting the impact of high
amplitude exercise on bradykinesia. More importantly, the impact of bradykinesia on the quality of life of a person with Parkinson’s disease is well documented, but the means of affecting it in the guidelines for example is very limited. The author wanted to spread this information as best as they could around Finland, and.

The author was certified to administer LSVT BIG in March 2017 and the planning of the lecture began in June 2017. It should be noted that the lecture follows a very similar structure to that of the unpublished article so it could be said the planning of the framework began as early as January 2015.

Amassing the theoretical basis for the entire thesis was done over a period of 4 months, starting from January 2017. The sources were numbered and the topic had to be narrowed down significantly in order to fit the general brackets of a bachelor’s level thesis. Most of the effort was spent on refining and planning the lecture itself. This was done through multiple conversations with people of all backgrounds aging from 21 to 26 and rehearsals recorded on a smartphone. The author wanted the lecture to be structured enough so that it would not stray from the topic itself, but alive enough in the sense that it could be shaped and modified on the go depending on the audience’s responses and reactions to items in the lecture. Thus, the power point presentation that was made to support the lecture was very minimal containing only three sentences per slide on average, and mainly pictures. This would enable the author to stay in topic and reduce any additional cognitive load on the audience allowing them to fully concentrate on the speech element.

8.2 Target audience

The target audience were the third year physiotherapy classes of SAMK with the other being an international class with people whose native language is not Finnish, thus the lecture was held in English as all the students were competent enough to listen. This also meant the language used by the author was simplified to the degree that everyone would understand as much as possible.
9 DISCUSSION

9.1 Evaluation of the process

The process of writing this thesis was an incredible exercise in critical thinking. Not only was it necessary to gather data on a topic that is not widely discussed in the context of Parkinson’s disease, bradykinesia, but also present it in a way that is interesting to an audience that is by default not interested in or aware of the topic. The applicability of this experience in the future is immense as sometimes for a physiotherapist it will be necessary to discuss uncomfortable and novel ideas with a client for example. It was also of great importance to read data on the LSVT BIG protocol and Parkinson’s disease in a manner that would help build a pattern of thinking about any topic in physiotherapy, hence the numerous examples of real life experiences using multiple view points on any given issue. This made it so that the audience gained tools that are useful no matter what branch of the medical field they decide to focus on.

The lecture lasted an hour and 25 minutes which was within the time frame set by the teacher. Most of this was spent on the lecture itself and there were not as many questions. It could be that most of the students were uncomfortable asking questions in English given their Finnish background, or that the topic was very novel. However, the questions became more abundant as the lecture progressed.

The powerpoint functioned well as a figurative spine for the lecture, but it was clear that using the powerpoint itself should have been practiced more. There were a few times were the author forgot the next slide, but it was not a significant issue. The lecture overall was very fluid and after the first ten minutes of nervousness the lecture started to flow well. It was also very interesting from the author’s point of view to see the students’ reactions to certain examples from popular culture and physiotherapy as that allowed for some improvised change in the way of presenting the topic at hand.

As mentioned earlier, the importance of having multiple perspectives on a given topic was one of the key points of the lecture. The author thinks they provided more than enough examples from a wide variety of fields of science and culture on most of the
ideas discussed and this also made the students interested in the topic. There was also quite a lot of laughter at times so the lecture was humorous as well and in that regard, a success.

In the author’s mind, the LSVT BIG protocol itself could have been explained much more thoroughly than it was. There is a reason why it was not the focal point of the lecture and these reasons are explained in the next part of this thesis report. However, the way the exercises can be adapted could have been explained better. There was also some confusion regarding the BIG movements. One student thought of BIG movements as making movements that are “as BIG as possible”, when the goal of the protocol itself is to make movements that a normal in range of motion, but BIG in the mind of the subject with an impaired proprioceptive system.

There could have been a method of gathering feedback from the students in the form of a questionnaire for example to provide raw data on how they experienced the lecture, what they thought was thought provoking for a physiotherapy student and what could have been improved given the audience consists of physiotherapy students. This would help the author design the lecture better for said audience if it is presented again sometime in the future.

9.2 Ethics and credibility

The reasons that LSVT BIG was not reviewed more thoroughly in this thesis are many. First of all, the author of this thesis has never had the ability to administer the entire protocol to a single client which would make it disingenuous to present views and experiences of whether or not this protocol is good and what type of client would benefit most from it. The focus was on what a student can learn from getting certified in LSVT BIG and the method of approaching a client in LSVT BIG as that is something the author has firsthand experience of. The importance of finding a common nominator and distilling it into a point of extreme focus was the underlying theme of the presentation.
Second reason for not presenting LSVT BIG in a detailed manner is that currently in the Finnish healthcare system, there are very limited possibilities of even attempting to administer the entire protocol to a client as this brings up the question: “Who is going to pay for it?” It is still very novel an approach to the treatment of Parkinson’s disease and it has limited evidence supporting its efficacy which means most clients would have to pay for it themselves, and that would require incredible ability to persuade the possible clients. The LSVT BIG is also said to provide the best benefits for people with Parkinson’s disease who are recently diagnosed or whose condition has not progressed very far. In Finland, the people with Parkinson’s disease who are hospitalized or sent to a rehabilitation facility have their condition progressed to the degree that the LSVT BIG protocol would not be as efficient as possible as a treatment tool.

9.3 Conclusions

To bring LSVT BIG to the forefront of rehabilitation for people with Parkinson’s disease in Finland would require a great paradigm shift in the overall approach. It would be necessary to go from a maintaining approach to a high intensity approach with the goal of drastically improving the physical capacity of the people with Parkinson’s disease.

The author is not trying to judge the conventional physiotherapy approach as it does provide good results for a lot of the clients. However, there are people who are willing to participate and would benefit more from very intense programming, and this is not provided currently in Finland on a large scale, or a default option.

Thus, the author of this thesis finds it important to understand the fundamental mode of thinking when administering the LSVT BIG protocol and to find patterns and principles that can be applied one way or another to other forms of therapy. The goal is to use it as an example of a new way of thinking about rehabilitation and bringing to light a point of view that could lead to the aforementioned paradigm shift and a better selection of rehabilitation methods for all people affected by Parkinson’s disease.
REFERENCES


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