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The Prevalence of Gilles de la Tourette Syndrome in children and adolescents with autism: A large scale study.

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Abstract

Background

An earlier small-scale study (n=37) of children with autism revealed that 8.1% of these were comorbid for Gille de la Tourette Syndrome (GTS). The present study is a large scale test of whether this result replicates.

Method

447 pupils from 9 schools for children and adolescents with autism were screened for the presence of motor and vocal tics.

Results

Subsequent family interviews confirmed the comorbid diagnosis of definite GTS in 19 children, giving a prevalence rate of 4.0%. A further 10 children were diagnosed with probable GTS (2.2%).

Conclusions

These results indicate that the rate of GTS in autism exceeds that expected by chance, and the combined rate (6.2%) is similar to the rates found in the smaller-scale study (8.1%). Methodological considerations and alternative explanations for an increased prevalence are discussed.

Gilles de la Tourette Syndrome, or Tourette Syndrome (GTS) is a neurodevelopmental disorder defined by the presence of chronic, multiple motor and vocal tics of childhood onset (APA, 1987). The average age of onset is reported to be 5 years of age (Leckman et al., 1998). The tics show a fluctuating course, and may decrease in severity during adulthood. The precise aetiology of GTS is unknown. In most cases however, GTS appears to be genetically transmitted (Curtis, Robertson & Gurling, 1992), although the exact pattern of inheritance is still unknown. Implicated neurological abnormalities include dysfunction of the basal ganglia and/or the prefrontal cortex (reviewed in (Chase, Geoffrey, Gillespie & Burrows, 1986), and biochemical abnormalities of the dopamine and serotonin neurotransmitter systems (reviewed in (Baker, Chokka & Bornstein, 1995). GTS is often accompanied by obsessive-compulsive behaviours (Eapen, Robertson, Alsobrook II & Pauls, 1997; Frankel et al., 1986). These may be an alternative expression of the putative GTS gene(s) (Pauls, Towbin, Leckman, Zahner & Cohen, 1986).

Autism is also a neurodevelopmental disorder, itself defined by abnormal social and communication development, with a pattern of restricted and repetitive interests and activities (APA, 1994). Autism has an earlier age of onset than GTS (usually by 18 months of age) and often shows a chronic course. The precise aetiology of autism is also unknown, although possible aetiological factors include genetic (Bailey et al., 1995; Folstein & Rutter, 1977), neurobiological (Bauman & Kemper, 1994) and cognitive (Baron-Cohen, 1995; Frith, 1989) abnormalities.

Recently, a growing number of case-reports have documented the co-occurrence of autism and GTS in the same individuals. Realmuto and Main (Realmuto & Main, 1982) were the first to report the development of GTS in a child with autism. These authors interpreted this as a chance association, as did a subsequent report of GTS in autism (Barabas & Matthews, 1983). However, Barabas and Matthews also discussed the possibility of a common neurochemical abnormality.

The estimated general population prevalence of autism is 1 per 1000 (Baron-Cohen et al., 1996), and the estimated general population prevalence of GTS is 2 per 10,000, (Robertson, 1994). This is likely to be an underestimate, as GTS is often undetected. Hence, if autism and GTS are truly independent, the rate of co-occurrence expected by chance would be 2 per 10 million of the general population, 1 per 1000 individuals with GTS, and 2 per 10,000 individuals with autism.

A few studies have documented the prevalence of GTS in populations of individuals with autism spectrum disorders. Kerbeshian and Burd (Kerbeshian & Burd, 1986) reported a clinical series of 6 individuals with Asperger Syndrome, of whom 3 (50.0%) had comorbid GTS. The same group (Burd, Fisher, Kerbeshian & Arnold, 1987) reported a rate of 20.3% of GTS in an ascertained sample of 59 individuals meeting DSM-III criteria for infantile autism or pervasive developmental disorder (PDD). Of the 12 children with comorbid GTS, 10 had an atypical PDD, whilst only 2 had autism. This group also showed significantly higher IQ than the group with PDD without GTS, and significantly higher measures of receptive and expressive language. Burd et al. believed this to be an indication that comorbid GTS in children with

autism provided a marker for improved developmental outcome. Kano's group (Kano, Ohta & Nagai, 1987; Kano, Ohta, Nagai, Yokota & Shimizu, 1988) described 2 children with autism and GTS, drawn from a sample of 76 children with autism, suggesting a much lower prevalence of 2.6%.

We are not aware of any studies reporting the rate of comorbid autism and GTS in the general population. However, Sverd (Sverd, 1991) reported 10 children with autism or PDD and comorbid GTS. Given the population statistics for the geographical regions from which these children were clinically ascertained, Sverd argues that these children represent a rate of comorbidity exceeding that of chance.

Although distinct disorders, autism and GTS share several behavioural features. Here we list some of these shared features, whilst pointing out how these may differ in the two syndromes: (1) Echolalia and palilalia are common in both GTS and autism, although in autism, unlike GTS, these behaviours may be appropriate to the level of speech development. (2) Types of obsessive-compulsive behaviours are frequently seen in both autism and GTS, although in autism these may be better described as rigid and ritualistic behaviours, such as an insistence upon sameness and resistance to change. (3) Like GTS, autism is associated with abnormal motor behaviours, although in autism these often take the form of stereotypies, such as spinning, rocking, and hand flapping.

A study carried out in a special school for children with autism (Baron-Cohen, Mortimore, Moriarty, Izaguirre & Robertson, 1999), finding that three out of the 37

pupils (8.1%) had comorbid GTS. Previous studies had generated prevalence rates retrospectively from clinical series. A special school population of children and adolescents with autism were used, with a prospective, multi-stage design, using direct observation in the classroom, and, later, both pupil and family interviews. This may have produced a more accurate estimate of the prevalence of GTS in children with autism than previous studies. However, the sample size was small. The current study aimed to replicate the earlier study, to establish the rate of GTS in a special school population, but this time with a much larger sample.

Method

Participants

33 schools for children with autism from around England were invited to take part in the study. Of these, 9 schools agreed to participate within the timescale of the study. We have no reason to suspect these were not representative of children with autism spectrum conditions more generally, as 3 schools were in the North, 4 were in the Midlands, and 2 were in the Greater London area. The schools catered for a mixture of day and residential students. The total number of children with autism within these schools was 458, with a mean age of 11:1 (years:months; range 3:6 to 19:8) and a sex ratio of 4.9:1(male:female). The children's parents were contacted, via the schools, to

request their consent for them to take part in the study. The parents of two children (0.4%) declined to take part. Nine children for whom parental consent was given were unavailable for observation at the time of the study. Hence the total number of children participating was 447, with a mean age of 11:1 (years:months; range 4:2 to 19:8), and a sex ratio of 5:1. The children's diagnoses showed varying degrees of autism. 280 were diagnosed as having autism, 141 received a diagnosis of autism spectrum condition, and 26 were diagnosed as having Asperger Syndrome. These diagnoses were not made by our team but were taken from the school notes, in all cases from reports by a child psychiatrist or paediatrician.

Procedure

Stage one

The records of all children included in the study were reviewed to identify any children who had previously been assessed for a tic disorder, or had previously received a diagnosis of GTS.

Stage two

Where possible, the symptoms of GTS were discussed with teaching staff, using an advisory leaflet adapted from the Tourette Syndrome UK. Some teachers were then asked to comment on the habits and movements of children in their classes.

Stage three

An observer (VS, a psychologist) carried out observations of all participating children. Children were observed at school, in their classrooms and during their usual

school activities. Class sizes ranged from 3 children to 9, with an average class size of 6. Each child was observed for at least 10 minutes, using a time-interval sampling observational procedure. The presence of motor and vocal tics was recorded. Each child was subsequently classified as having motor tics, vocal tics, motor and vocal tics, or no tics, on observation. Children classified as having motor *and* vocal tics were entered directly into Stage 6. Children classified as having only motor *or* vocal tics were entered into Stage 4.

Stage four

Children classified as having motor *or* vocal tics (but not motor *and* vocal tics) at Stage 3 were re-observed at their school by the observer, for a further 10 minutes each, a few days later. Children for whom both motor *and* vocal tics had been observed, when all observations (Stages 3, 4 and 5) were pooled, were also entered into Stage 6.

Stage five

Some children (n=55) were later observed by an independent observer (HH, also a psychologist) for 10 minutes each. Of these children, 23 had been classified by the first observer as showing tics, and 32 had been classified as showing no tics. The names given to this observer were presented in a random order, with no indication of their initial tic classification.

Stage six

The parents of the children classified as showing both motor *and* vocal tics on observation were invited to meet a psychiatrist (MMR or JJ) to discuss their child's

possible diagnosis of GTS, and to obtain a family history. Children and parents were interviewed using a short version of the National Hospital Interview Schedule (NHIS) (Robertson & Eapen, 1996). The Yale Global Tic Severity Scale (YGTSS) (Leckman et al., 1989) was also used. The diagnostic interviews were conducted at the children's schools, or at University College London Middlesex Hospital (n=1). Interviews were conducted at the child's school as far as was possible, to reduce anxiety. Each child was also observed for 30 - 45 minutes, as part of the interview. DSM-III-R (APA, 1987) criteria for GTS were used in preference to DSM-IV (APA, 1994) criteria, as DSM-IV requires the additional criterion that tic symptoms must cause marked distress or significant impairment. The adoption of this subjective criterion is inappropriate for many research purposes (Erenberg & Fahn, 1996; Freeman, Fast & Kent, 1995; Kurlan et al., 1997). A particular issue for this study is that impairment and distress may be difficult to establish in children with limited communication and in some children with learning disability.

If the parents were unable to attend the interview, but wished their child to be included in this stage of the study, the child was accompanied by his or her keyworker from the school, and a phone interview with the parents was conducted later by a psychiatrist. It should be noted that in a study of this kind there is a risk that stereotypies will be confused as tics. The distinction between these is hard to draw, but if either of the raters or clinicians thought a behaviour could be a stereotypy, this was discounted as a tic, in order to err on the side of being conservative.

Results

Stage one

No evidence for previous assessments or diagnoses of GTS or alternative tic disorders was found in the records of any of the participating children.

Stage two

Our previous study (Baron-Cohen et al., 1999) showed teachers' reports to be fairly reliable. Therefore the children identified by their teachers as showing tic-like behaviours were noted and this data was used to help classify children.

Stage three - initial observation (VS)

30 children were identified as having motor and vocal tics by the first observer (VS) after Stage 3. These children were entered directly into Stage 6. A further 114 children were identified for whom motor *or* vocal tics were observed by the first observer. These children were entered into Stage 4.

Stage four - re-observation (VS)

Of the 114 children who were entered into Stage 4, 18 were unavailable for re-observation by the first observer. Their classification therefore remained the same. For the remaining 96 children, a further 10 were identified for whom both motor and

vocal tics had been observed, when both Stage 3 and 4 observations were pooled. These children were also entered into Stage 6.

Stage five - re-observation (HH)

55 children were seen by the second observer (HH), one to two weeks after the initial observations. All observations, from Stages 3, 4 and 5, were pooled (for example, if one observer saw motor tics and the other vocal tics, that child was classified as showing motor and vocal tics). Agreement on ticcing vs. not ticcing between the two observers was 70.4%. The two observers agreed that 12 out of the 55 children were showing tic-like behaviours, 26 showed no tic-like behaviours. They disagreed on the classification of 16 children. Both observers were trained in the identification of tics by MMR at the Tourette Clinic, at the National Hospital for Neurology and Neurosurgery, Queen Square, London. The implications of this result are discussed below. When the observations of both observers were combined, the final results were as follows: Out of the 447 children entered into the study, 43 children were identified as showing both motor and vocal tics, 98 were identified as showing motor tics only, and 11 were identified as showing vocal tics only.

Stage six

Forty-three children entered Stage 6 of the study. The parents of 10 children declined to be interviewed. One child was unavailable at the time of the appointment. For the remaining children, at least one parent was interviewed. 23 children were accompanied by their parents to interview, 9 were accompanied by their keyworker or teacher, and the parents later interviewed by phone. The diagnosis of definite GTS

was confirmed after family interview for 19 of the 32 children. A further 10 children were diagnosed as having probable GTS. If the child showed symptoms at interview and there was a personal history of symptoms, a diagnosis of definite GTS was made. If one of these two criteria was met, but not the other, a diagnosis of probable GTS was made. For the remaining children, 2 were diagnosed with chronic motor tic disorder, and one was diagnosed as having Rett Syndrome. This is a Pervasive Developmental Disorder, characterised by the development of autism, dementia, apraxia of gait and stereotyped use of the hands, following a period of at least 5 months normal functioning after birth (Hagberg, Aicardi, Dias & Ramos, 1983).

For definite cases, the rate of true positives was 59.38%, although if we include probable GTS cases, the true positive rate rises to 90.63%. The rate of false positives was 9.38%. The method employed in this study does not allow the calculation of true and false negatives.

Medication information was provided by the parents, and 6 of the 32 children were taking psychotropic medication (4 definite, 2 probable GTS), 3 were taking anti-convulsants (2 probable GTS, 1 Rett Syndrome plus probable Chronic Motor Tic Disorder) and one was taking Ritalin (definite GTS). The onset of tics in this child predated the commencement of medication.

YGTSS scores were calculated for all 32 children. The scores ranged from 4% to 63%, with a mean of 28% (sd = 16.7).

For the children found to have comorbid GTS and autism, 20 had been diagnosed with autism (7.1% of children with autism had comorbid GTS), 7 had been diagnosed with an autism spectrum condition (5.0% of children with an autism spectrum condition had comorbid GTS) and 2 had been diagnosed with Asperger Syndrome (7.7% of children with Asperger Syndrome had comorbid GTS). When the frequencies of comorbid GTS in children with autism, autism spectrum conditions and Asperger Syndrome were compared, there were no significant differences (chi-squared = 0.43, df = 2, ns).

Family histories were collected for all 32 children. For 25 of the 32 children (78%), there was found to be a paternal and/or maternal family history of tics and/or obsessive-compulsive behaviours. These results are shown in Table 1.

INSERT TABLE 1 HERE

Discussion

The observed rate of 6.48% of GTS (including 'probable' GTS cases) in this special school population of children with autism far exceeds that expected by chance. This

study, with its prospective, multi-stage design, using combined observational and family interview/history methods, and large sample size, is likely to have yielded the most accurate estimate yet of the prevalence of GTS in children with autism. Previous studies have retrospectively assessed the rate of GTS in clinical series of individuals with autism, and the earlier study carried out by this team (Baron-Cohen et al., 1999) had a much smaller sample size, although it used a similar method. The rates found in these two studies are also similar (chi-squared = 0.43, df = 1, ns). Thus the current study supports the findings of the smaller study, and the methods used.

It was suggested (Burd et al., 1987) that comorbid GTS in children with autistic-type conditions provides a marker for improved developmental outcome. The present study was unable to collect data on IQ, and measures of receptive and expressive language, and so cannot be directly compared with Burd et al.'s study. However, the fact that GTS was found to be equally common in children with autism, children with autistic spectrum conditions and children with Asperger Syndrome shows that GTS is not related to the severity of autism in the child.

It is also notable that a significant proportion of children not identified as having GTS did, however, show motor *or* vocal tics (but not both motor *and* vocal tics) on observation. 109 children (24.4%) were showing tics on observation, but did not show full GTS symptoms. Children with GTS show a fluctuating course of tic expression, and so it is plausible that some of those children showing only motor *or* vocal tics, if observed for a longer period, would have shown both motor *and* vocal tics, and so would have entered the final stage of the study. Indeed, this is

demonstrated by the 10 children who were identified as having both motor *and* vocal tics only after re-observation at Stage 4. This is also evident in the 4 children who were identified as having only motor *or* vocal tics, or even no tics in one case, by one observer, but as having both motor *and* vocal tics by the other, either a week later or a week earlier, and the one child who was observed as having motor tics only by one observer, and vocal tics only by the other. This may point to the need for a longer period of observation, to increase the chances of identifying those children with GTS. The observed rate of 6.48% of GTS in children with autism may therefore be an underestimate.

The high rate of tics observed in these children (34.0% of children were classified as showing tics) is interesting, as no children had previously been diagnosed as having a tic disorder. This may indicate that children with autism also show a higher prevalence of alternative tic disorders, such as transient tic disorder, chronic motor tic disorder and chronic vocal tic disorder, but that this is being overlooked, possibly since they are occurring in the context of the other problems associated with autism. The possibility of an increased rate of other tic disorders in autism has relevance for genetic studies.

Is it also possible that the high observed rate of tics in children with autism reflects the difficulty in distinguishing tics from other abnormal movements and noises in this population? This differential diagnosis problem has been previously documented (Burd et al., 1987). In particular, complex motor tics can be difficult to distinguish from stereotypies in the absence of self reported subjective experiential information,

particularly regarding the volitional nature of behaviours. Differentiating vocal tics from the wide range of vocal productions in children with autism may be even more problematic. Apart from the clear cut tics and stereotypies, there are quite a number of behaviours that can only be understood through careful enquiry about the nature of the movement or noise, its longitudinal course, and possible alternative explanation for the symptom. For example, a vocalisation which was initially noted as a sniffing tic was later discovered, after questioning, to be a repetitive imitation of a TV character with which the child was currently obsessed. However, as mentioned in the Burd et al. study, tics, in contrast to stereotypies, are typically short lived, contextually inappropriate, and interrupt the flow of behaviour or speech. It was also attempted (Kano et al., 1988) to systematize the difference between motor tics and stereotypies by their topography (tics tend to be clearer in the face, neck shoulders and arms, compared to hands and fingers), their nature (spasmodic versus rhythmic) and also by the quality of the subjective experience and their response to psychosocial factors. These factors were taken into account when the observations were made, and we are therefore confident that the result obtained represents a true co-morbidity.

It is well known that the tics encountered in GTS wax and wane in severity and fluctuate with time. Stress or anxiety may increase tics, whereas concentrating on a task may reduce tics. Thus, if the raters observed the children with autism at different times, for up to ten minutes, and on different days (sometimes more than a week apart), it is quite conceivable that different tics occurred at different times. The levels of stress and anxiety may also have been different, and the children may also have been observed at different times during different tasks (e.g., concentrating). The

raters were trained at the Tourette clinic at the National Hospital Queen Square during both new-patient and follow-up clinics. Inter-rater reliability was assessed on the tic observation section of the NHIS. Training was carried out for approximately six weeks, seeing about 40 GTS patients (VS), while the other psychologist (HH) sat in on the clinic and rated many more GTS patients over a year's duration. Agreement between the two raters was only modest, and this may well have been due to factors described above.

A study by Eapen et al (Eapen, Robertson, Zeitlin & Kurlan, 1997), found that children in a special school population had an increased prevalence of GTS. 55% of emotionally/ behaviourally disturbed children (EBD) and 20% of children classed as learning disabled (LD) were diagnosed as having GTS. Whereas the EBD result could indicate that children with EBD have such problems due to the disruptive effects that GTS can have on the individual, the LD result could point to a larger picture - that children with learning difficulties are more prone to GTS. IQ data were not available for the children in the current study, thus we cannot say whether the results we obtained, of 6.48% GTS in children with autism, can be attributed to their more general learning difficulties. Further research in this area is necessary to better clarify the links between these conditions.

The generally accepted prevalence figure for GTS is around 0.5 per 1000 (Bruun, 1984); this figure has also been reached in a careful epidemiological study (Apter et al., 1993). A more recent pilot study by Mason et al (Banerjee, Mason, Eapen, Zeitlin & Robertson, 1998) in the UK yielded higher results and found the rate of GTS in a

mainstream school population to be around 3%. This study, however, had a small sample size (n=166), and the identified cases were not re-assessed and formally diagnosed by an expert. It has been criticised (Traverse, 1998) and stirred debate (Banerjee et al., 1998). The actual prevalence rate of GTS has therefore yet to be determined, but the currently accepted figure remains around 0.5 per 1000. We await the results of our larger definitive study in a UK school population.

The YGTSS scores in our GTS individuals ranged from 4% to 63% with a mean of 29% (see Table 1). The majority of the scores indicate mild to moderate severity. The scale range is 0% - 100%. In the only UK GTS clinic study published using the scale (a modified version with the total range being 0 - 55) the GTS cases scored a mean of 26.2 (range 11 - 55), (Robertson, Banerjee, Fox-Hiley & Tannock, 1997). This would be between 45% and 55% using the currently used version of the scale. In another study (Robertson et al., submitted), on 280 consecutive GTS clinic cases, the YGTSS scores ranged from 1% to 100% (mean 49%; sd = 23). Both these studies indicate that the GTS/autism individuals are not nearly as severe as clinic patients. In the present study, some of the YGTSS global tic severity scores may have been higher than expected in this population due to the presence of, for example, echophenomena, which occur in both GTS and autism and which symptom receives a separate score on the YGTSS.

The short version of the NHIS (Robertson & Eapen, 1996) was used in this study. This goes into detail with regards to individual tics, and the interviewer records whether specific tics have taken place in the past (ever), in the past week (which

allows the YGTSS to be completed), as well as those observed at interview. Tics include simple ones such as frowning, raising eyebrows, blinking, winking, eye movements, nasal twitches, mouth twitching, pouting and opening, tongue protrusion, facial grimacing, platysma tightening, head nodding and shaking, shoulder shrugging and flicking the hair out of the eyes; simple vocalisations include grunting throat clearing, sniffing snorting, grunting and coughing. These were the majority of tics noted in the study. Complex tics (motor and vocal) and stereotypies may, however, well be difficult to differentiate from each other, and include, for example, hand-flapping (common in people with autism; rare in people with GTS, and not regarded as a tic in this study), twirling (also not encountered in this study), vocalisations, inappropriate fluctuations in pitch of the voice, and squealing. Of course, some symptoms such as echolalia and echopraxia, are common in both autism and GTS, and may be indistinguishable from each other phenomenologically in each condition. It is acknowledged that the relatively higher scores in the complexity score of the YGTSS may well have been partly due to the presence of symptoms such as ecophenomena; the senior author (MMR) rated the pupils on the YGTSS and included only what she considered to be tics, apart from the ecophenomena. It must be noted that 18 pupils had echolalia, while 12 had echopraxia (10 of these had echolalia as well). We acknowledge the difficulties in differentiating between the two.

Our new screening method suggests that a larger scale cognitive study of the effects of comorbidity would now be possible. These effects were investigated in the Baron-Cohen and Robertson (Baron-Cohen & Robertson, 1995) case studies. A child with

autism, a child with GTS and a child with both conditions were tested for theory of mind, intention-editing and executive function deficits. The predictions, that the child with autism would show a deficit in theory of mind tests, that the child with GTS would show a deficit in intention-editing tests, and that the child with both conditions would show deficits in both these areas, were supported. A larger scale study, with similar methods, may help to confirm these results, which have the natural limitations of single case studies.

One of the purposes of this study was to find children who were being overlooked for treatment. In comorbid cases of autism and GTS, the latter is often overlooked (as in 100% of our cases) and the symptoms are therefore left untreated. Finding these comorbid cases allows the children's pharmacological management to be reviewed, and alleviation of tics can help to improve their quality of life. Three such cases were found in this study. By themselves, these cases provide a clinical justification for such screening. For the other children, diagnosed with mild GTS, and not needing medication, knowledge of their condition can help parents and teachers in their continuing support for the child.

The observed elevated rate of GTS in children with autism is consistent with the operation of common aetiological factors, and does not support a chance co-occurrence. Possible common aetiological factors include neurochemical and frontal lobe abnormalities. That there is a substantial family history of GTS or GTS spectrum disorders suggests that there may also be independent aetiological mechanisms at play. Future work addressing these possibilities will be important in furthering our

understanding of the respective pathogeneses of these two neurodevelopmental disorders.

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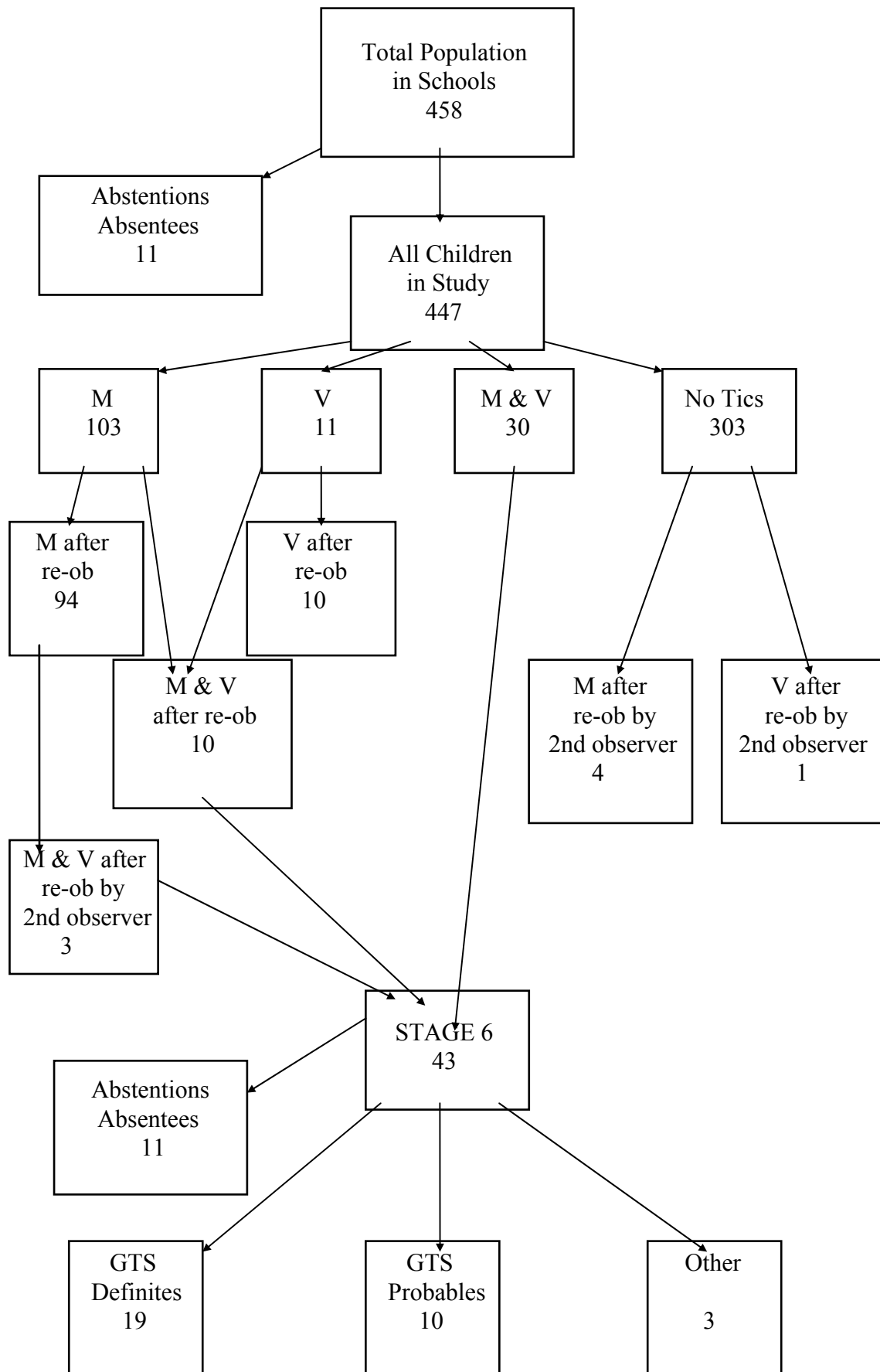


Fig. 1. GTS cases: screening and selection flow chart.
 M = motor tics V = vocal tics M & V = motor plus vocal tics

Table 1: Diagnosis, Severity of tics, and family history.

Child	Diagnosis 1	Fam. History TS/Tics/OCB	Yale Score	Diagnosis 2
1	autism	M	6%	mild GTS
2	autism	x	15%	probable GTS
3	autism	P	15%	mild GTS
4	spectrum	x	17%	probable GTS*
5	autism	M	18%	definite CMT
6	autism	P	48%	mild GTS
7	spectrum	M	17%	definite GTS
8	autism	x	40%	probable GTS
9	spectrum	x	26%	probable GTS
10	autism	P	41%	severe GTS
11	autism	M	54%	mild GTS
12	autism	?M	40%	severe GTS
13	autism	M	24%	mild GTS
14	autism	P	49%	mild GTS
15	spectrum	M	62%	mild GTS
16	autism	x	12%	mild GTS
17	autism	x	35%	probable GTS
18	autism	PM	7%	prob. CMT*
19	spectrum	x	16%	mild GTS
20	autism	P	30%	mild GTS
21	spectrum	M	21%	mild GTS
22	autism	PM	4%	probable GTS*
23	AS	P	56%	moderate GTS
24	autism	PM	7%	definite CMT
25	autism	P	33%	mild GTS
26	autism	M	37%	mild GTS
27	spectrum	M	25%	probable GTS
28	autism	M	25%	mild GTS
29	autism	P	32%	probable GTS
30	autism	M	15%	mild GTS
31	autism	PM	63%	probable GTS
32	AS	M	18%	mild GTS

*

Child 4: probable GTS, definite Chronic Motor Tic Disorder (CMT)

Child 18: probable CMT, definite Rett's Syndrome

Child 22: probable GTS, definite Chronic Vocal Tic Disorder

Family History: Tourette Syndrome / Tics / Obsessive-Compulsive Behaviours

M: history on maternal side of family

P: history on paternal side of family

x: no family history