Adult onset tic disorders

Sylvain Chouinard, Blair Ford

Abstract

***Background*—Tic disorders presenting during adulthood have infrequently been described in the medical literature. Most reports depict adult onset secondary tic disorders caused by trauma, encephalitis, and other acquired conditions. Only rare reports describe idiopathic adult onset tic disorders, and most of these cases represent recurrent childhood tic disorders.**

***Objective*—To describe a large series of patients with tic disorders presenting during adulthood, to compare clinical characteristics between groups of patients, and to call attention to this potentially disabling and underrecognised neurological disorder.**

***Methods*—Using a computerised database, all patients with tic disorders who presented between 1988 and 1998 to the movement disorders clinic at Columbia-Presbyterian Medical Center after the age of 21 were identified. Patients' charts were retrospectively reviewed for demographic information, age of onset of tics, tic phenomenology, distribution, the presence of premonitory sensory symptoms and tic suppressibility, family history, and associated psychiatric features. These patients' videotapes were reviewed for diagnostic confirmation and information was obtained about disability, course, and response to treatment in a structured follow up interview.**

***Results*—Of 411 patients with tic disorders in the database, 22 patients presented for the first time with tic disorders after the age of 21. In nine patients, detailed questioning disclosed a history of previous childhood transient tic disorder, but in 13 patients, the adult onset tic disorder was new. Among the new onset cases, six patients developed tics in relation to an external trigger, and could be considered to have secondary tic disorders. The remaining patients had idiopathic tic disorders. Comparing adult patients with recurrent childhood tics and those with new onset adult tics, the appearance of the tic disorder, the course and prognosis, the family history of tic disorder, and the prevalence of obsessive-compulsive disorder were found to be similar. Adults with new onset tics were more likely to have a symptomatic or secondary tic disorder, which in this series was caused by infection, trauma, cocaine use, and neuroleptic exposure.**

***Conclusions*—Adult onset tic disorders represent an underrecognised condition that is more common than generally appreciated or reported. The clinical characteristics of adults newly presenting to a movement disorder clinic with tic disorders are reviewed, analysed, and discussed in detail. Clinical evidence supports the concept that tic disorders in adults are part of a range that includes childhood onset tic disorders and Tourette's syndrome.**

(*J Neurol Neurosurg Psychiatry* 2000;**68**:738–743)

Keywords: Tourette's syndrome; secondary tic disorders; obsessive-compulsive disorder; attention-deficit hyperactivity disorder

Department of Neurology, University of Montreal, Quebec, Canada
S Chouinard

Department of Neurology, Columbia University New York, New York, USA
B Ford

Correspondence to:
Dr Blair Ford, Neurological Institute, 710 168th Street, New York, NY 10032, USA
Ford@movdis.cis.columbia.edu

Received 12 August 1999
and in revised form
6 December 1999
Accepted 14 December 1999

Tic disorders are commonly considered to be childhood syndromes. Childhood onset is a defining feature of Tourette's syndrome, a chronic disorder of severe motor and vocal tics that begins before the age of 18[1] or 21,[2] depending on the criteria. The childhood onset of tic disorders has been consistently described since Gilles de la Tourette's seminal report, in which all eight patients presented with tics during childhood.[3] The DSM-IV classification of tic disorders does not include a category for tic disorders that develop during adulthood, other than tic disorder "not otherwise specified". Tic disorders experienced during adulthood are largely considered to be persistent tics from childhood.[4–6] In adults presenting with a tic disorder, it is often assumed that the patients cannot remember having experienced childhood tics.[7] As such, tic disorders newly presenting during adulthood have only occasionally been described in isolated case reports, and mostly in the context of symptomatic, or secondary, tic disorders. The goal of the present study was to describe a large series of patients with tic disorders presenting during adulthood, to compare clinical characteristics between groups of patients, and to call attention to this potentially disabling and underrecognised neurological disorder.

Methods

From a computerised database of patients, we extracted the records of all patients who presented between 1988 and 1998 to the Center for Movement Disorders, Columbia Presbyterian Medical Center, for evaluation or treatment of a tic disorder that had developed after the age of 21. All patients were initially diagnosed by a movement disorder specialist, based on the clinical appearance of tics as sudden, brief, purposeless, stereotyped movements or sounds. We reviewed videotaped examinations of the patients for diagnostic confirmation. We considered a tic disorder to be present when the patient stated that the tics were socially, occupationally, or functionally disabling in any way. Only those patients in whom a tic disorder was the sole complaint and primary diagnosis were considered in this

study, but we included patients with symptomatic or secondary tics if their movement disorder consisted exclusively of tics. Patients with tics caused by somatoform illness and related conditions were not included. All patients who presented with tic disorders developing after the age of 21 were considered to have adult onset tics. We attempted to establish whether patients with adult onset tics had experienced tics during childhood. Patients with tic disorders that began during childhood and persisted through adulthood were not included. We did not exclude patients with adult onset tics if their tic disorder began after the age of 21 but who had experienced tics lasting less than 12 months during childhood, and who did not present to a physician at that time.

We retrospectively reviewed patients' charts for demographic information, age of onset of tics, description of tics, distribution, the presence of premonitory sensory symptoms, the degree of tic suppressibility, family history, associated psychiatric features, disability, course, and response to treatment. Where possible, we obtained psychiatric records. The diagnosis of obsessive-compulsive disorder was based on DSM-IV criteria,[1] requiring recurrent obsessions or compulsions severe enough to be time consuming or cause significant distress or impairment. To obtain more recent follow up information, a structured questionnaire was administered to 18 patients by phone or direct interview.

Results

Of 411 patients with tic disorders in our database, 22 (5.4%) presented for evaluation of tics after the age of 21. For 20 patients, videotaped examinations were available for confirmation of diagnosis. There were 17 men and five women. The mean age of onset was 40 years, ranging from 24 to 63 years. Nine patients had a history of tics during childhood, but in 13, there was no prior history of tics, as far as could be determined from interview with the patient and family members.

In 16 patients, the aetiology of the tic disorder was considered idiopathic, but in six, all with new onset tic disorder, there seemed to be a causal relation between the tic disorder and an environmental factor. In one patient with new adult onset tics, the disorder developed during an extended cocaine binge. In two patients, a new tic disorder developed after a head injury sustained during a car accident. A fourth patient described the onset of neck tics after straining her neck during weight lifting. A fifth had tardive tics due to neuroleptic exposure. A sixth patient developed a prominent throat clearing tic after a severe pharyngitis. Symptomatic or secondary tic disorders always presented as a new onset tic disorder in adults, and never as recurrent childhood tics, a relation that was statistically significant ($p<0.05$, Fisher's exact test).

The clinical characteristics of our patients with new adult onset tics are listed in table 1. Among 13 patients with new onset adult tics, three patients had isolated motor tics, five patients had multiple motor tics, three patients had multiple motor and vocal tics, one patient had an isolated vocal tic, and one patient experienced multiple vocal tics. Tic suppressibility was noted in nine patients and eight patients had a premonitory sensory symptom before the tics. A family history of tic disorder was present in five patients and five had symptoms of obsessive-compulsive disorder. The duration of illness in patients with new adult onset tics was on average 10.5 years at the time of most recent follow up, ranging from 1.5 to 45 years. Nine patients elected to have treatment, and in four, there was modest improvement. Over the course of the illness, tics tended not to vary in repertoire, and isolated tics remained unchanged. Tic severity tended to wax and wane, and no patient experienced a prolonged symptom remission.

The clinical characteristics of patients with a recurrent childhood tic disorder during adulthood are listed in table 2. For the nine patients with recurrent childhood tics, the mean age of recurrence was 47 years, ranging from 25 to 63 years. All patients had experienced transient childhood tics of mild degree, and the mean symptom free hiatus in these patients was about 32 years, ranging from 12 to 56 years. Five patients exhibited multiple motor tics, three patients had multiple motor and phonic tics, and one had an isolated motor tic. No patients had verbal tics. All patients described a premonitory sensory symptom and were able to suppress the tics. The childhood tics consisted of facial tics or blinking in seven patients and in two patients, the appearance of the childhood tics was not specified. No patient had undergone evaluation or treatment during childhood. Four patients had symptoms of obsessive-compulsive disorder. A positive family history for tic disorder was present in four patients and two had a family history of obsessive-compulsive disorder. The mean duration of illness from the recurrence of tics to the most recent follow up was 13 years, ranging from 3 to 20 years. Seven patients underwent treatment for their tics, of whom three noted some improvement. No patient had a full or sustained remission during adulthood.

CASE REPORTS

New onset tics during adulthood

Case 2—This patient developed a tendency to grunt in his 30s. With time, the grunting became more frequent and intrusive, and was joined by several other stereotyped sounds and vocalisations, including frequent short verbal utterances: "yo" "hey, hey", "me too". The vocalisations began to interfere with the patient's social life, preventing him from going to the cinema or social gatherings. He described a premonitory sensation but was powerless to control the vocalisations. Treatment with haloperidol, benztropine, clonidine, and clonazepam did not help, and he experienced akathisia on haloperidol. The tics were improved on verapamil. There was no history of previous tics or psychopathology, but the family history was positive for tic disorders. On examination, the patient exhibited his stere-

Table 1 Clinical characteristics of patients with new adult onset tics

Patient	Sex	Age of onset	Description of tics	Distribution of tics	Family history of tic disorder?	Associated psychiatric symptoms	Medication trials	Course	Follow up (y)	Additional features
1	M	24	Multiple motor tics	Neck and arms	Y	—	No benefit from TRHX, CLNZ, TBZ, PMZ	Stable	2	
2	M	30	Primary vocalization tics with multiple motor tics	Face, arms	Y	—	Improvement on VPM	Waxing and waning, improved by follow up	45	
3	F	32	Single motor tic	Neck	N	—	No benefit	Stable	18	Tic developed after straining neck while weight lifting
4	M	33	Multiple vocal and motor tics	Larynx, neck, arms	N	—	No benefit from CBZ, CLNZ	Stable	2	Tics developed after MVA with head trauma
5	M	40	Multiple motor tics	Face	N	—	No benefit from DZP, CLNZ, TBZ, PMZ	Waxing and waning	4	
6	M	41	Multiple phonic and motor tics	Face, neck, arms	N	—	Botulinum toxin	Improved	20	Tics developed after MVA
7	M	42	Primary neck tic with multiple facial and arm tics	Face, neck	Y	OCD	Untreated	Improved	6	
8	M	49	Multiple motor tics	Face, neck, arms	N	OCD	No benefit from CLNZ, HAL, PMZ	Waxing and waning	1.5	
9	F	69	Single vocalisation	Vocal	N	OCD	Untreated	Stable	5	Tics developed during pharyngitis
10	M	46	Multiple complex motor tics	Face, neck, arms	Y	Substance misuse disorder-	Unresponsive to levodopa, helped with CLNZ	Waxing and waning	2	Tics developed during period of heavy cocaine use
11	M	40	Single complex motor tic: blowing movements	Face	N	Schizo affective	Untreated/lost to follow up	Persistent	5	Tic status developed after 5 year exposure to molindone
12	M	46	Single respiratory tic	Face, larynx	N	OCD	Improvement on DZP	Waxing and waning	3	
13	M	25	Multiple verbal tics, including coprolalia	Vocal	Y	OCD	Untreated	Waxing and waning	15	

CBZ=Carbamazepine; CLNZ=clonazepam; DZP=diazepam; HAL=haloperidol; MVA=motor vehicle accident; OCD=obsessive- compulsive disorder; PMZ= pimozide; TBZ=tetrabenazine; TRHX=trihexiphenidyl; VPM=verapamil.

otyped verbalisations, as well as a loud yelling tic, facial grimacing, and arm movements.

Case 5—This 63 year old man was referred for evaluation of abnormal facial movements. Since the age of 40, he had been aware of involuntary blinking, head bobbing, and neck jerking movements. There was no history of abnormal vocalisation. He described that he was able to partially suppress the movements, which were preceded by an inner sensation. Treatment with haloperidol, pimozide, tetrabenazine, and diazepam gave no relief. There was no history of tics or obsessive-compulsive disorder, or family history of tic disorders or obsessive-compulsive disorder. A review of the videotape taken during the first examination showed numerous minor tics consisting of blinking, anterocollic jerks, shoulder jerks, and lower face grimacing. On a

Table 2 Clinical characteristics of patients with recurrent childhood tics

Patient	Sex	Age of onset	Description of tics	Distribution of tics	Childhood tics	Family history	Associated psychiatric symptoms	Medication trials	Course	Duration of follow up (y)
14	M	25	Phonations and multiple motor tics	larynx, face, neck, limbs	Multiple motor tics	Tics	OCD	No benefit from DZP, ALPZ, PMZ	Waxing and waning, improved	20
15	M	31	Multiple motor tics	Face, neck	Unspecified	—	—	Improvement on CLNZ, FLUOX, VPA	Waxing and waning, improved	11
16	M	32	Multiple motor tics	Face, neck	Ear wiggling	OCD	—	No benefit from CLNZ	Waxing and waning, improved	8
17	M	41	Multiple motor tics	Face	Blinking and mannerisms	OCD	OCD and anxiety disorder	Untreated	Waxing and waning	16
18	F	28	Multiple motor tics	Face, neck	Blinking	Tics	OCD	Improvement on CLNZ	Relapsing-remitting	20
19	M	61	Throat clearing phonation and multiple motor tics	Face, larynx, neck	Unspecified	Tics	OCD	No benefit on TRHX, improvement on CLNZ	Stable	13
20	F	62	Jaw opening tic, abdominal tensing tic	Face, abdomen	Blinking	—	—	No benefit from TRHX, BAC	Stable	10
21	M	63	Multiple phonations, facial tics	Neck, face, abdomen	Facial grimacing	Tics	—	No benefit from CLNZ, CLON, HAL	Stable	17
22	F	29	Single tongue thrusting tic	Tongue	Blinking	—	—	Untreated	Stable	12

ALPZ=alprazolam; BAC=baclofen; CLNZ=clonazepam; CLON=clonidine; DZP=diazepam; FLUOX=fluoxetine; HAL= haloperidol; OCD=obsessive compulsive disorder; PMZ=pimozide; TRHX=trihexiphenidyl; VPA=valproate.

recent follow up, the patient described that his tics were still present but had fluctuated over the years.

Case 9—This 72 year old woman had developed a stereotyped socially disabling vocalisation 3 years earlier in the context of pharyngitis. She developed a harsh expectoration sound, like a person forcefully clearing their throat. With time, the vocalisation became more frequent, stereotyped, and more prominent when under stress. She described the problem as a "strainful exudement". The sound was often absent, particularly when she was quiet, relaxed, concentrating on a task, or asleep. On neurological examination, the major abnormality was the vocalisation itself, which sounded like an exaggerated, raspy, explosive throat clearing. The vocalisation did not interrupt speech, chewing, or swallowing. The vocalisation could be suppressed when she spoke, counted, or even recited numbers in her head without speaking. Although never treated for obsessive-compulsive disorder, the patient had a lifelong compulsive tendency to keep notes, which she meticulously catalogued, stored, and revised periodically. She was especially interested in the act of recording and the reiteration of detail. There was no history of neuroleptic exposure or head trauma before the vocalisation, and no history of childhood tics. She underwent trials of phenytoin, haloperidol, clonidine, trihexiphenidyl, clonazepam, pimozide, and baclofen, all in low dose and without benefit.

Case 10—This 46 year old man developed stereotyped facial grimacing during a 2 year period of daily intranasal cocaine use. He had been aware of twisting facial movements that occurred transiently during cocaine intoxication, but the movements always subsided as the drug effect waned. At one point during the period of cocaine intake, he became aware that his movements were persisting even when off the drug. With time, he developed a prominent complex tic consisting of stereotyped head turning and rotational shoulder movements. The movements were only briefly suppressible, up to 10 seconds. There was no history of previous tics, neuroleptic exposure, head trauma, anoxia, or encephalitis. The patient's sister had facial movements, but the precise nature of these could not be determined as she lived out of the country. Clonazepam helped to suppress the tics. Within several months, the facial grimacing and shoulder movements resolved, but he developed a stereotyped flicking movement of the left hand.

Case 12—A 46 year old man with obsessive-compulsive disorder was referred by his psychiatrist because of frequent yawning spells. It soon became apparent that his movements, superficially resembling yawning, were different from his normal yawning. The yawning movements were preceded by a sensation of drowning or suffocation that could only be relieved if the yawning movement was "just right: I need that good breath". The yawning was temporarily suppressible, and did not occur if the patient was distracted. The yawning could be substituted for by a sigh, which also abolished the sensation of asphyxia. Diazepam helped to reduce the movement. There was history of previous tics, and the family history was notable for Parkinson's disease. As a child, the patient had obsessive rituals that did not impair him.

Case 13—A 25 year old writer developed involuntary repetitive vocalisations, including coprolalia. These vocalisations were generally suppressible but occasionally came out during stress or anxiety. He stated that often a vocalisation would occur without warning, triggered by the sudden recollection of an unpleasant event. Alone, he often experienced prolonged bouts of cursing and other vocalisations. He also had a tendency to ruminate excessively, imagining himself involved in elaborate rescue missions for members of his family in danger. There was no history of motor tics or previous childhood tics. A paternal uncle had stereotyped facial winking. The patient declined treatment for his tics or obsessive rumination.

Recurrent childhood tics

Case 20—This 62 year old woman developed sudden brief stereotyped jaw opening, preceded by a sensory prodrome of skin tightness around her mouth. By allowing the jaw opening movement to occur, the tightness sensation temporarily abated. The movement could be suppressed by clenching her teeth tightly together. She experienced about 10 events daily, without much change over time. There was no exposure to neuroleptic drugs. As a 12 year old child, she recalls being sent home from camp one summer because of frequent eye blinking attributed to nervousness that resolved.

Case 21—This 79 year old man was referred for a 16 year history of breathing problems. He complained of shortness of breath attributed to abdominal twitching. On examination, he had a frequent tendency to expel air against a closed glottis, producing a suppressed coughing sound. In addition, he had frequent blowing, snorting, sniffing and panting, all of which could be temporarily suppressed or distracted. He also had a stereotyped winking tendency, with tight eyelid closure, and screwing up the left cheek. The facial grimacing had been present as a child, but had waned. In the family history, a nephew had disabling Tourette's syndrome, with multiple motor tics and vocalisations, including coprolalia.

Case 22—This patient developed a stereotyped tongue movement at the age of 29 during a particularly stressful period in her life. Her movement consisted of a tendency for the tongue to curl back and press itself against the upper teeth. The tongue movements were relieved by a dental appliance but this was so uncomfortable that she eventually stopped wearing it. The movement stopped for about 1 year after onset but then recurred, and remained for the next 10 years. The patient described the tongue movement as transiently suppressible, at the cost of a sense of rising inner tension. With each tongue movement, there was transient relief. On examination, the tongue tip quickly elevated, and her tongue retracted and pulled back to the left side of her mouth in a stereotyped manner. The movement did not

Table 3 Previously reported cases of idiopathic tic disorders occurring during adulthood

Reference	*Age/sex*	*Recurrent childhood tics or new onset adult tics?*	*Clinical description*	*Family history*	*Associated psychiatric symptoms*
11	35 M	New onset	Multiple motor and vocal tics	—	Anxiety/depression
12	46 F	Recurrent	Multiple motor and vocal tics	—	none
13	35 M	New onset	Multiple motor and vocal tics	Tic disorder	ADHD
14	62 F	Recurrent	Multiple motor and vocal tics	—	—
	71 M	Recurrent	Multiple motor and vocal tics	—	—
	65 M	Recurrent	Multiple motor and vocal tics	—	—
	63 F	Recurrent	Multiple motor and vocal tics	—	—
15	53 F	Recurrent	Multiple motor and vocal tics	—	—
16	55 M	New onset	Multiple motor and vocal tics	Tic disorder	Depression
17	81 M	New onset	Multiple vocal and phonic tics	—	—
18	30 M	New onset	Multiple motor and vocal tics	—	—

occur during speaking, and there was no dysarthria or other speech impediment. Sometimes during the interview there was a rapid stereotyped lateral jaw deviation to the left, but no other repetitive or stereotyped movements, such as tongue protrusion, sustained posturing, palatal myoclonus, or pharyngeal movements. When she was 5 years old, the patient had had motor tics consisting of blinking, which resolved spontaneously within several months. There was no history of vocalisations, repetitive rituals, obsessions, or other related symptoms.

Discussion

Tic disorders newly presenting during adulthood have occasionally been described in the neurological literature, mostly in relation to an acquired brain lesion,[8–10] or as incidental tics in the context of another neurological or psychiatric disease, such as Huntington's disease.[8] There are few descriptions of primary, or idiopathic, adult onset tics in the literature, and about 55% of reported cases represent recurrences of childhood tic disorders,[11-14–19] as shown in table 3. The largest report is that of Klawans and Barr, who described four patients with childhood onset tics that remitted before the age of 21, only to recur after the age of 60.[14] Taken together, the paucity of reports of adult onset tic disorders suggests that they represent an unusual entity, especially idiopathic tic disorders, rarely encountered in clinical practice, in marked contrast to the high prevalence of childhood tic disorders.

The present series of 22 patients is the largest report describing the clinical features of tic disorders presenting during adulthood. In our population, adults newly presenting with tic disorders represent 5.4% of patients of all ages evaluated for tics. We cannot estimate the prevalence of adult onset tic disorders in the general population, but the number of patients we have encountered suggests that the phenomenon is more common than previously reported. When adults present for the first time to a physician for evaluation of a tic disorder, a certain number will have true new onset tics, whereas the rest will prove to have a recurrence of a childhood tic disorder. Among adults with new onset tics, about 50% will have an apparent external precipitant of the disorder. Establishing that an adult patient with apparent new onset tics did not have tics during childhood can rarely be done with certainty because some patients are unaware of their tics, and reliable observers who knew the patient as a child may not be available. As such, it is important to divide adult onset tic disorders into two categories based on the time course of presentation: a new tic disorder after the age of 21 years or the recurrence of childhood tics that had previously resolved. In the present series, the categorical breakdown among 22 patients was: idiopathic new onset tics in seven (32%), new onset secondary tic disorder in six (27%), and recurrent childhood tic disorder in nine (41%).

The clinical features of adult onset tic disorders resemble those described in childhood onset tic disorders.[7 20 21] However, the range of tic phenomenology in our population seemed more restricted relative to childhood tics, and the severity of tic disorders was milder. There was little coprolalia, as has been previously noted in childhood tics persisting into adulthood.[6] Automatisms of the type described in autistic children were not found in this sample, but may reflect referral bias, as adults with autistic behaviours are not generally referred to our clinic. In our population, there were no phenomenological differences between patients with de novo adult onset tics and those patients with recurrent childhood tics. Both groups exhibited tics that most often involved the face, neck, and shoulder. Most patients had multiple motor tics. In the series, only six patients had an isolated single tic disorder, but three others had a primary tic that overshadowed a background of multiple, minor tics. Single tics and dominant tics persisted over the full course of the disorder, waxing and waning in severity but not varying in repertoire. In both groups, no patient had a complete remission of their tics in adulthood. Treatment attempts were generally not successful, and rarely sustained. Eventually, most patients elected to discontinue medical treatment, prefering to live with their tics than to experience the side effects of medication. The concept that obsessive-compulsive disorder is part of the range of neurobehavioral manifestations of Tourette's syndrome is well accepted,[22 23] and in our series was present in nine of 22 patients (40.9%).

Disability was not formally quantified in our population, and we could not distinguish disability due to tics from disability caused by attendant psychopathology. In general, patients stated that they were mainly socially disabled by their tics, in agreement with studies describing social embarrassment and isolation as the most disabling consequences of tic disorders.[24] All of our patients were self referred for tics, a referral bias that usually selects for more severely

affected people. However, based on interviews with the patients and review of the patients' videotaped neurological examinations, the patients in this series would be considered to have only mild (score 0 to 24) or moderate (score 25 to 39) severity tic disorder, using a standard rating scale.[25] We did not encounter any patient presenting during adulthood with a tic disorder at the extreme end of the severity range, with tics so disruptive and intractable that social, familial, or occupational functioning is all but precluded. All adults with this degree of disease severity followed at our centre developed their disorders during childhood.

The prevalence of symptomatic tic disorders seems significantly higher among adults with new onset tic disorders than for adults with recurrent childhood tics, a finding inferred from the number of published reports of secondary tic disorders in the literature.[9] Adults with new onset tics are also more likely to have an underlying cause or provocative event for their tics than children with tic disorders. By contrast, childhood onset tic disorders are only rarely linked to a specific aetiology. Almost one third of the patients in this series reported the onset of tics after a clear inciting event, including neck strain, head injury, local infection, cocaine binge usage, and exposure to neuroleptic drugs, all conditions known to precipitate tics.[7] The literature suggests that tics caused by peripheral trauma tend to be single, non-varying isolated tics, and the two patients in our series who come closest to a peripheral physical injury (cases 3 and 9) support this notion. The question arises as to whether those with secondary tics would have gone on to develop tics in the absence of the precipitating event. In our cases of secondary tic disorders, and among adult onset post-traumatic cases described in the literature, about half had a history of attention deficit hyperactivity disorder, obsessive compulsive traits, or a family history of tic disorder, or obsessive-compulsive disorder, all biological substrates often linked to primary, or idiopathic, tic disorders.

The true prevalence of adult onset tic disorders remains unknown. Simple observation suggests that tics in adults may not be uncommon, but the phenomenon has not been studied systematically. One difficulty in obtaining an accurate prevalence figure of adult tic disorders is that many people with tics are unaware of their symptoms. People may also be unaware of tics in family members. Tics wax and wane, and are suppressible, sometimes unconsciously, escaping detection even by an expert in a standardised setting. The frequency and intensity of tic disorders decreases with time, making adult cases of tics even more difficult to ascertain.[26 27] All of these problems may lead to underrecognition of tics, which in turn has likely impeded the development of biological or genetic markers for tic disorders.

Do adult onset tics constitute one end of the range of Tourette's syndrome? In this series of 22 patients, it would seem that the phenomenology of adult tic disorders, the clinical features, prognosis, family history, and associated neurobehavioural elements are entirely typical of childhood onset tic disorders. The clinical evidence suggests that adult tic disorders are part of a range of illness that includes childhood onset tics and Tourette's syndrome. We therefore propose that classifications of tic disorders include an adult age category that is subdivided by disease course into tic disorders that persist from childhood, tic disorders that represent a recurrence of transient childhood tics, and genuine new onset adult tic disorders. Within the category of adult onset tics, it also seems important to differentiate primary from secondary tic disorders, and most of the secondary cases will fall into the new onset adult tic disorder category. We anticipate that the classification of tic disorders, now based entirely on clinical criteria, will become clearer with the development of genetic markers.

We are grateful to Dr Paul Greene for his critical review of the manuscript and help with the computerised database, and to Dr Stanley Fahn for his support.

1 American Psychiatric Association. *Task force on nomenclature and statistics. Diagnostic and statistical manual of mental disorders.* 4th edition (DSM-IV). Washington DC: APA, 1994.
2 The Tourette syndrome classification study group. Definitions and classification of tic disorders. *Arch Neurol* 1993;**50**:1013–16.
3 Gilles de la Tourette G. Etude sur une affection nerveuse caractérisée par de l'incoordination motrice accompagnée d'écolalie et de coprolalie. *Arch Neurol (Paris)* 1885;**9**:19–42,158–200.
4 Shapiro AK, Shapiro E, Wayne HH. Birth, developmental and family histories and demographic information in Tourette's syndrome. *J Nerv Ment Dis* 1972;**155**:335–44.
5 Burd L, Kerbeshian J, Wikenheiser M, *et al*. Prevalence of Gilles de la Tourette syndrome in North Dakota adults. *Am J Psychiatry* 1986;**143:787–8**.
6 Goetz CG, Tanner CM, Stebbins GT, *et al*. Adult tics in Gilles de la Tourette's syndrome: description and risk factors. *Neurol* 1992;**42**:784–8.
7 Jankovic J. Phenomenology and classification of tics. *Neurol Clin* 1997;**15**:267–75.
8 Sacks OW. Acquired tourettism in adult life. *Adv Neurol* 1982;**35**:89–92.
9 Kumar R, Lang AE. Secondary tic disorders. *Neurologic Clin* 1997;**15**:309–31.
10 Factor SA, Molho ES. Adult-onset tics associated with peripheral injury. *Mov Disord* 1997;**12**:1052–5.
11 Araneta E, Magen J, Musci MN, *et al*. Gilles de la Tourette's syndrome symptom onset at age 35. *Child Psychiatry Hum Dev* 1975;**5**:224–30.
12 Ziegler DK. Tourette's syndrome and essential tremor in septuagenarian [letter]. *Arch Neurol* 1982;**39**:132.
13 Marneros A. Adult onset of Tourette's syndrome: a case report. *Am J Psychiatry* 1983;**140**:924–5.
14 Klawans HL, Barr A. Recurrence of childhood multiple tic in late adult life. *Arch Neurol* 1985;**42**:1079–80.
15 Sandyk R, Awerbuch G. Recurrence of complex motor and vocal tics in an elderly woman responsive to opiates. *Int J Neurosci* 1989;**44**:317–20.
16 Fliman M, Dickman M, Perl E. Gilles de la Tourette syndrome: onset in old age. *J Am Geriatr Soc* 1991;**39**:277–9.
17 Sutula T, Hobbs WR. Senile-onset vocal and motor tics. *Arch Neurol* 1993;**40**:825–6.
18 Bharucha KJ. Multiple adult-onset tics resembling Tourette's syndrome [abstract]. *Mov Disord* 1996;**11**:607.
19 Sethi KD. Adult-onset tic disorders [abstract]. *Neurol* 1991; **41**(suppl 1):227.
20 Comings DE, Comings BG. Tourette syndrome: clinical and psychological aspects of 250 cases. *Am J Hum Genet* 1985;**37**:435–50.
21 Tolosa E, Jankovic J. Tics and Tourette's syndrome. In: Jankovic J, Tolosa E, eds. *Parkinson's disease and movement disorders.* 3rd ed. Baltimore: Williams and Wilkins, 1993:513–52.
22 Pauls DL, Towbin KE, Leckman JF, *et al*. Gilles de la Tourette syndrome and obsessive compulsive disorder: evidence supporting a genetic relationship. *Arch Gen Psychiatry* 1986;**43**:1180–2.
23 George MS, Trimble MR, Ring HA, *et al*. Obsessions in obsessive-compulsive disorder with and without Gilles de la Tourette's syndrome. *Am J Psychiatry* 1993;**150**:93–7.
24 Wand RR, Matazow GS, Shady GA, *et al*. Tourette syndrome: associated symptoms and most disabling features. *Neurosci Biobehav Rev* 1993;**17**:271–5.
25 Goetz CG, Tanner CM, Wilson RS, *et al*. A rating scale for Gilles de la Tourette's syndrome: description, reliability and validity data. *Neurology* 1987;**37**:1542–4.
26 Tanner CM, Goldman SM. Epidemiology of Tourette syndrome. *Neurol Clin* 1997;**15**:395–402.
27 Leckman JF, Zhang H, Vitale A, *et al*. Course of tic severity in Tourette syndrome. *Pediatrics* 1998:**102**:14–9.