

Running head: MULTIPLE SCLEROSIS AND QUALITY OF LIFE

Multiple Sclerosis and Personality:

How Changes in the Brain Affect Quality of Life

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### Abstract

Multiple Sclerosis (MS) is a chronic illness that can affect every aspect of a patient's life. The cerebral deterioration and cognitive dysfunction trademarked by this disease leads to changes in mood, emotion, behavior and function. These changes, especially the changes in function, as well as a diagnosis of an incurable, neurological disease have an incredible impact on a patient's quality of life and personality. The depression and perceived helplessness common in MS patients may not stem from the diagnosis alone, but from atrophy in the brain and decline in neurological status. This paper gives a brief history and description of MS symptomology and its impact on quality of life and explores the relationship between cerebral atrophy and changes in personality.

Multiple Sclerosis (MS), or disseminated sclerosis, is an inflammatory, autoimmune disease of the central nervous system (the brain and spinal cord), where the body's immune system attacks its own tissue, specifically nerve tissue. The nerve tissue, also known as white matter, deteriorates leading to cognitive decline. This process is known as demyelization. Demyelization is named for the breaking down of the fatty protective layer around the brain's neurons called the myelin sheath. The loss of the myelin sheath not only brings about many forms of cognitive dysfunction but also many forms of body weakness, muscle stiffness, fatigue, and sensory loss. Multiple Sclerosis gets its name from the sclerosed or scar tissue left behind by the deterioration of the nerve tissue. This scar-like tissue that replaces the damaged myelin further interferes with nerve signaling. The deficits mentioned above occur as a result of the reduction of action potentials along the axons due to the build-up of scar tissue. In the United States, MS has been diagnosed in approximately 400,000 people (NationalMSSociety.org, 2008). Two to three times as many women have been diagnosed with MS than men, and it seems more common in the Caucasian population. Each year, 10,000 new cases are reported (NationalMSSociety.org). Even though it may appear that more and more cases of MS are diagnosed each year, epidemiologists have not found evidence that incidences of MS are increasing but have just become more easily recognized. Awareness of MS and more accurate diagnoses are most likely accountable for higher numbers of people diagnosed with this disease now than in the past.

### History of Multiple Sclerosis

Though some historical accounts cite that MS may have been first indicated before the 14<sup>th</sup> century, one of the most well-documented cases of probable MS was St. Lidwine of Schiedam, Holland (1380-1433). At 15 years of age, Lidwine suffered a skating accident after

which her health declined. It is recorded that she experienced sensorimotor symptoms, gait ataxia, paralysis and sensation disturbances and visual deficits. The first certain clinical account of MS was recorded by Auguste d'Este (1794-1848) as an autobiography. In a relapse-remitting pattern, he documented numbness in his legs, vertigo, loss of strength in his limbs, profuse perspiration, as well as other symptoms now known to be common in MS. With MS being so multifaceted, treatment during the 1800's was unscientific and unhelpful, as doctors were not certain of the diagnosis. d'Este was slapped to regain his strength, given leeches for diplopia (double vision) and hot baths while he was covered in plaster to relieve his pain (Butler & Bennett, 2003). It is now understood that heat exacerbates the symptoms of MS (Uthoff's symptom), so often these treatments only made the condition worse. Direct current electricity (Galvanism) was commonly administered directly to the muscles of patients suffering from MS and similar disturbances. Treatment for d'Este also included ingesting strychnine, nitric acid, ammonium carbonate, and mercury as well as many other noxious substances. Heine, an acclaimed poet in the early 19<sup>th</sup> century believed to have died from MS, wrote that he was overdosed on opium and morphine and was bloodletted. His symptoms included dysarthria, incontinence, facial hyperesthesia, facial paresis and other symptoms similar to d'Este and was given several diagnoses such as neurosyphillis, spinal muscular atrophy, spinal tuberculosis, and encephalomyelitis (Heine, 1948).

Multiple Sclerosis as it is known today was first comprehensively described by Jean-Martin Charcot (1825-1893), referred to as the father of clinical neurology. He noted that the lesions on the brain were not frequently found in gray matter, and conjectured that the variations in the places of lesion might be represented by different functional disorders.

Charcot focused on tremors, trying to differentiate MS from paralysis agitans (Parkinson's disease) by evaluating the patient's handwriting and often made a diagnosis according to the type of tremors the patient was experiencing (Butler & Bennett, 2003). His somatic studies on nystagmus and vertigo led Charcot to be the first in the medical community to document the cognitive deficits in MS patients, observing what he called *stupid indifference* characterized by slow formation of ideas, memory deficits and the blunting of emotional and intellectual abilities (Charcot, Lecture VII, 1868/1877).

## Symptoms of Multiple Sclerosis

### Cognitive Symptoms

The cognitive dysfunctions of MS are highly correlated to brain atrophy. The cognitive deficits most commonly found affect memory, learning, attention, and information processing. Free recall and delayed intentions (such as remembering to do something later) are the aspects of memory most affected. Attention is often compromised, as MS patients often complain of distractibility and difficulty in multitasking. As memory and attention are undermined, learning new tasks have shown to be taxing and sometimes stressful. In information processing, transfer of information between hemispheres of the brain in MS patients is slowed (Bagert, Camplair, & Bourdette, 2002).

Depression, coupled with feelings of perceived disability, discouragement and helplessness, is also prevalent in patients with MS somewhere between 37% and 54% of its population (Bagert et al., 2002). Neuroimaging has shown a relationship between depression and cerebral atrophy (Feinstein, 2004) so it would be incorrect to say that the depression is in response to having the disease alone.

Multiple Sclerosis depressive syndrome seems to overlap with major depression, though some feelings are more typical, such as irritability, frustration and discouragement than are poor self-esteem or guilt, which are facets of primary depression. According to Feinstein, suicide rates are up to 7 times higher in MS patients than in the general population or in populations of other neurological disorders, occurring in approximately 30% of patients. This may be attributed not only to emotional changes brought on by brain atrophy, but also to the personality alterations in individuals affected by MS. Depressive disorder, as well as physical deficits (discussed later), may contribute to the inability to participate in once-usual activities, and diminish a MS patient's quality of life (also discussed later). Due to these changes, personality is also affected. Though, with therapy and pharmacological treatment, the depression that most MS patients experience is treatable and should not severely impair quality of life.

Other mood disorders and cognitive deficits affect MS patients though, none as prominent as depression. Bipolar Affective Disorder is two times more prevalent in MS patients than in the general population but this disparity has not been shown to be caused by steroid treatment alone and may be a result of a genetic predisposition in females (Feinstein, 2004). Though there are not sufficient data to claim a prevalence rate, psychosis known as the presence of hallucinations or delusions, has been documented in few MS cases and may be considered more than chance (Feinstein), though there have not been enough studies to produce any significant data. Multiple Sclerosis patients with psychosis were significantly more likely to have higher temporal horn lesion load than psychosis patients without MS. Euphoria may also be seen in MS patients, and just as with mania in those with Bipolar Affective Disorder or psychosis, treatment may be difficult if complicated by agitation, motor overactivity or delusional thinking.

Pathological Laughing and Crying (PLC), defined as laughter without mirth and crying without sadness, has also been linked to about 10% of MS cases (Feinstein). Neuroimaging studies have not been conducted with MS patients, but those with PLC are likely to have frontally mediated cognitive affects. With PLC patients, the patients are aware of and confused or frustrated by their incongruent emotions.

### Somatic Symptoms

Demyelization, which reduces the ability of axons in the brain to carry out action potentials, results in many somatic symptoms and deficits. Chronic fatigue is a common comorbidity or symptom of MS and is often debilitating. Attempts to find a causal relationship between MS and fatigue have not shown any definitive results (Bagert et al., 2002) but fatigue is cited as the number one symptom suffered by MS patients. Visual symptoms are probable in MS and are typically one of the first signs of the disease. Nystagmus (i.e., jittery eye movement) and diplopia are earlier stages of optic nerve deterioration, subsequently optic neuritis in the form of eye pain, blurred or loss of color vision and possible blindness. Brain lesions along the motor pathway give rise to paresis or paralysis, spasticity (i.e., muscle stiffness that restricts movement), muscle spasms, atrophy and dysarthria (i.e., slurred speech). Dysarthria is not to be confused with dysphasia, impairments to speech comprehension and production as seen in MS patients which is actually a cognitive, not motor, deficit. Restless Leg Syndrome, characterized by *creepy, crawly* sensations especially at night, may be a harbinger for MS. An archetype of MS is the L'Hermitte's sign, which is an indicator of lesions in the cervical spine described as an electrical shock or buzzing sensation felt when moving the head, especially in a forward and down motion. Other sensory deficits include anaesthesia, complete numbness or loss of sensation in a limb and trigeminal neuralgia, acute and chronic pain in regions of the face and jaw.

Multiple Sclerosis is associated with many types of bowel, bladder and sexual dysfunctions as well as many sleeping, swallowing and respiratory problems. Most outwardly notable symptoms in MS patients are deficits in coordination and balance. Many forms of ataxia (i.e., loss of coordination) are common such as cerebellar ataxia caused by lesions in the cerebellum. The cerebellum is responsible for synchronizing voluntary muscle movement, so lesions in this area cause difficulty walking (i.e., gait ataxia), reduced control of range of movement, and inability to maintain a steady posture. Vestibular ataxia is caused by lesions in the brain stem resulting in loss of balance, dizziness, and vomiting. This is not an exhaustive, comprehensive list of deficits presented by MS patients but are the most customarily suffered. Also, each case of MS is unique and each individual diagnosed with it will experience different symptoms and deficits. With as severe and restrictive as some of these symptoms are, some MS patients find themselves withdrawing from enjoyable or necessary daily activities and may become isolated. These tendencies intensify depression or feelings of loss and over time may become habits, where the individual's personality has altered under the weight of their depression and disabilities. This emotional change in personality combined with cerebral atrophy in the frontal lobes (which seems responsible for personality traits or characteristics) will lead to permanent changes in an individual's personality and quality of life.

#### Quality of Life and Multiple Sclerosis

Rating scales and objective measures have recorded MS patients' reduced ability to perform everyday activities due to their cognitive and somatic deficits. The MS Quality of Life Scale (MSQOL-54) is a multidimensional quality of life measure that summarizes two scores: physical health and mental health.

The MSQOL has 12 subscales: physical function, role limitations-physical, role limitations-emotional, pain, emotional well-being, energy, health perceptions, social function, cognitive function, health distress, overall quality of life, and sexual function. There are also two single-item measures: satisfaction with sexual function and change in health. The scores given by the MSQOL are meant to give insight on the impact MS has on the individual's daily life. With this scale, an individual can assess how MS effects their daily activities and their ability to perform these activities. In later stages of MS, when participating in necessary activities and special interests become complicated by physical and mental deficits, diagnosed individuals may remove themselves from activities they once enjoyed. This lack of fulfillment and change in routine can lead to more instances of depression, or confinement and detachment from loved ones, which can further lead to alterations in personality.

#### Effects on the Brain and Personality

Benedict et al. (2008), precisely said that *there has been gaining interest in the ways personality is affected by cerebral disease and how personality influences brain activity and adaption to illness*. Patients who have been diagnosed with MS are believed to undergo mood, and ultimately personality changes due to atrophy of nerve tissue in the brain and the stress and implications of having an unpredictable, incurable neurological disorder. Personality is defined as underlying traits that form the basis for individual differences in behavior (Benedict et al.). It is already known that traumatic brain injury can result in change in personality, such as in the well-known case of Phineas Gage, who survived a tamping rod piercing his frontal lobe in 1848, resulting in Phineas *no longer being himself*. Cognitive dysfunction appears in 70% of the MS population (Kalmar, Halper, Guadino, Moore, & DeLuca, 2008). These deficits and difficulties debilitate and undermine quality of life in persons diagnosed with MS.

Cognitively impaired individuals with MS are more likely to not have employment (Kalmar et al., 2008). Rao et al. (1991) found that MS patients with cognitive dysfunction had more sexual dysfunction, greater difficulty with household related tasks, greater inability to drive a car, and fewer social interactions. Studies have shown that neuropsychological deficits undermine the ability for MS patients to manage stressful life events (Mohr & Goodkin, 2003). With a disease as multifactorial as MS, persons with it must markedly reform their former way of living. The inabilities and dysfunctions that MS patients suffer create mental and physical barriers to activities they once enjoyed or were able to accomplish. These barriers may help the person develop a sense of discontent or unfulfillment in life and personal accomplishments. This train of thought is what most often leads people diagnosed with MS to major depressive disorder. The depression along with changes in the brain, cognitive and physical ability and mood has profound alterations in personality. Benedict et al. (2008), conducted a rigorous study with 44 MS patients, whom were all interviewed, neuroimaged using a magnetic resonance imaging (MRI) machine, had brain tissue extracted and were given several verbal, learning and personality tests. A reduction in extraversion (i.e., tendency to be outgoing and sensation seeking), openness (i.e., desire for new knowledge, ideas, and experiences), conscientiousness (i.e., extent of task-orientation and achievement-striving), and lower volume of the cerebral cortex were obtained. There was sufficient evidence to claim that cortical atrophy in MS is associated with adverse impact on personality (Benedict et al.).

### Etiology of Multiple Sclerosis

Multiple Sclerosis occurs when the myelin basic protein and proteolipid are recognized as foreign by the immune system, and is attacked. The etiology of this disorder is still largely unknown. The primary hypothesis of the origins of MS cites viral infections (either through

vaccination or infection) in genetically susceptible individuals. Viral infections are also known to exacerbate MS lesions in patients, suggesting that viruses are directly involved in the disease.

In a study conducted by Gronning et al. (1993), 55 people diagnosed with MS and 200 controls (participants not diagnosed with MS) were given a questionnaire and interviewed by the authors. Analysis showed a slightly statistically significant trend ( $p = 0.06$ ) showing developing measles in adolescence is associated with increased risk for MS. The inflammatory autoimmune and demyelization aspects as well as the scar tissue formation are characteristic of infectious diseases. Also shown were higher instances of tonsillectomies in MS patients during adolescence compared to controls; tonsillectomies decrease immunoglobulin (fewer antibodies to fight off disease or infection) in the immune system.

In an experiment conducted by Lycke et al. (1996), the correlation between viral infection and MS was investigated using antiviral therapy acycloguanosine (acyclovir). Sixty MS patients were divided into either a placebo group or an acyclovir treatment group (800 mg, 3 times daily for 2 years) based on exacerbation rates (focal disturbance of function affecting white matter lasting more than 24 hours) determined during a 2-year pre-study period prior to the experiment. Exacerbations were classified according to severity and frequency. The exacerbation rate (determined by the data and primary outcome measure) of the placebo group was shown to be 1.57 ( $n = 30$  and 94 exacerbations), and the treatment group's rate was found to be 1.03 ( $n = 30$ , and 62 exacerbations). The acyclovir mean annual exacerbation rate was reduced by 0.44 while the placebo group had an increased mean annual exacerbation rate of 0.27 (Lycke et al.). There seems to be sufficient evidence not only to connect antiviral treatment to reduced exacerbation of symptoms in MS patients, but also to associate viruses with MS disease origins.

The multifactorial evidence of the causes of MS stems from non-human and twins studies. A strong genetic influence on MS is shown in the concordance rates of 5% in dizygotic twins and as much as 31% in monozygotic twins, though environmental or exogenous factors must still be present for the disease's development (Norris, 1997). Epidemiological studies have shown MS to be more common in northern latitude than in southern latitudes. Multiple Sclerosis seems to occur within clusters, making it more common in some geographic regions than in others and this has made the presence of environmental factors more obvious.

#### Treatment of Multiple Sclerosis

To treat MS, it is necessary to treat its symptoms as well. Treatment of symptoms would include therapy or pharmaceuticals for depression, or when mood is obstinately low electroconvulsive therapy (ECT) may be necessary though reports of exacerbation in the MS itself may be a side effect (Feinstein, 2004). When selective serotonin reuptake inhibitors (SSRIs) do not lessen the depression or mania in MS patients, lithium may be used to alleviate depression and euphoria in bipolar affective disorder. Initially trials with Interferon Beta-1b (i.e. a substance that inhibits certain white blood cells) suggested that a side effect of interferon treatment is suicide ideation, as many suicide attempts and one completed suicide were exhibited within the treatment group, as opposed to none in the placebo group (Pandya, 2002). Recently, beta interferon (given in the form of a subcutaneous injection) has been shown to slow the growth of white blood cells, stopping their production of myelin-destroying compounds and also corrected a deficiency in T cells, the white blood cells that control the immune system (sfn.org, 2004). Beta interferon inhibits the action of gamma interferon. Studies with gamma interferon have provided evidence that MS attacks are triggered by immune-system-growth factors.

Fewer hospitalizations and severe attacks were reported in MS patients taking beta interferon. MRI scans showed fewer brain lesions than patients on a placebo treatment. Aside from the possible side effect of major depressive disorder, beta interferon produces inflammation such as itchy rashes at the injection site and influenza similar symptoms, like fever, chills, runny or stuffy nose and body aches.

Cognitive-behavior therapy (CBT) is a necessary part of treating MS, especially when the individual is experiencing depression. Mohr and Goodkin (2003) concluded that antidepressant medications coupled with CBT were superior in treating depression in MS patients than supportive-expressive group psychotherapy or any insight-oriented therapies. This same study also showed a correlation between successful CBT and a significant drop in T-cell production of gamma interferon, which is linked to the viral origins of MS and relapse and exacerbation in symptoms. Treating emotional and mood dysfunctions as well as depression can be administered over the phone for those with mobility problems. Physical impairment, issues with transportation and lack of financial resources are the most common reasons cited by patients as impediments to accessible therapy. In a study conducted by Mohr et al. (2005), only 10% to 45% of patients diagnosed with depression ever make an appointment for therapy, and of those that attend the initial appointment, nearly half will drop out before treatment is complete. In MS patients, an 8-session telephone administered CBT has been shown to be more effective than usual care in reducing depressive symptoms (Mohr et al.). Telephone therapy provides the necessary treatment for individuals diagnosed with MS while allowing a solution for the patient's needs, desires or conflicts, such as avoiding stigma, child care and regular appointment scheduling, lack of time or physical immobility.

## Conclusion

Multiple Sclerosis has no known cure - yet. Advancements in medical science and technology have proven tremendous progress in just the last few years in understanding this disease, its symptoms, how to diagnose and treat MS. The future of MS depends on discovering the certain pathogen, believed to be a virus, which causes the disease. There has been speculation that a protein (antigen) from a common pathogen that resembles an antigen derived from myelin may be of importance in the development of the disease. Without knowing the exact origins of MS, it is difficult to counteract its damages.

Unlike most other neurodegenerative diseases, the cerebral atrophy caused by MS can deteriorate axons in the frontal lobe of the brain, which are considered the neurological underpinnings for the development of personality. This kind of axonal atrophy is not necessarily found in many other neurological diseases. Lesions in the frontal lobe combined with changes in body and mood often lead MS patients to act incongruently with how they acted previous to suffering from the disease. This decline in function, quality of life and lack in personality is especially despairing for not only the individual with MS, but their loved ones. Though, an insurmountable amount of growth in the field of MS research has led to the knowledge on how to treat it, its impact on the brain, and how to have a relatively normal life diagnosed with it – it is still imperative that the origins of the disease be found. Once the cause of MS is determined and a treatment discovered, individuals with the disease can progress toward living the life they had before the disease and being themselves again. Specialists in the fields of medicine, psychology, pathology, epidemiology and many more are working towards bringing to light the uncertainties of MS and finding a cure.

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