Dr. B. Duncan McKinlay
Psychologist

London, ON, Canada
url. www.lifesatwitch.com

Website:
Tourette Syndrome • OCD • ADHD
Sensory • ODD • IED/'Rage'

Publishing:
Nix Your Tics! Eliminate Unwanted Tic Symptoms: A How-To Guide for Young People

Increasing Our Knowledge of Gilles de la Tourette Syndrome Through The Use Of Electroencephalograms: A Summary Of Findings

June 1997

NOTE — THIS PAPER WAS WRITTEN AS AN EEG COURSE REQUIREMENT IN DURING THE FIRST YEAR OF MY MASTERS PROGRAM. HENCE IT IS SOMEWHAT DATED AND BASED ON THE LEVEL OF KNOWLEDGE I POSSESSED AT THAT TIME. PLEASE READ WITH THIS IN MIND AND WITH A GRAIN OF SALT!

Gilles de la Tourette Syndrome, or Tourette's Syndrome (TS), is a genetically inherited, neurological disorder of the dopaminergic and serotinergic systems, and is thought to be localized in both the basal ganglia (particularly the caudate nucleus), and prefrontal cortex (Comings, 1990). While TS was traditionally characterized by both motor and vocal "tics" – involuntary twitches -- which would wax and wane in severity, it has more recently been re-defined as a disorder of general disinhibition (Comings, 1990). Normal inhibitory controls malfunction or fail, most acutely under moments of stress, which lead to random movements and sounds, obsessive thoughts, impulsive behaviour, attentional problems, low frustration tolerance, or any combination of the above. While TS was initially little more than an obscure and bizarre curiousity, this new, more accurate, characterization of it as a subtype of a larger "Generalized Disinhibition Disorder", and recognizing it's comorbidity and overlap with many other more prevalent disorders, has prompted more interest and research into this syndrome than ever before.

From the beginnings of their trade electroencephalographers seemed to have established an interest in TS literature in this domain can be found as early as 1958 (Dolmierski & Klossowna, 1958). The electroencephalogram (EEG) abnormalities found in TS'ers were far ranging, and seldom agreed in severity, type, and prevalence (which ranged from 25-100%) (Shapiro et. al., 1978). Kelman (1965) found that 42.1% of his 19 subjects had abnormal EEG patterns. He described slow rhythms, non-focal spiking, and infectious cerebral changes suggestive of a temporal lobe disorder. In 6 of his 7 patients, Feild reported bi- and triphasic sharp activity in the Rolandic and Sylvian areas (Feild, 1966). 10 of 18 subjects were found by Lucas and Rodin (1973) to have abnormal EEG's: 5 had mildly dysrythmic, nonspecific theta activity, 2 experienced generalized dysrythmia, and one had random waves in both the delta and theta frequencies, maximized in the temporal and occipital areas.

Other abnormalities documented include occasional diphasic sharp waves, bisynchronous over the parietal region, and generalized short bursts of slow waves (Corbin et. al., 1968), activity slower than the normal basic frequency in 12.5 % of TS subjects (Krumholz, 1983), asymmetrical temporal rhythms and hypersynchronous or paroxysmal discharges similar to epilepsy (Dalmierski and Kloss, 1962), excessive theta activity and bilateral fronto-temporal sharp waves between episodes of tics and rushing thoughts (Logue, 1973), epileptiform alterations found 5 to 7 times more frequently than in the normal population (Verma, 1986), and spikes,polyspikes, or spike-like discharges with tics or tic impulses in postencephalitic patients who had acquired Tourettism (Sacks, 1982).

In an attempt at parsimony, Cohen, Bruun, and Leckman (1988) claimed most researchers agree that minor, nonspecific abnormalities on the EEG record occur more frequently among TS'ers than within the normal population. While this was true, Shapiro (1978), in a comprehensive review of the literature, stated that the

Dr. B. Duncan McKinlay
Psychologist

London, ON, Canada
url. www.lifesatwitch.com

Website:
Tourette Syndrome • OCD • ADHD
Sensory • ODD • IED/'Rage'

Publishing:
Nix Your Tics! Eliminate Unwanted Tic Symptoms: A How-To Guide for Young People

majority of researchers do not adequately describe the abnormalities seen. More importantly, he noted that the data was not broken down by age; while Shapiro had found that 48.4% of his 79 patients displayed abnormal EEG's, when they were split by age, he found that 68.8% of subjects under the age of 17 had abnormal readings, compared to only 26.7% of subjects older than 17. It has been suggested that this age difference reflects the detection of abnormalities associated with delayed maturation (Obeso, 1982).

In further attempts to "clean up" the research, other experimenters began to account for whether subjects were currently taking medications (typically dopamine antagonists) or not, and whether there existed any confounding neurological impairments. Verma, Syrigou-Papavasiliou, and LeWitt (1986) found that when an unmedicated, neurologically, and intellectually intact TS sample was used, a mere 20% showed any unusual EEG patterns. Krumholz et. al. (1983) controlled for medication, neurological dysfunction, and mental retardation, and found that only 5 of 40 patients exhibited any EEG abnormalities; these were generally described to be excessive nonspecific slow activity (excessive slow background activity, centro-parietal spikes, and sharp waves). From this, Krumholz et. al. then concluded that eliminating these extraneous variables greatly reduced the frequency of abnormal EEG readings in the TS population. The picture may not be that simple, however. Shapiro (1978) claimed that when TS'ers with minimal brain dysfunction (MBD) were compared to a sample with only MBD, the former group had more moderately abnormal and markedly abnormal EEG's. It is possible that factors such as MBD interact with TS, and are not simply confounding variables. Another problem with simply factoring out other neurological problems is that there is still considerable debate concerning how exactly TS is to be defined; depending on one's interpretation of the disorder, samples may or may not include individuals who also have obsessions, attentional problems, or even seizures (Comings, 1990). Indeed, Shapiro et. al. observed that a higher incidence of minimal neurological dysfunction exists among the TS population; until this correlation can be explained, one must be cautious in partialling out EEG abnormalities due to this associated problem.

Three variables which seem to make little difference are sex, tic severity, and state of consciousness. Verma (1986) found that the ratio of males to females with abnormal EEG readings was not significantly different from the ratio of males to females in the sample. In most studies reviewed, more men were used than women – this is to be expected, however, since TS is recognized to be three times more prevalent in men than women. Regarding severity of tics, Krumholz's five patients who exhibited excessive nonspecific slow activity after the effects of medications, neurological dysfunction, and MR had been factored out ranged from mildly Tourettic (n=2) to severe (n=1) (Krumholz, 1983) Age of onset and duration of tics were also inconsequential. Finally, state of consciousness (awake versus asleep) yielded nothing of significance (Krumholz, 1983).

Moving away from the general EEG recording, some researchers have concentrated their attentions on unusual activity surrounding ticking. Obeso claimed that there was no evidence of paroxysmal activity time-locked to tics (Obeso, 1982). However, he did find that the usual pre-movement EEG potentials, typically recorded over the sensory motor cortex contralateral to the body movement, were absent in six Touretters when ticking. Instead of the expected slow potential, four of the six exhibited unusual negative, abrupt potentials 100 ms prior to the movement onset: Obeso thought this to be a motor potential, possibly a discharge of pyramidal tract neurons (Obeso, 1982). This pattern had nothing to do with the movements in and of themselves; Obeso ascertained this by asking the Touretters to voluntarily mimic those same tics. When they did so, the usual pre -movement potentials appeared (Obeso, 1982). As the usual pre-movement EEG changes are believed to take place only in preparation before willed, voluntary movements, Obeso concluded that the tics in TS or not psychologically [cortically] generated (Obeso, 1982). He also concluded that, since pre-movement potentials were not being detected at the onset of a "true" TS tic, the structures responsible for TS are most likely buried deep within the brain. This is supported by the majority of the literature today that points to the basal ganglia

Dr. B. Duncan McKinlay
Psychologist

London, ON, Canada
url. www.lifesatwitch.com

Website:
Tourette Syndrome • OCD • ADHD
Sensory • ODD • IED/'Rage'

Publishing:
Nix Your Tics! Eliminate Unwanted Tic Symptoms: A How-To Guide for Young People

(Comings, 1990), and somewhat agrees with Corbin et. al.'s belief that, while cortical neuronal populations are involved in TS, they are subject to the influence of deeper structures (they suggested the reticular activation system). Corbin et. al. supported their hypothesis through the observations that ticking decreases when subjects are asked to problem-solve, and increases when subjected to auditory and proprioceptive stimuli (Corbin et. al., 1968). Lastly, post-movement potentials of the tics have not ever been found to significantly differ from other planned post-movement potentials (Obeso, 1982).

Another area of concentration has been in Evoked Responses (ER's). Krumholz studied Visual, Auditory, and Somatosensory ER's in 17 TS patients, and found no ER differences between TS'ers and controls. Obeso (1982) also studied ER's: by stimulating the median nerve he found no abnormalities in the induced potentials. Of his 127 patients, however, he only tested three. Weate et. al. (1993) claimed that while short latency Evoked Potenials (EP's) were normal in TS'ers, long latency Event Related Potentials (ERP's) show some differences. Finally, Domino (1982) studied 5 medication-free TS'ers and 5 TS'ers on haloperidol and concluded that Visual ER's are normal only if unmedicated; Domino claimed that haloperidol acted to slow visual impulse transmission, which prolonged the ER latencies (Domino, 1982).

In the nineties, EEG abnormalities in TS are considered rare (Weate, 1993), and neither EEG readings nor ER findings are considered helpful in the diagnosis or therapy of TS (Krumholz, 1983). Of latest interest are abnormalities in Contingent Negative Variations (CNV's) in TS. CNV's are slow, negative, brain potentials which occur after a particular stimulus in anticipation of a second stimulus associated with the first. CNV's are thought to reflect the levels of the neurotransmitter dopamine in the central areas of the brain. The study of CNV's in TS was prompted by the fact that dopamine is implicated in TS.

Weate et. al. (1993) tested 12 patients (10 male) between the ages of 10 and 21. All were unmedicated. Fz, Pz, and Cz sites were used. They found that the CNV amplitude was significantly higher ($p<.01$), and that Post-Imperative Negative Variation (PINV) was significantly more often present among TS patients than among controls (Weate et. al., 1993). These findings are similar to those associated with individuals suffering from depression, schizophrenia, and Parkinson's patients undergoing dopamine replacement therapy (Weate et. al., 1993). Frontal PINV has also been associated with obsessive thoughts, and distractibility problems – problems previously cited to be prevalent in TS (Tecce & Cattanach).

As a TS'er himself, the author of this paper has often described TS as being "stuck in a rut" – whether it be a motoric rut (i.e. being unable to stop a tic), a cognitive rut (i.e. being unable to stop a thought), or an emotional rut (i.e. being unable to pull oneself from a particular mood), the sensations are similar: an irrational, pressing need or "itch" to repeat these movements, sounds, thoughts, or feelings. If CNV can be thought of as a type of "classical conditioning" at the neuronal level, then this exaggerated CNV in TS'ers is indicative of a kind of "hyper-association" of different stimuli. Assuming then that the problem in TS is that associative links between stimuli are too strongly made, one would expect that associations among TS'ers would be formed easier and with less trials, and perhaps associations which are too weak to be made in a "normal" population *would* be forged in a Tourettic population. Based on these premises, it is easy to see why and how tics, obsessive thoughts, and other rituals become generalized and widespread. Consider an illustration; if a "normal" person was to ever, by chance, blink his eyes while walking under an archway, this incidental pairing would be too random or inconsequential (or too rare an occurrence) for strong associations to be tied between walking under an archway and blinking. If, however, TS'ers are hypersensitive to any associations, no matter how unimportant, the next time a TS'er walks beneath an archway (s)he will trigger a powerful negative preparatory variation in anticipation of blinking. Psychologically, this would be experienced as an irrational urge or "itch" to blink. By

this logic, one would expect the urge to be "satiated" once one blinked, and one would also expect that association to be strengthened even more, so that the next time that individual walks under an archway, the "itch" would be even more impossible to ignore.

There is certainly considerable evidence which shows that tics can be "traced" to their origins (Sacks, 94). For example, the author of this paper has had for years a "head tic", which involves a whipping of his head to the left. This tic began years ago when the author grew the bangs of his hair so long that he needed to occasionally "toss" his hair out of his eyes. The fact that the tic involves throwing one's head to the left is explained by the fact that the author used to part his hair on the right side, hence the bangs hung over his left eye. Finally, there exists evidence which claims that CNV is decreased by distraction methods (Tecce & Cattanach). Distraction is also a key strategy for decreasing tics, and for getting a TS'er past obsessive thoughts.

In conclusion, the history of EEG recordings on TS'ers is quite voluminous. Much of the early work was not focused, and a confusing variety of findings were cited. Later researchers began controlling for such variables as age, sex, medications, and MBD, which resulted in a much smaller percentage of abnormal EEG readings in the TS population. Particular areas of interest have been EEG activity surrounding tics, Evoked Responses, and Contingent Negative Variations.

BIBLIOGRAPHY

Bornstein, R.A., King, G., & A. Carroll (1983). *Neuropsychological abnormalities in Gilles de la Tourette's syndrome.* In The Journal of Nervous and Mental Disease, (171), 8, 497-502.

Cohen, D.J., Bruun, R.D., and J.F. Leckman (eds.). Tourette's Syndrome and Tic Disorders: Clinical Understanding and Treatment. Wiley Series in Child and Adolescent Mental Health (John Wiley & Sons: Toronto). 1988.

Comings, David E.(1990). Tourette Syndrome and Human Behaviour. California: Hope Press.

Corbin, K.B., Field, J.R., Goldsleen, N.P., et. al. (1968). *Further observations on Tourette's syndrome.* In Proc. Aust.Association of Neurology, (5), 447-453.

Dolmierski, R., and Klossowna, M. (1958 and 1962). *On the disease of Gilles del la Tourette*. In Neurological Neurochir. Psychiatry pol., 8, 639.

Domino, E.F., Piggott, L., Demetriou, S., et. al. (1982). *Visually evoked responses in Tourette syndrome.* In Gilles de la Tourette Syndrome (eds. A.J. Friedhoff & T.N. Chase), Advances in Neurology, (35). New York: Raven Press. 115-120.

Feild, J.R., Corbin, K.B., Goldstein, N.P., et. al. (1966). *Gilles de la Tourette syndrome*. In Neurology, 16, 453-462.

Dr. B. Duncan McKinlay
Psychologist

London, ON, Canada
url. www.lifesatwitch.com

Website:
Tourette Syndrome • OCD • ADHD
Sensory • ODD • IED/'Rage'

Publishing:
Nix Your Tics! Eliminate Unwanted Tic Symptoms: A How-To Guide for Young People

Fernando, S.J.M. (1967). *Gilles de la Tourette's syndrome: a report on four cases and a review of published case reports.* In British Journal of Psychiatry, (113), 607-617.

Kelman, D.H. (1965). *Gilles de la Tourette's diseasein children. A review of the literature.* In Journal of Child Psychology and Psychiatry, 6, 219-226.

Krumholz, A., Singer, H.S., Niedermeyer, E., et. al. (1983). *Electrophysiological studies in Tourette's Syndrome.* In Annals of Neurology, (14), 638-641.

Logue, P.E., Platzek, D., Hutzell, R., et. al. (1973). *Neurological, neuropsychological and behavioural aspects of Gilles de la Tourette's syndrome: a case.* In Perceptual and Motor Skills, 37, 855-861.

Lucas, A.R., & E.A. Rodin (1973). *Electroencephalogram in Gilles de la Tourette's disease.* In Fifth World Congress of Psychiatry, Mexico City, 85-89.

Obeso, J.A., Rothwell, J.C., & C.D. Marsen (1982). *The neurophysiology of Tourette's Syndrome.* In Gilles de la Tourette Sundrome (eds. A.J. Friedhoff & T.N. Chase), Advances in Neurology, (35). New York: Raven Press. 105-114.

Robertson, Mary M. (1989). *The Gilles de la Tourette Syndrome: the current status.* In British Journal of Psychiatry, (154), 147-169.

Robertson, M. M. (1994). *Annotation: Gilles de la Tourette syndrome – an update.* In Journal of Child Psychology and Psychiatry. Vol (35), 4, 597-611.

Robertson, M., Trimble, M.R., & A.J. Lees. (1988). *The psychopathology of the Gilles de la Tourette syndrome: a phenomenological analysis.* In British Journal of Psychiatry, (152), 383-390.

Sacks, O.W. (1982). *Acquired Tourettism in adult life.* In Gilles de la Tourette Sundrome (eds. A.J. Friedhoff & T.N. Chase), Advances in Neurology, (35). New York: Raven Press. 89-92.

Sacks, O.W. An Anthropologist on Mars. 1994.

Shapiro, A.K, Shapiro, E.Sl, Bruun, R.D., et al. Gilles de la Tourette Syndrome. Raven Press: New York. 1978.

Tecce, J.J., and L. Cattanach. *Contingent Negative Variation (CNV)* in Electroencephalography: Basic Principles, Clinical Applications, and Related Fields (Chapter 50). 887-910.

Verma, N.P., Syrigou-Papavasiliou, A., & P.A. Lewitt (1986). *Electroencephalographic findings in unmedicated, neurologically and intellectually intact Tourette syndrome patients.* In Electroencephalography and Clinical Neurophysiology, (64), 12-20.

Weate, S., Newell, S., Bogner, J., et. al. (1993). *Contingent negative variation in Gilles de la Tourette syndrome.* In Clinical Electroencephalography, (24), 4. 188-191.