Premonitory Urges as "Attentional Tics" in Tourette's Syndrome

By: Michael J. Kane, M.A.

Kane, M.J. (1994). Premonitory urges as “attentional tics” in Tourette’s Syndrome. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*, 805-808.

Abstract:
The author, a graduate student with Gilles de la Tourette's syndrome, proposes that pre-tic sensory experiences result from a specific attentional deficit. Based on his own introspective case study, the author argues that the premonitory urges that precede tics are not unique sensory events, but rather are manifestations of somatosensory hyperawareness which serves as the aversive stimulus toward which tics are purposively directed. An "attentional tic" theoretical framework for the study of Tourette's syndrome is suggested and discussed in reference to inhibitory theories of attention.
Key Words: Tourette's syndrome, sensory tics, attention, inhibition.

Article:

Joseph Bliss (1980), a patient with a 62-year history of Gilles de la Tourette's syndrome (TS), first documented the intentional nature of tics by articulating the subjective experience of having tics and the perceptual events that surround tics. Bliss asserted that tics are preceded by aversive sensations which develop in TS patients through a process in which sensory events such as tactile perceptions take on a special exigence. Tics are thus produced to relieve these sensations, but they only allow a fleeting reprieve. According to Bliss, then, TS is not a movement disorder; rather, it centrally involves a hypersensitivity to somatosensory stimulation.

Bliss' (1980) views were initially confirmed by case studies (Bullen and Hemsley, 1983; Kurlan et al., 1989) and were then elaborated by surveys in which more than 80% of TS patients reported that tics are produced as intentional responses to aversive sensory phenomena (A. Cohen and Leckman, 1992; Lang, 1991; Leckman et al., 1993).

Now that "sensory tics" (Shapiro et al., 1988) have been widely reported (see also George et al., 1993), it is necessary to refine our understanding of the tic experience. To this end, I present below an introspective case study that provides a unique phenomenological account of TS. In doing so I formulate a working explanation of TS tics' intentionality and the quintessence of the pre-tic condition; furthermore, I suggest that the etiopathogenesis of TS is less "sensory" than "attentional" and that pre-tic sensations are manifestations of somatosensory *hyperattention.* That is, due to deficient attentional inhibition, the TS patient suffers from an oppressive hyperawareness of what his or her skin, muscles, and joints feel like.

CASE STUDY

I experienced TS onset 19 years ago, at age 7. My first symptom was head jerking, and subsequent (primarily simple tic) symptoms included facial tics, extremity tics, vocal *tics* (e.g., squeaking, grunting), and touching tics. From ages 11 to 13 I was prescribed haloperidol, but eventually, because of side effects, I was prescribed clonidine. One year later, clonidine was deemed ineffective and all medication was discontinued.

Pre-Tic "Sensations" and the Purpose of Tics

Although some clinicians may believe tics to be as subjectively purposeless as a muscle twitch, there is really no phenomenological similarity between an involuntary muscle spasm and a TS tic; muscle spasms are introspectively uncontrollable, whereas tics are intentional and predictable. From the quickest eye blink to the most deliberate shoulder shrug, tics are preceded by a "sensory" experience which has proved difficult to

describe (for example: "unfulfilled sensation" [Bliss, 1980]; "itch" [Bullen and Hemsley, 1983]; "creaky feeling" [A. Cohen and Leckman, 1992]; "feeling of pressure" [Leckman et al., 1993]). Importantly, these sensations are not mere precursors to tics; they precipitate tics. That is, more than providing a signal of imminence, the pre-tic sensation acts as the aversive stimulus toward which tics are directed.

An itch is an appropriate analogy for the pre-tic sensation inasmuch as it is an adverse and urgent stimulus for action (Bliss, 1980). The analogy fails, however, because unlike the pre-tic state, an itch is most commonly a narrowly localized sensation which may be terminated by scratching. The term *"pre-tic* sensation" is a misnomer because the TS feeling is acute and omnipresent; sensations do nor suddenly appear before the onset of tics. To have TS is to be pathologically "itchy," and tics act as the most effective (yet ephemeral) method of "scratching."

Perhaps the best description for the sensory state of TS is a somatic hyperattention: It is not as itch-like as it is an enduring somatosensory bombardment. I experience the TS state as one of keen bodily awareness, or a continual consciousness of muscle, joint, and skin sensations. For example, when sitting in a chair, I do not lose awareness of the tactile sensation of the seat against my body, nor can I ignore the deeper somatic sensations of what my back and legs feel like.

The TS state is omnipresent with few exceptions (e.g., during intense concentration or attentional focus, such as in lecturing), but it is not constant in bodily location or intensity. Its primary location may span the torso or may be isolated to a single eyelid, often depending on the presence or absence of salient external stimulation. For example, I have experienced increases in throat tics after wearing a necktie, increases in facial tics with sunburned skin, increases in toe movement after wearing tight shoes, and increases in head-jerking after swimming.

TS state intensity also varies under certain conditions, that is, my level of somatic awareness at any particular body site will continuously change. For instance, if a tic is temporarily stopped, its respective bodily location becomes less ignorable. If all tics are suppressed, virtually all of my joints and muscles begin to demand my attention. The TS state heightens to a stiffening feeling, such that my skin feels like a hardened casing and my joints feel as though they are becoming rigid. The intensity rises until it becomes so unpleasant and distracting that tics must be executed (with a compulsion that rivals the scratching of a severe itch).

The reason that tics are only marginally effective in providing relief, however, is that unlike scratching an itch, tics do not make the hyperattention go away. Tics merely reset the TS state temporarily back to a baseline. Thus, tics are not themselves pleasant, but they do provide a temporary respite from the persistent hyperawareness.

DISCUSSION

Although most TS patients report sensory tic symptoms, some (particularly young children) do not. Future work, then, should aim to determine the factors responsible for these individual differences. While in my own case, my realization of the pre-tic state may have resulted from a developmental shift in my ability to distinguish compulsive behavior from involuntary behavior, it also likely emerged from a change in symptoms.

My childhood tics (such as eye blinking) were produced in rapid succession, making it difficult to trace their impetus to overt sensations. In late adolescence, however, I also developed more deliberately executed dystonic tics (such as neck craning). These tics were painstakingly slow and bizarre (i.e., lacking in any ethological significance), which allowed and encouraged the intense introspective analysis that helped me to recognize their intentionality. For any given patient, then, I suggest that the awareness and/or reporting of premonitory urges will, most importantly, depend on his or her symptom selection, in addition to an understanding of subtle concepts such as voluntary, impulsive, and compulsive actions.

Although a conscious awareness of the TS stare demands sophisticated meta-attention, it is not necessary to elicit tics. I propose that TS patients are not subject to exotic sensations per se (as the notion of sensory tics suggests); rather, they suffer from a chronic inability to suppress the preconscious processing of (or covert orienting to; see Posner et al., 1980) *normal* somatosensory stimulation. That is, somatic hyperattention is locally exacerbated by external stimuli (e.g., by wearing a necktie) because loci of high somatosensory stimulation enhance the TS state by recruiting attention to affected body sites. Once these sites become selectively attended to, they may serve as the target for tics for hours or even days.

While the above may suggest a primary sensory abnormality in TS, the establishment of an intense, local sensory hyperawareness does not always depend on external stimulation or motor activity. In fact, the TS state may be present in the absence of sensory input. Thus, as is consistent with the attentional tic hypothesis, merely directing attention or thought to a body site may locally exacerbate the TS state. Even thinking about experiencing tics in the abstract is enough to provoke somatic attention.

Finally, even with the execution of simple tics, sometimes the hyperattention does not diminish. For example, I recently developed a complex tic of touching my stomach after meals, which coincides with a preexisting stomach muscle tensing tic. Seemingly, if tics were executed solely to relieve local body sensations, one tic should be sufficient. Of late, however, I have not been exercising and I am thus concerned about my weight. After meals, repeated stomach touching has come to serve as a check for stomach protrusion. The muscle tensing tic continues; the attention recruited by my abdomen, however, is no longer limited to its feel, for it now incorporates worry. Thus, somatosensory hyperawareness has become entangled with a more cognitive and emotional hyperattention. The inability to inhibit somatic attention has merged with an inability to inhibit thought.

Note here the similarity to obsessive-compulsive disorder (OCD), a condition with a well-established comorbidity with TS (e.g., Cummings and Frankel, 1985). While it is beyond the scope of this paper to speak directly to OCD issues, questions concerning the attentional continuum between TS and OCD, and likewise, the attentional association between sensation and thought, may be further explored through an attentional framework for studying premonitory urges and thoughts in TS and OCD (John March, personal communication).

Such a framework may already be available in cognitive psychology. Although cognitive psychologists primarily study visual attention, hypotheses from this domain may apply to the somatic attention dysfunction which I propose occurs in TS. Such behavioral work has focused on the ways in which goal-relevant environmental information becomes highlighted against goal-irrelevant distracter stimulation, and there is a growing literature which suggests that objects that are selected *against* are actively suppressed (Hasher and Zacks, 1988; Tipper and Cranston, 1985). Thus, recent theories argue that selective attention may depend on both excitatory and inhibitory processes, through which relevant stimuli are targeted for processing while irrelevant stimuli are actively denied access to response systems (e.g., Navon, 1989).

Empirical evidence for such attentional inhibition has come from the "negative priming" paradigm (Tipper and Cranston, 1985), in which subjects selectively respond to a class of targets (e.g., green letters) while ignoring a class of distractors (e.g., red letters). Negative priming is said to occur when the response to a target is slowed as a result of that stimulus having recently appeared as a distractor. This slowing is believed to arise from the time taken to overcome the suppression applied to the current target when it had previously been a distracter.

As measured by negative priming, deficient inhibitory processing of distractors has been demonstrated in subject populations thought to exhibit attentional dysfunction, such as older adults (e.g., Hasher et al., 1991; Kane et al., 1994), obsessive-compulsive adults (Enright and Beech, 1993), and schizophrenics (Beech et al., 1989), whose thought disorders may result from their inability to inhibit irrelevant thoughts and sensory stimuli (e.g., Frith, 1979). Analogously, experimental support already exists for attentional dysfunction in TS (e.g.,

Bruun, 1984), and future visual and/or tactile negative priming studies with TS patients may empirically support the hypothesis that somatic hyperawareness is a manifestation of deficient inhibition.

Although a review of the attention literature is not intended here, specifying the mechanisms that support my phenomenological description of the TS state will require a broader perspective on "attention" than that already discussed. Experimental work under an inhibitory framework may provide a starting point; however, other behavioral indices of attention, such as "engagement" and "disengagement" (e.g., Posner et al., 1980), might prove important to examine with TS patients, particularly in determining whether inhibitory failure occurs during covert orienting or at a later, more conscious level of processing.

Furthermore, a cognitive framework for TS study may be complemented by (and inform) our current understanding of the neural circuitry common to both tics and attentional performance. In this regard, a promising model was recently proposed to account for obsessive-compulsive behavior (Wise and Rapoport, 1989). The model provides that motivational input from orbitofrontal and anterior cingulate cortex may disinhibit thalamocortical circuits in the absence of sensory input, and suggests that in OCD there is a failure to block the disinhibitory influence of motivation on behavior. As a result, persons with OCD commit abnormal actions which rely on internal, rather than external, stimulation (e.g., repetitive washing of clean hands). For TS, a similar model may prove applicable to tics' attentional stimulus. For example, the neural circuits between sensory associational cortex, the basal ganglia, and the frontal lobes warrant consideration, for they have been implicated in TS (e.g., Chappell et al., 1990) and also figure prominently in neuropsychological models of attentional selection Q. Cohen and Servan-Schreiber, 1992; Pennington, in press; Weinberger, 1993) and inhibition (Dempster, 1992) which are consistent with behavioral data (Hasher and Zacks, 1988; Zacks and Hasher, in press).

Summary

The TS state of chronic somatic hyperattention may lie on a pathological end of a somatic-sensitivity continuum which is delineated by inhibitory attentional control, and thus the basic dysfunction in TS symptomatology may be explained as failed attentional inhibition. Furthermore, it is the ultimate link of the physical and cognitive experience of TS which suggests that the notion of attentional tics most effectively captures the essence and the cause of tics. The current conception of sensory tics does not suggest as parsimonious a framework as attentional tics for examining relatively complex tic phenomena or unravelling the intimate connections which are becoming dearer between TS and OCD.

REFERENCES

Beech A, Powell T, McWilliam J, Claridge G (1989), Evidence of reduced "cognitive inhibition" in schizophrenia. *Br J Clin Psycho!* 28:110-116 Bliss J (1980), Sensory experiences of Gilles de la Tourette syndrome. *Arch Gen Psychiatry* 37:1343-1347

Bruun R (1984), Gilles de la Tourette's syndrome: an overview of clinical experience. *J Am Acad Child Psychiatry* 23:126-133

Bullen JG, Hemsley DR (1983), Sensory experience as a trigger in Gilles de la Tourette's syndrome. *J Behav Ther .Exp Psychiatry* 14:197-201

Chappell PB, Leckman JF, Paula D, Cohen DJ (1990), Biochemical and genetic studies of Tourette's syndrome: implications for treatment and future research. In: *Application of Basic Neuroscience to Child Psychiatry,* Deutsch SI, Weizman A, Weizman R, eds. New York: Plenum Medical Book Co, pp 241-260

Cohen AJ, Leckman JF (1992), Sensory phenomena associated with Gilles de la Tourette's syndrome. *J Clin Psychiatry* 53:319-323

Cohen JD, Servan-Schreiber D (1992), Context, cortex, and dopamine: a connectionist approach to behavior and biology in schizophrenia. *Psycho! Rev* 99:45-77

Cummings JL, Frankel M (1985), Gilles de Ia Tourette syndrome and neurological basis of obsessions and compulsions. *Biol Psychiatry* 20:1117-1126

Dempster FN (1992), The rise and fall of the 'inhibitory mechanism: toward a unified theory of cognitive development and aging. *Developmental Review* 12:45-75

Enright Si, Beech AR (1993), Reduced cognitive inhibition in obsessive-compulsive disorder. *Br J Clin Psycho!* 32:67-74
Frith CD (1979), Consciousness, information processing and schizophrenia. *Br J Psychiatry* 134:225-235
George MS, Trimble MR, Ring HA, Sallee FR, Robertson MM (1993), Obsessions in obsessive-compulsive disorder with and without Gilles de la Tourette's syndrome. Am *J Psychiatry* 150:93-97
Hasher L, Stoltzfus ER, Zacks RT, Rypma B (1991), Age and inhibition. *J Exp Psycho! Learn Mem Cogn* 17:163-169
Hasher L, Zacks RT (1988), Working memory, comprehension, and aging: a review and a new view. In: *The Psychology of Learning and Motivation,* vol 22, Bower GG, ed. San Diego, CA: Academic Press, pp 193-225
Kane MJ, Hasher L, Stoltzfus ER, Zacks RT, Connelly SL (1994), Inhibitory attentional mechanisms and aging. *Psycho! Aging* 9:103-112
Kurlan R, Lichter D, Hewitt D (1989), Sensory tics in Tourette's syndrome. *Neurology* 39:731-734
Lang A (1991), Patient perception of tics and ocher movement disorders. *Neurology* 41:223-228
Leckman JF, Walker DA, Cohen DJ (1993), Premonitory urges in Tourette's syndrome. *Am J Psychiatry* 150:98-102
Navon D (1989), The importance of being visible: on the role of attention in a mind viewed as an anarchic intelligence system. I. Basic tenets. *European Journal of Cognitive Psychology 1:191-213*
Pennington BF (in press), The working memory function of the prefrontal cortices: implications for developmental and individual differences in cognition. In: *Future Oriented Processes in Development.* Haith M et al., eds. Chicago: University of Chicago Press
Posner MI, Snyder CRR, Davidson BJ (1980), Attention and the detection of signals. *J* Exp *Psycho! Gen* 109:160-174
Shapiro AK, Shapiro ES, Young JG, Feinberg TE (1988), Gilles de Is Tourette's syndrome. New York: Raven Press
Tipper SP, Cranston M (1985), Selective attention and priming: inhibitory and facilitory effects of ignored primes. *QJ Exp Psycho! [A]37D:591-611*
Weinberger DR (1993), A connectionist approach to the prefrontal cortex. *I Neuropsychiatry* 5:241-253
Wise SP, Rapoport JL (1989), Obsessive-compulsive disorder: a basal ganglia disease? In: *Obsessive-Compulsive Disorder in Children and Adolescents,* Rapoport JL, ed. Washington, DC: American Psychiatric Press, pp 327-346
Zacks RT, Hasher L (in press), Directed ignoring: inhibitory regulation of working memory. In: *Inhibitory Mechanisms in Attention, Memory, and Language,* Dagenbach D, Carr TH, eds. New York: Academic Press