TIC DISORDERS

Hannah Metzger, Sina Wanderer & Veit Roessner

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Samuel Johnson, one of the most influential English literary figures, particularly because of his “Dictionary of the English Language” published in 1755, appears to have suffered from Tourette’s disorder.

Portrait by Joshua Reynolds
Tics may range from a discrete, hardly noticeable flinching of the eye to a painful, socially incapacitating und subjectively shameful phenomena involving several muscle groups. Those afflicted by tics, as well as their family, may experience substantial suffering due to the symptoms, be it through bullying or to inappropriate response by caregivers resulting in a dysfunctional parent-child relationship. People in the extended environment may also react with irritation, for instance where vocal tics occur in inappropriate settings, such as the cinema or the classroom. On the other hand, some of the people afflicted successfully develop strategies to control their tics and learn to live and cope with them.

Classification usually follows the criteria of the International Classification of Diseases (ICD-10; World Health Organisation, 1996) or the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). Tic disorders are best classified among the neuropsychiatric disorders. Table H.2.1 shows the classification of tic disorders.

Whether a transient or a chronic tic disorder is present depends on the duration of symptoms: in the case of a transient tic disorder, symptoms last less than 12 months. Transient tic disorders mostly occur in school age children and usually do not require specific treatment.

Diagnosis of Gilles-de-la-Tourette syndrome (or simply Tourette syndrome or disorder) is warranted in cases where several motor tics and at least one vocal tic are present at the same time or have been present in the past. Motor and vocal tics do not have to be present at the same time but should have occurred almost every day over one year at least to warrant the diagnosis. The onset of Tourette syndrome is generally before the age of 18; it rarely occurs for the first time in adulthood.

**Epidemiology**

It is estimated that 4% to 12% of all children suffer from tics at some time during their development. Approximately 3%-4% are afflicted by a chronic tic disorder and 1% with Tourette's syndrome (Rothenberger et al, 2007). Children and adolescents are 10 times more likely to suffer from tics than adults (Kerbeshian & Burd 1992). This may be due to the high spontaneous remission rate in younger patients. Boys are afflicted three to four times more often than girls (Freeman, 2007). A familial predisposition has been established (O'Rourke et al, 2011).

**Table H.2.1 Classification of tic disorders according to ICD-10 and DSM-IV**

<table>
<thead>
<tr>
<th>ICD-10</th>
<th>DSM-IV</th>
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</thead>
<tbody>
<tr>
<td>F95.0</td>
<td>Transient tic disorder</td>
</tr>
<tr>
<td>F95.1</td>
<td>Chronic motor or vocal tic disorder</td>
</tr>
<tr>
<td>F95.2</td>
<td>Combined vocal and multiple motor tic disorder (Gilles-de-la-Tourette syndrome)</td>
</tr>
<tr>
<td>F95.8</td>
<td>Other tic disorder</td>
</tr>
<tr>
<td>F95.9</td>
<td>Unspecified</td>
</tr>
</tbody>
</table>
Cultural differences

The prevalence of Tourette syndrome is about 1% worldwide (Robertson et al, 2009). However, Tourette syndrome is less likely to occur in some countries. Differences in prevalence across various countries seem partly to reflect the fact that not all follow the same classification system. China, for example, has reported slightly lower prevalence rates; rates appear to be lower among African Americans in the US and occurrence is extremely rare in Sub-Saharan black Africans. Possible reasons for these findings include the implementation of different diagnostic systems, other medical priorities with less likelihood to seek treatment, ethnic and epigenetic differences, genetic and allelic differences in different races and the presence of a mixture of races (Robertson, 2008). By contrast, a cross-cultural review by Staley et al (1997) concluded that demography, family history, clinical features, associated conditions, comorbidity and treatment outcome were basically the same across cultures.

Age at onset and course

Tics generally occur for the first time between the ages of two and 15 years. However, the peak age of onset is between six and eight years. Typically, the first symptom is a simple motor tic in the face, such as eye blinking or grimacing. With time, they spread to shoulders, extremities and torso. Often vocal tics appear two to four years after the start of the motor tics (Leckman et al, 1998).

In most cases tics fluctuate in their location, complexity, type, intensity and frequency. This can be confusing and frustrating for parents of children afflicted by tics. Fluctuations often occur at irregular intervals, approximately every six to 12 weeks, without any apparent reason (Roessner et al, 2004). This changing course is one of the main distinguishing features when differentiating between Tourette syndrome and the abnormal movements found in conjunction with other illnesses, such as dystonia or chorea, which typically do not change or show less accentuated fluctuations.

Figure H.2.1 Evaluation of treatment efficacy in Tourette’s syndrome in light of the natural waxing and waning

At date 1 (in which a reduction in tics naturally occurs) a therapeutic intervention is followed by a reduction in tics irrespective of the intervention’s potential to increase or not to have an effect on tics. The reduction could be falsely attributed to the intervention when in fact was the result of the tics natural waxing and waning. A therapeutic intervention at date 2 could be followed by an increase in tics despite its potential to reduce tics. The effect of the therapeutic intervention might attenuate the natural waxing of the tics but is biased by the spontaneous increase. This means that a meaningful appraisal of treatment efficacy in Tourette’s disorder can only be ascertained in most cases after a long observation period. Source: Roessner et al (2011). Reproduced with permission.

Tic

Sudden, rapid, recurrent, nonrhythmic motor movement or vocalization.
Usually there is a worsening of symptoms during adolescence. As children progresses into young adulthood, tics often go into remission (Sandor et al, 1990). As a result, children and adolescents are 10 times more likely to be affected than adults.

With increasing age, tic-afflicted patients also gain better control over their tics and are often able to suppress them for minutes or up to several hours. However, after a period of suppression, patients often feel compelled to exhibit their tics with heightened intensity (Banaschewski et al, 2003). For this reason some children may be able to suppress their tics over the course of the school day but, as soon as the child arrives at home, tics reappear with more intensity and the feelings of heightened tension generated by the suppression of tics will temporarily fade.

The severity of the tic disorder during childhood only has a limited predictive value concerning the illness in adulthood. A poor prognosis is usually associated with:

- Familial history
- Existence of vocal or complex tics
- Comorbid hyperkinetic disorder
- Obsessive-compulsive symptoms
- Aggressive behavior against self or others.

Spontaneous remission of chronic simple or multiple tics occurs in 50% to 70% of cases and 3% to 40% for Tourette syndrome (Erenberg et al, 1987).

**ETIOLOGY AND RISK FACTORS**

Although the cause of primary tic disorders has not been conclusively determined, it is widely assumed to be the result of an interaction of genetic, neurobiological and psychological factors as well as environmental influences. A dysregulation within cortico-striato-thalamo-cortical circuits with deviations within the dopaminergic and serotonergic systems is believed to be responsible for the occurrence of tics. It seems that overactivity of the dopaminergic system in the basal ganglia leads to deficient subcortical inhibition and impaired automatic control of movement, which then clinically presents itself as motor or vocal tics (Leckman et al 1997; Singer, 2011).

A familial predisposition is as a risk factor. Heritability has been estimated to be around 50% (Singer & Walkup, 1991). Various prenatal, perinatal and postnatal factors are considered possible factors that increase the risk. They include premature birth, perinatal hypoxia, low birth weight as well as excessive nicotine and caffeine consumption by mother during pregnancy. On rare occasions tics may develop as secondary symptom of tumors, poisoning, infection, head trauma or vascular disease (Burd et al, 1999; Mathews et al, 2006).

Medical imaging techniques have determined that, on a neuroanatomical level, patients with tics show a reduced volume of the basal ganglia as well as the corpus callosum, but heterogeneity of study samples in terms of several confounders (e.g., long-term use of medication, tic performance and suppression over years) prevents firm conclusions. Furthermore, deviation of glucose metabolism in the basal ganglia, prefrontal and the somatic sensorimotor cortex, insula and
temporal lobe has become apparent. Apart from dopaminergic overactivity, other neurotransmitters implicated include dysfunctions within the serotonergic and noradrenergic systems.

In terms of psychosocial factors, poor child rearing techniques have been ruled out as risk factor. However, environmental influences, first and foremost psychosocial stress, undoubtedly modulate tic severity. Experiences that cause fear, emotional trauma and social pressure generally result in an exacerbation of tics.

**TICS**

Tics are sudden, abrupt, fast movements comprising various muscle groups, with or without vocal utterances, which occur involuntarily. Tics are brief but repetitive – though not rhythmic – and usually appear in short bursts or even series. They may be classified according to the degree of complexity (simple, complex) as well as their quality (motor, vocal) (Rothenberger et al, 2007).

**Motor tics** range from simple, sudden movements such as eye blinking or grimacing, to complex behavioral patterns, for example crouching down or hopping. In extreme cases, complex motor tics may present themselves as obscene gestures (referred to as *copropraxia*; e.g., pulling trousers down) or even have an element of self-harm (e.g., hitting oneself in the head). In some cases the afflicted person is compelled to repeat or imitate a movement observed in another person (*echopraxia*).

**Vocal or phonic tics** are involuntary utterances of sounds, noises, sentences or words. A simple vocal tic may be a slight coughing, clearing of throat, wheezing, squeaking or loud shouting. More complex vocal tics involve syllables, words or sentences. *Coprolalia* is the utterance of obscene or aggressive words or sentences. Coprolalia occurs seldom, in less than 20% of persons affected by Tourette disorder (Rothenberger et al, 2007). In other cases sufferers feel compelled to repeat their own previously spoken words (*palilalia* – it is called *echolalia* if they repeat words previously spoken by someone else).

By the age of 10 or 11 years, children begin to report a premonitory urge. This can be any kind of sensation, typically a tickling, itching or prickling feeling, in the area of the muscle groups involved, announcing the imminent occurrence of a tic (Steinberg et al, 2010).

<table>
<thead>
<tr>
<th>Motor Tics</th>
<th>Vocal Tics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye blinking</td>
<td>Coughing</td>
</tr>
<tr>
<td>Rolling of eyes</td>
<td>Throat clearing</td>
</tr>
<tr>
<td>Grimacing</td>
<td>Sniffing</td>
</tr>
<tr>
<td>Shaking of head</td>
<td>Whistling</td>
</tr>
<tr>
<td>Twitching of shoulders</td>
<td>Grunting</td>
</tr>
<tr>
<td>Twitching of torso and pelvis</td>
<td>Animal sounds</td>
</tr>
<tr>
<td>Twitching of abdomen</td>
<td>Uttering of syllables</td>
</tr>
<tr>
<td>Movements of the hands and arms</td>
<td>Uttering of words</td>
</tr>
<tr>
<td>Movements of the feet and legs</td>
<td>Shouting</td>
</tr>
</tbody>
</table>
DIAGNOSIS

A detailed medical history should be obtained from birth onwards. Additionally, standardized questionnaires may be used. The Child Behavior Checklist (Achenbach, 1991) can be employed to obtain information concerning possible comorbid disorders (see Chapter A.5). The Strengths and Difficulties Questionnaire (Goodman, 1997) can also be recommended for this purpose (see Chapter A.5). Tic-specific or semi-structured interviews include the Yale Global Tic Severity Scale (YGTSS) (Leckman et al, 1989) and the Tourette's Syndrome Severity Scale (TSSS) (Walkup et al, 1992). Parental or self-rating can be done using the Yale Tourette Syndrome Symptom List-Revised (TSSL-R) (Leckman et al, 1989).

A thorough physical and neurological examination should be conducted, including an EEG. The main purpose for this is to exclude other possible illnesses that could cause the symptoms. Usually no further tests, such as an MRI, are necessary unless there are pathological findings. ECG, thyroid function tests or other procedures (e.g., metabolic tests) are not necessary in the absence of abnormal findings.

Tests of cognitive ability are not necessary either unless there is indication of learning problems. Completing questionnaires provides a good opportunity to observe the patient in a challenging situation, even though patients are often able to suppress tics for a certain period so that the true extent of the symptoms may not be observed.

Differential diagnosis and comorbidity

Specific circumstances may cause variation of tic symptoms. Emotional states such as fear, joy or tension frequently lead to an increase. Distractions, an occupation requiring high concentration and consumption of cannabis or alcohol

<table>
<thead>
<tr>
<th>Aspect of tics</th>
<th>Disorder – differential diagnoses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoccupation with tic control</td>
<td>Attention problem</td>
</tr>
<tr>
<td>Tic repetition</td>
<td>Obsessive-compulsive phenomena</td>
</tr>
<tr>
<td>“Exaggerated” tic</td>
<td>Psychogenic origin</td>
</tr>
<tr>
<td>Monotonous tic</td>
<td>Stereotypy</td>
</tr>
<tr>
<td>Eye rolling</td>
<td>Absence</td>
</tr>
<tr>
<td>Rapid shuffling steps</td>
<td>Akathisia, juvenile Parkinson, compulsion</td>
</tr>
<tr>
<td>Distortions and similar²</td>
<td>Dystonia/Dyskinesia</td>
</tr>
<tr>
<td>Convulsive grimacing</td>
<td>Blepharospasm</td>
</tr>
<tr>
<td>“Jerk” tics</td>
<td>Chorea</td>
</tr>
<tr>
<td>“Shuddering” tics</td>
<td>Myoclonus</td>
</tr>
<tr>
<td>Tics during sleep</td>
<td>Restless legs, epilepsy, parasomnias</td>
</tr>
</tbody>
</table>

1This could be a movement or sound performed in an over-emphasized manner, therefore lacking the suddenness or uncontrollability of a typical tic.
2This would involve movements of a more coiling or writhing nature.
may lead to a decrease. Tics hardly ever interfere with intentional movements such as cycling. It is possible for tics to appear during any of the sleep stages, albeit with reduced frequency, intensity and complexity. Due to the aforementioned characteristics, it is possible to differentiate tic symptoms from most other movement disorders (see table H.2.3).

Approximately 65% of children and adolescents with chronic motor or vocal tic disorder have a comorbid condition (Conelea et al, in press). Around 90% of those with TS develop one or more psychiatric disorders (Freeman, 2007). The probability of having a comorbid disorder increases with the severity of tics, early onset and familial loading.

**TREATMENT**

**Psychoeducation**

Psychoeducation, involving the patient as well as relevant caregivers, should be provided at the beginning of treatment. Further, individual causal factors and options for treatment should be discussed. Referral to self-help groups is also useful. Treatment is usually delivered as an out-patient; inpatient treatment can be necessary in severe cases requiring more extensive investigations, when severe comorbid disorders are present or to monitor and achieve optimal drug treatment. Figure H.2.2 shows a decision tree (Roessner et al, 2011).

Psychoeducation involves providing detailed information to the relevant persons, in the case of young people this usually will involve parents and teachers. Information should be provided regarding the disorder, its course, investigations and options for treatment. Information which often is of use to teachers involves recommending allowing the child to sit exams on their own or to be permitted to leave the classroom for short periods – to lessen the urge to release the tics.

In cases of mild severity – taking into account the high rate of spontaneous remission – psychoeducation is all that is required. For this reason it makes sense to adopt a “wait-and-see” approach, keeping a watchful eye on recurrences or possible comorbid disorders (Wanderer et al, 2012).

**Psychotherapy**

Cognitive behavioral methods are the most effective psychotherapeutic intervention. This treatment should be administered by trained professionals well
Figure H.2.2 Decision tree for the treatment of tic disorders including Tourette syndrome (Roessner et al, 2011).

DBS: deep brain stimulation
THC: Tetrahydrocannabinol
versed in the complexities of the disorder. It may involve the following (Verdellen et al, 2011):

- For well-motivated and insightful patients, habit reversal training has been shown to be effective. Training comprises a set of techniques intended to help patients become aware of impending tics and practicing a competing response to inhibit or interrupt the tic. These techniques include relaxation training, contingency management and generalization training. To heighten tic awareness, methods such as:
  - Response description (patients learn to describe the topography of their tics and develop a detailed, usually written, description of each tic)
  - Response detection (patients receive feedback concerning the occurrence of a tic, until they can detect the target behavior unassisted)
  - Early warning procedures (patients practice identifying the early signs of a tic, such as specific urges, sensations or thoughts) and
  - Situation awareness (patients describe people, places or situations in which the tics occur most commonly) are included in the training.
  - Competing response training involves the patient learning to purposefully initiate a tic for one to three minutes or until the urge to perform the tic has disappeared.

- Exposure and response prevention is based on the association of a premonitory urge followed by the vocal or motor tic, which results in the relief of the urge sensation. The aim is to break the association between the urge and the resultant tic, which – according to learning theory – has been strengthened over time. By confronting the patient with the premonitory urge over a longer period (exposure) and having them resist giving in to it (response prevention), the patients would learn to endure the urge without having to act out the tic (habituation). Patients are also encouraged to self-monitor symptoms by recording times and situations in which tics occur over certain periods to ascertain when and where tics are most frequent.

- Massed (negative) practice involves intentionally and repeatedly acting out the tic in an effortful and rapid way over a certain period with short rests in between. The long-term effectiveness of this seems limited but can help patients if they are about to enter situations which require tic-free appearance (i.e., going to the cinema).

- Relaxation training is assumed to help reduce tics because tic intensity often increases in times of stress and anxiety. Relaxation training includes progressive muscle relaxation, imagery, autogenic training or deep breathing and is mostly applied as one part of a multi-modal treatment plan. In terms of effectiveness, Peterson and Azrin (1992) found that tics were reduced by 32% with relaxation training, by 55% with habit reversal training and by 44% with self-monitoring techniques.
• **Contingency management**, again as a part of a multi-modal program, seeks to positively reinforce tic-free intervals (e.g., through tokens, praise or demonstration of affection) and to ignore tics. According to learning theory this should result in a lessening of the behavior (in this case, the occurrence of tics). Because this method is usually incorporated in multi-component treatment packages, it has been difficult to assess the value of this specific technique.

• Sometimes the presence of a tic disorder in a child may result in significant problems among family members. In such cases *family therapy* should be recommended.

**Medication**

There is good empirical evidence supporting the use of a variety of medications for Tourette syndrome (see below). However, medication for Tourette syndrome is often prescribed “off-label”. For instance, the only drug approved for tics in Germany is haloperidol, which on current evidence is considered only a medication of third choice. Pharmacological treatment is recommended when tics result in significant subjective discomfort, such as muscular pain or physical injury, ongoing social problems (e.g., isolation or bullying), emotional problems, or significant functional impediment, typically in academic performance (Roessner et al, 2011). The aim is to achieve the best balance between maximum benefit and minimum side effects. It is not to be expected that tics will disappear completely with medication; at best, symptoms will be alleviated.

Before initiating pharmacological treatment, the following investigations should be carried out: blood and liver function tests, prolactin levels, ECG, EEG, as well as physical and neurological examinations (to establish a baseline, exclude cardiac contraindications, including long QT syndromes, other physical illnesses or potential contraindications for the use of medication). Additionally, it is important to assess whether the tic disorder or another comorbid disorder is causing the greatest impairment, in order to determine which of the disorders should be primarily treated. For instance, treating comorbid ADHD can result in improved ability to suppress the tics without having to specifically treat them – conversely, psychostimulant drugs may rarely worsen the tics.

Generally speaking, medication should start slowly, with effectiveness and tolerability being assessed at regular intervals. Once optimal dosage has been ascertained, medication should be taken regularly for at least one year before considering a discontinuation. At the very latest, medication should be reduced in late adolescence to ascertain whether continuation is necessary, taking into account the high rates of spontaneous remission. Table H.2.5 summarizes the European recommendations about medication for tic disorders.

• **Haloperidol** is the only medication officially approved for the treatment of tic disorders in Europe (from the age of three years). Haloperidol has strong antidopaminergic action and results in a reduction of tics in approximately 80% of cases. However, adverse reactions such as extrapyramidal symptoms occur quite frequently, which makes haloperidol not the treatment of first choice.
### Table H.2.5 Medications with some evidence of effectiveness for the treatment of Tourette’s syndrome (Roessner et al, 2011)

<table>
<thead>
<tr>
<th>Medication</th>
<th>Level of evidence</th>
<th>Starting dosage (mg)</th>
<th>Therapeutic range (mg)</th>
<th>Common adverse effects</th>
<th>Investigations at start and during follow up</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alpha-adrenergic agonists</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clonidine</td>
<td>A</td>
<td>0.05</td>
<td>0.1-0.3</td>
<td>• Orthostatic hypotension • Sedation, sleepiness</td>
<td>Blood pressure</td>
</tr>
<tr>
<td>Guanfacine</td>
<td>A</td>
<td>0.5-1.0</td>
<td>1.0-4.0</td>
<td></td>
<td>ECG</td>
</tr>
<tr>
<td><strong>First generation antipsychotics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Haloperidol</td>
<td>A</td>
<td>0.25-0.5</td>
<td>0.25-15.0</td>
<td>• EPS • Sedation • Increased appetite and weight</td>
<td>Blood count, ECG, Weight, Transaminases, Neurologic status, Prolactine</td>
</tr>
<tr>
<td>Pimozide</td>
<td>A</td>
<td>0.25-0.5</td>
<td>0.25-15.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Second generation antipsychotics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aripiprazole</td>
<td>C</td>
<td>2.50</td>
<td>2.5-30</td>
<td>• Sedation • Akathisia • EPS • Headache • Increased appetite, weight • Orthostatic hypotension</td>
<td>Blood count, Blood pressure, Weight, ECG, Transaminases, Blood sugar and lipids</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>C</td>
<td>100-150</td>
<td>100-600</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quetapine</td>
<td>C</td>
<td>100-150</td>
<td>100-600</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Risperidone</td>
<td>A</td>
<td>0.25</td>
<td>0.25-6.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ziprasidone</td>
<td>A</td>
<td>5.0-10.0</td>
<td>5.0-10.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Benzamides</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sulpiride</td>
<td>B</td>
<td>50-100 (2mg/kg)</td>
<td>2-10 mg/kg</td>
<td>• Problems with sleep • Agitation • Increased appetite</td>
<td>Blood count, ECG, Weight, Transaminases, Prolactine, Electrolytes</td>
</tr>
<tr>
<td>Tiapride</td>
<td>B</td>
<td>50-100 (2mg/kg)</td>
<td>2-10 mg/kg</td>
<td>• Sedation • Increased appetite</td>
<td></td>
</tr>
</tbody>
</table>

EPS: extrapyramidal symptoms. Evidence level: A, >2 randomized controlled trials; B, 1 randomized controlled trial; C, anecdotal (case studies, open trials).
• **Tiapride** is a selective D2 receptor antagonist that shows virtually no antipsychotic action. It has a good side effect profile and is well tolerated with few extrapyramidal symptoms. The most frequent adverse reactions are drowsiness, moderate transient hyperprolactinemia and weight gain. There is no evidence of negative effects on children’s cognitive performance. Tiapride is currently the first-choice medication for treatment of Tourette syndrome in Germany.

• **Risperidone** is a second generation antipsychotic agent with a high affinity for D2 and 5-HT2 receptors. Efficacy is similar to that of haloperidol but with a more favorable side effect profile (see Table H.2.5).

• **Aripiprazole** has shown promising effects specifically in patients who had not responded to, or had not tolerated well other medications. Aripiprazole has a high affinity for D2 receptors but, in contrast to other second generation antipsychotics, it is also a partial agonist of 5HT1A receptors and a potent antagonist at 5HT2A receptors. This profile has raised hopes that it may be better than the other drugs (Roessner, 2011). It also has the advantage that weight gain is low. Nausea and sedation are the most frequently reported adverse effects.

Although the best evidence from clinical trials is still that for the typical antipsychotics haloperidol and pimozide, European clinical practice has gradually substituted these substances with atypical antipsychotics, foremost risperidone. Availability of and experience with specific medications, however, also plays a role in the choice. In the German-speaking world, tiapride is regarded as the first choice drug in the treatment of tic disorders in children and adolescents (Rothenberger et al, 2007). Although evidence is comparatively limited, Roberston and Stern (2000) also recommend tiapride as well as sulphiride in their review of treatment for tic disorders, due to their balance of efficacy and tolerability. Aripiprazole has shown potential regarding efficacy in refractory cases. There is, however, a lack of placebo-controlled studies for this medication.

**Treating Tourette syndrome comorbid with other disorders**

When there are comorbid psychiatric disorders and Tourette syndrome, it should always be ascertained which of the presenting conditions is causing the greater impairment. Treating one disorder frequently has a positive effect on the other and may render further specific treatment unnecessary.

**Attention deficit hyperactivity disorder (ADHD)**

If ADHD is comorbid with a tic disorder, the former may be treated with psychostimulants, such as methylphenidate. There has been a longstanding argument about whether psychostimulants may cause, trigger or worsen tics in these cases and, therefore, whether they should be contraindicated in these circumstances. Recent studies have shown that in most cases psychostimulants do not lead to an exacerbation of tics (Pringsheim & Steeves, 2011). Treating patients with atomoxetine or clonidine has proven effective as long as the Tourette syndrome is only of mild to moderate severity (Roessner et al, 2011). These two medications mainly reduce ADHD symptoms only having a marginal effect on
tics. Should the aforementioned medication have little effect on tics, the addition of risperidone can be considered. Alternatively, ADHD symptoms may be treated with methylphenidate, which can be combined with risperidone if necessary.

**Emotional disorders**

In cases presenting with mild to moderate depressive or anxious symptoms comorbid with Tourette syndrome, pharmacological treatment solely with sulpiride can be considered. Sulpiride has positive effects on tics as well as the comorbid emotional problems with few extrapyramidal symptoms and vegetative adverse reactions (Roessner et al, 2011), with the exception of galactorrhea.

Another option for treating Tourette syndrome with comorbid depression or obsessive-compulsive disorder is using a selective serotonin reuptake-inhibitor (SSRI). Antipsychotic drugs can be prescribed in combination with SSRIs in cases of moderate to severe tic symptoms.

**Alternative medicine treatments**

Substantial anecdotal evidence exists concerning the benefits of physical activity (rhythmic sports, such as swimming) and recreational activities in general. Patients should be informed and encouraged accordingly. There is no evidence that diet, vitamins or mineral supplements as well as hypnosis are of benefit and should not be recommended.

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**REFERENCES**


