

Childhood Headaches: A Clinical Review

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Headache is a common complaint at all ages. It is only within recent decades that the significance of pediatric headache has been fully recognized.

In 1962 Bille¹ investigated the incidence of headaches in 9,000 school aged children in Sweden. It was estimated that by age six, 39 percent of children had suffered from headache. This figure dramatically increased by age 15 to 70 percent. Other studies have reported similar trends with an incidence as high as 82 percent².

Headaches significantly impact the lives of children and adolescents. They result in school absence, decreased extracurricular activities, and may contribute to poor academic achievement³. Headaches account for a large number of health care office visits each year. Despite the prevalence and consequences of childhood headaches, children continue to receive less treatment than adults who suffer from headaches.

To properly manage childhood headache, the care provider must understand common headache patterns as well as the signs and symptoms that may indicate serious intracranial disease. Treatment of pediatric headaches is complicated by unanswered questions regarding the safety and efficacy of adapting adult pharmacological therapy to the diverse pediatric population.

Classifying Pediatric Headaches

While no uniform criteria exist for classifying pediatric headaches, in 1988 the International Headache Society published diagnostic criteria and a classification scheme for headache disorders⁴. These guidelines are lengthy and have been criticized for their poor applicability to children. Despite this, it is beneficial to group headaches into two general

categories to facilitate proper evaluation and treatment. Thus, headaches can be divided into *benign* and *structural* based on etiology.

Structural headaches are associated with underlying CNS pathology. Benign headaches include migraine, tension headache and chronic daily headache.

Migraine headaches, which constitute the majority of benign childhood headaches, are recurrent episodes of head pain, classically considered pulsatile and unilateral in nature and often accompanied by nausea, vomiting and photophobia⁴. They tend to occur abruptly and last several hours. The pain is often relieved by sleep. In 1976, Prensky⁵ proposed criteria for diagnosing pediatric migraine that require three of the following six: aura (visual, sensory or motor), unilateral head pain, throbbing pulsatile pain, nausea, vomiting or abdominal pain, relief after sleep and a positive family history of migraine. In practice however, pediatric migraines are often bilateral and clear localization of the pain can be difficult to obtain from children.

Gladstein proposed criteria that omits unilateral and throbbing pain requiring two of four of the following: aura, positive family history, relief with sleep and nausea, vomiting or autonomic symptoms⁶.

Migraine headaches can be subdivided based on the presence or absence of an aura and/or association with neurologic complications⁷. Migraine with aura or "classic migraine" are seen in approximately 14-30 percent of children with migraine. Typical auras are reported as spots, colors, image distortions or visual scotoma. Auras usually have a gradual onset and last for several minutes before the headaches onset.

Migraine without aura or "common migraine" constitute the majority of all childhood migraines and appear to have a

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strong familial tendency. They are often described with bilateral pain and frequently preceded by a behavioral prodrome with mood changes or withdrawal from activity.

Complicated migraines are headaches accompanied or manifest by transient neurologic symptoms. These symptoms may occur immediately before, during or after the headache. In some situations the headache may be mild or nonexistent.

Hemisindrome (*hemiplegic*) migraine, ophthalmoplegic migraine and basilar artery migraine are typical examples of complicated migraine. Hemiplegic migraine, while unusual, are seen more commonly seen in children than in adults. They are characterized by an abrupt onset of hemiparesis that is usually followed by a headache. Hemianesthesia may also precede a headache.

Ophthalmoplegic migraine may occur at any age, usually with orbital or periorbital pain as well as third, fourth or sixth cranial nerve involvement. The pain disappears in hours but the ophthalmoplegia may last for days. Basilar artery migraines are more common in girls than in boys. They are characterized by dizziness, weakness, ataxia and a severe occipital headache with vomiting. Less common disorders of complicated migraine have also been described in which head pain is not a prominent feature.

The *Alice in Wonderland* syndrome is characterized by distortions of vision, space and/or time. Patients may note micropsia and/or metamorphopsia as well as other sensory hallucinations. Confusional migraine in juvenile patients is characterized by impairment of sensorium, agitation, lethargy sometimes progressing to stupor.

In addition to these complicated migraines, several migraine variants also have been recognized. Benign paroxysmal torticollis of infancy and benign paroxys-

mal vertigo of childhood are characterized by recurrent episodes of either a head tilt and/or vertigo and ataxia. The torticollis typically occurs during the first year while the vertigo occurs in young children, usually two to three years of age. Cyclic vomiting and recurrent abdominal pain also are frequently considered migraine variants. Before diagnosing either of these entities however, thorough investigations and thought need to be given to the possibility of a gastrointestinal etiology.

Tension headaches or muscle contraction headaches are benign and often described as a band-like feeling around the head or occasionally pain in the neck and/or shoulders². These headache can last for days and may be associated with stressful events at home or school. Sleep may temporarily relieve the headache.

Correct Diagnosis

While the minority of headaches in children are due to serious underlying pathology, early recognition is paramount for appropriate diagnosis and management. Structural headaches are frequently caused by space occupying lesions, inflammation and/or an increase in intracranial pressure. Neurosurgical measures will frequently need to be undertaken for treatment. While no one sign or symptom indicates a structural etiology, there are several signs and symptoms that warrant further consideration.

Any headache that is worse in the morning and improves as the day progresses or any headache aggravated by sneezing, coughing or straining may be due to increased intracranial pressure. Headaches persistently localized to the occipital region warrant attention. Consideration also needs to be given when any focal neurological signs or symptoms present without headache. Worsening of

headache severity and/or frequency especially with rapid progression may also suggest an intracranial pathologic process⁸. Any significant change in a previously diagnosed headache should be carefully reevaluated. Headaches, which fail an adequate trial of therapy, may imply improper diagnosis.

In addition to classifying headaches based on their symptoms, they also can be classified by their temporal pattern. Common temporal patterns include: acute, acute recurrent, chronic nonprogressive, and chronic progressive.

Acute headache is defined as a recent onset of headache with no prior history of similar episodes. It is important to establish if any neurologic symptoms accompany this headache. The differential of acute headache includes systemic infection, trauma, central nervous system infectious processes or first episode of migraine. Reoccurrence of similar headaches up to several times a month with symptom free intervals in between are classified as acute recurrent headaches with the common diagnosis of migraine.

Chronic nonprogressive headaches differ from acute recurrent headaches predominantly by their greater frequency and persistence for years with no associated neurologic symptoms or change in headache severity. Chronic nonprogressive headaches may have emotional or behavioral components. A common headache in this category is tension headache.

An important newly recognized diagnostic entity that also occurs with this temporal pattern is chronic daily headache (CDH). Chronic daily headache was first described in adults who reported daily or nearly daily headaches. It was soon recognized that although patients were similar in the number of headaches experienced, the description of their

Table 1**Factors Precipitating Migraine****Common factors**

Stress/Anxiety
 Menstruation
 Oral Contraceptives
 Physical Exertion/Fatigue
 Lack of Sleep
 Glare
 Hunger
 Foods/Beverages with: Nitrates, glutamate, caffeine, tyramine, salt

Less Common Factors

Reading/Refractive error
 Cold foods
 High altitude
 Drugs: Nitroglycerin, Indocin, Hydralazine

headaches fell on a continuum between migraine and tension⁹. Recent work has demonstrated a similar spectrum in children. The most common CDH pattern appears to be what is termed comorbid where migraines are superimposed on a background pattern of frequent tension headaches¹⁰.

Chronic progressive headaches also occur at least several times a week, but unlike the nonprogressive variety, these headaches increase in frequency and/or severity with time. As discussed above, due to the changing headache pattern, the care provider needs to be concerned that these headaches are secondary to a structural etiology.

Imaging

While it is not necessary to image every child with the complaint of headache, any suspicion or concern that the headache may have a structural etiology should be further evaluated with neuroimaging. Given the broad differential of structural headaches and the imaging choices available, many practitioners may be uncertain as to which scans will yield the most information in a cost-effective manner. Magnetic resonance imaging (MRI) is generally more costly, takes longer and may require sedation, but its superior imaging capabilities offers the advantage of detailed structural definition. The use of gadolinium enhances MR sensitivity to vascular lesions and those that disrupt the blood-brain barrier. A CT brain scan with contrast can define most structural lesions. A CT scan without contrast is somewhat more limited in its sensitivity although can easily define hydrocephalus and hemorrhage.

All patients presenting with any features of a structural headache should undergo high quality imaging, preferably an MRI with gadolinium enhancement, although routine MRI or CT with contrast is acceptable. In less suspicious clinical situations or for parental or patient

reassurance, routine MRI or high quality CT with contrast is sufficient. Routine non-contrast CT should be reserved for more acute situations where time is crucial and intracranial hemorrhage is suspected.

Treatment of Benign Headache

The first step in the treatment of benign childhood headaches should begin with the reassurance that the headache is not due to a brain tumor or some other central nervous system pathology. Reviewing with the parents and patient the headache pattern, associated symptoms such as nausea, dizziness, photophobia and the benign nature of the physical examination, including funduscopy, often reassures them that the headache process is not of a progressive nature.

If an imaging study has been performed, this information can also be very reassuring to the family. This simple but crucial review will help to alleviate stress and worry that is likely to be contributing to the patient's symptoms and parent's anxiety. Realizing that their pain, although unpleasant, is not life threatening often

allows the patient and parents to apply healthier coping strategies.

The role of sleep, darkness and a quiet room are essential in the management strategy for acute migraine and tension headache. Stress both as an etiology and as a consequence of headache makes it a logical target for nonpharmacologic therapy. Encourage scheduled time for meals, bedtime, relaxation and exercise. Relaxation techniques and biofeedback may prove beneficial. Individual treatment decisions should be based on the age of the child and receptiveness to behavioral techniques but have been highly effective for both migraine and tension-type headaches.

Psychotherapy is indicated for any patient under significant stress. Family therapy should be considered in situations involving divorce, illness of a sibling or in any situation where the family unit is contributing to the child's medical problem.

Eliminate identified precipitants (Table 1). Alcohol, drugs or caffeine may trigger headaches and appropriate lifestyle changes should be encouraged. The role of diet in headaches continues to remain

Table 2

Pharmacologic Treatment of Migraine	
Analgesics/Anti-inflammatory	
	Tylenol
	Ibuprofen
	Naproxen sodium
Abortive	
	Sumatriptan/Naratriptan
	Ergots
	Isometheptane (Midrin)
Prophylactic	
	beta-Blockers (propranolol, nadolol)
	Tricyclics (amitriptyline, nortriptyline)
	Cyproheptadine
	Antiepileptic drugs (valproic acid, phenobarbital, phenytoin)
	Verapamil

controversial. however if a given food or beverage is associated with headaches in an individual, avoidance can have a significant impact.

Pharmacological Treatment of Migraine

Pharmacologic treatment for migraine can be divided into symptomatic, abortive and prophylactic therapies⁷ (Table 2). The symptomatic treatment of migraine relies mainly on analgesic/anti-inflammatory medications. Tylenol (10-15 mg/kg/dose q4h) and Ibuprofen (5-10 mg/kg/dose q6h) are commonly used and available over the counter. Naproxen sodium may be somewhat more effective but overuse can lead to gastrointestinal side effects.

Abortive therapy consists of medications used to interrupt a headache after its onset. Sumatriptan (Imitrex), a 5-HT₁ receptor agonist, causes vasoconstriction and is a very effective drug in aborting migraine with an efficacy approaching 70 percent in adults. It is available as an oral tablet, nasal inhalant, a subcutaneous

patient autoinjection system and in vials for injection. The injected administration of sumatriptan is more effective than the oral.

Side effects of sumatriptan include tingling, dizziness, warm sensations, chest pain and cardiac arrhythmias. Sumatriptan is absolutely contraindicated with cardiac disease, hypertension and pregnancy¹¹. Recommended adult doses are 25-100 mg orally and 6 mg subcutaneous. Both routes may be repeated after two hours. Maximum daily adult oral dose should not to exceed 300 mg. A new nasal spray delivery system is now available that delivers 5mg, 10mg or 20mg intranasal with each spray. Efficacy appears to be somewhat superior to the oral route but less effective than injection.

In children 12-17 years, controlled clinical trials with oral sumatriptan has failed to show efficacy. Subcutaneous sumatriptan has been effective although is currently not recommended by the manufacturer for use in patients under 18 years. In children, a trial subcutaneous dose is 0.1 mg/kg/dose. Current

autoinjection sumatriptan is only available in 6mg unit doses.

Recently a new 5-HT receptor agonist, naratriptan, has been released for use as an oral tablet. While its onset of action is slower than sumatriptan, due to its longer half-life its duration of effect is longer thus reducing the need for repeated doses. Extensive trials have not been performed in children with naratriptan.

Isometheptane (Midrin) and ergots are also available for abortive therapy for migraines⁷. They are most effective when administered with an aura or the initial onset of the headache. Since auras are less common in pediatric migraine and children may be less able to communicate early symptoms of a headache, administering these abortive therapies at the appropriate time can be difficult. Ergots are generally not used for young children (under six years) and may cause gastrointestinal upset. There is also a risk of rebound headache with excessive use in patients with frequent headaches.

Prophylactic therapy should be considered when headaches are frequent enough to interfere with the patient's lifestyle. The decision to begin a child on prophylactic therapy requires consideration of the risks of long term drug use, balanced against the benefit of potential headache relief. As with abortive therapy, several classes of pharmacologic agents are available (Table 2).

Beta-blockers are commonly used as prophylactic therapy for childhood migraine¹². Both propranolol and nadolol are effective. Nadolol has the advantage of being longer acting and can be given once daily. Beta-blockers are contraindicated in patients with asthma, diabetes and may cause depression in adolescents. Tricyclic agents (amitriptyline, nortriptyline) are also frequently used for prophylactic therapy especially in older children and adoles-

cents. Nortriptyline tends to be less sedating than amitriptyline.

Cyproheptadine (Periactin) an antihistamine/antiserotonin drug also is effective in preventing migraine, especially in younger children⁷. Side effects of cyproheptadine include sedation, appetite stimulation and weight gain. Anticonvulsant medications such as valproic acid, phenobarbital and phenytoin, have also been used as prophylactic agents with reasonable success. These agents are especially useful when a seizure disorder coexists with migraines. Finally, calcium channel blockers (verapamil) have been used especially in adults for migraine prophylaxis but their efficacy in the pediatric population is variable.

In general, the effect of prophylactic therapy is not immediate. It can often take up to two weeks before the patient experiences improvement. Providing this information to the patient and parent leads to improved compliance and more realistic goals. It is important to give an appropriate trial before attempting a new treatment.

Migraines are known to spontaneously remit during childhood. The need for continued prophylaxis should be reassessed at six month to one year intervals. This can be achieved by tapering the medication until either the headaches resume or the patient remains headache-free off of therapy.

Pharmacologic Therapy for Tension and Chronic Headaches

Acute tension headaches often are responsive to symptomatic treatments. Sleep, darkness or a quiet room may relieve some tension headaches as well as acute migraine. Ibuprofen and acetami-

nophen are commonly recommended for relief of headache pain. Patients who are prone to tension headaches should attempt to minimize stress and may benefit from behavioral/relaxation therapy. Narcotics and other potentially addictive medications should be avoided.

The treatment of the chronic daily headache (CDH) combines therapies used for tension and migraine. It is recommended that the patient discontinue the use of over-the-counter analgesics and all narcotics. Chronic intermittent analgesic use may result in rebound headaches.

Tricyclic antidepressants appear to be most helpful in treating CDH in children. Psychological, behavioral and relaxation interventions are also beneficial for this population and should be considered along with tricyclics. When the CDH pattern includes well defined migraine attacks, abortive therapy may provide symptomatic relief.

Conclusion

Physician awareness is the key to properly diagnosing and treating pediatric headache. Recognition that pediatric headaches can be divided into benign and structural groups is crucial in the approach and treatment of childhood headaches. With recent technological and pharmacological advances, this common pediatric complaint can be effectively addressed in the majority of patients.

The primary care physician can manage a significant proportion of pediatric headaches and reserve referral for complicated headache patterns, headaches refractory to treatment and any headache suspicious for a structural etiology.

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Tic Disorders in Childhood: Physician Awareness is Key



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Tic disorders are a common occurrence in school-age children and constitute the most common movement disorder brought to the attention of the care provider by families and teachers. The spectrum and severity range from mild, transient simple motor tics to the potentially devastating Tourette Syndrome.

Tics are defined as sudden, rapid, brief, repetitive and purposeless involuntary stereotyped movements or utterances. They frequently are referred to as "habit spasms" or "nervous twitches." An appreciation and understanding of the spectrum as well as the associated disorders allows care providers to counsel parents and to treat the child appropriately when indicated.

(imitating other people's movements) and copropraxia (obscene gestures).

Similarly, simple vocal tics consist of sniffing, snorting, coughing, grunting, barking, throat clearing, clicking, squeaking and hissing. Complex vocal tics consist of echolalia (repeating other people's words), palilalia (repeating one's own words), and most distressing, coprolalia. There is actually a third type of tic called sensory tics. These include an uncomfortable sensation such as a tickle, irritation, temperature change or unusual feeling causing the patient to produce a voluntary movement or sound. Although uncommon in children, they have been reported in up to 40 percent of adult Tourette patients².

In general, tics have several common characteristics to assist in their diagnosis. Tics typically wax and wane and frequently occur less in an office setting than by history. In this situation a video diary supplied by the family can be helpful to the physician.

Tics frequently increase with anxiety, emotional stress, anger, excitement and fatigue. While they may occur during sleep, they are usually less noticeable and they decrease with relaxation. They also frequently attenuate during absorbing activities. Tics are briefly suppressible, although attempts to do so frequently result in an "inner (emotional) tension," which is relieved by "releasing" the tic. Despite these facts, it is important to recognize that tics are an involuntary movement and are not caused by stress.

Once the diagnosis of tics has been made, an attempt to classify the disorder must be undertaken. This approach divides tic disorders into three groups (Table 2):

- "transient" (lasting less than one year)
- "chronic" (lasting more than 1 year)
- "nonspecific"

Transient tic disorder is the mildest and most common tic disorder occurring in up to 25 percent of school children³. Transient tics are simple tics and usually of a motor

Table 1
Tic Classification

	Simple	Complex
Motor	Eye blinking Head twitching Head thrusting Shoulder shrugging Mouth opening	Facial grimacing Touching Smelling Jumping Echokinesis Copropraxia
Vocal	Sniffing Snorting Coughing Throat clearing Grunting Barking	Echolalia Palilalia Coprolalia

Tic Classification

Tics are typically classified as either motor or vocal. Both motor and vocal tics can be either "simple" or "complex"¹ (Table 1). The most common simple motor tics are repetitive eye blinking, head twitching or thrusting, shoulder shrugging, and mouth opening. Complex motor tics consist of motor actions such as facial grimacing, touching, smelling, jumping, echokinesis

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Table 2
Tic Syndrome Classification

- I. Transient Tic Disorder
(duration < 1 year)
- II. Chronic Tic Disorder
(duration > 1 year)
 - A) Chronic Single Tics
 - B) Chronic Multiple Motor or
Vocal Tics
 - C) Tourette Syndrome
- III. Nonspecific Tic Disorder

variety, although transient vocal tics can occur. Due to their mild and benign nature, treatment is not necessary or recommended.

Chronic tic disorders include three subtypes: chronic single tic disorder, chronic multiple motor or vocal tic disorder and Tourette syndrome. Chronic single tic disorder requires the occurrence of only a single motor, or less commonly, vocal tic for more than one year. Chronic multiple tic disorder (CMTD) consists of multiple tics, either all motor or less frequently, all vocal, but not both, occurring over one year.

Several studies have shown that CMTD tends to be a mild form of Tourette syndrome and that both are transmitted as inherited traits in the same families ⁴.

Tourette syndrome (TS) is a chronic, complex, fluctuating tic disorder of variable severity characterized by both motor and vocal tics of both simple and complex types. Current essential diagnostic criteria for the diagnosis of Tourette syndrome include (Table 3):

- onset before age 21 years,
 - multiple motor tics,
 - one or more vocal tics,
 - a waxing and waning course,
 - the gradual replacement of old tics with new ones,
 - the absence of other medical explanations for tics, and
 - duration for more than one year.
- Tourette syndrome is more common in

males than females (3:1). The mean age of onset is between 6 to 7 years of age with most patients presenting before age 13 years. Eye blinking, facial grimacing or head twitches are the most common initial tics.

Although Tourette syndrome has been thought to be a life-long disorder, in 30 to 40 percent of children with TS all tic symptoms will disappear by late adolescence and an additional 30 percent will experience a marked improvement. The remaining patients will have symptoms persist into adulthood ⁵. At present there are no reliable prognostic characteristics that permit identification of those patients who will have spontaneous improvement in their symptoms. Remission appears to be independent of the use of medications.

Nonspecific tic disorder is a category for children who do not meet the criteria for a specific tic disorder. Included in this group are children who develop tics in response to medications such as stimulants or neuroleptic withdrawal and those who have onset of their tics following an acute insult such as herpes encephalitis. The recently described PANDAS (Pediatric Autoimmune Neurologic Disorders Associated with Streptococcal infection) may also be in this category ⁶.

Comorbid problems

A variety of comorbid behavioral problems are identified in children with Tourette syndrome ⁷. The two most prevalent and often problematic are obsessive-compulsive disorder and attention-deficit hyperactivity disorder. Additionally, learning difficulties, speech and language disorders and sleep problems have commonly been observed.

Common compulsive symptoms include ordering and arranging habits, checking rituals, frequent counting, and rituals to decontaminate objects or body parts such as hand washing. Obsessive-compulsive behavior has been reported in more than 40 percent of children with Tourette's ⁸. Attention-deficit hyperactivity disorder has

been noted in 50 percent of children with Tourette's and may precede the onset of the tics.

Determining impact

Determining the major source of difficulty is essential before any specific recommendations and treatment can be instituted. Tics rarely cause significant physical discomfort or damage, however their adverse effect on psychosocial development and interpersonal relationships can be immense.

It is essential for the care provider to inquire not only about the number, intensity and frequency of the tics, but also to question the degree of impairment and impact they have on the child's activities and social interactions. It must also be determined whether there are any associated problems such as attention-deficit hyperactivity disorder, obsessive-compulsive disorder or behavioral difficulties.

After a complete assessment the care provider should educate the patient and family about the disorder and define the target symptoms that need to be addressed. Just because a symptom is present does not mean it requires pharmacotherapy.

Table 3
Tourette Syndrome Criteria

1. Onset prior to 21 years of age
2. Presence of multiple types of motor tics
3. Presence of one or more type of vocal tic
4. A waxing and waning course
5. Gradual replacement of old tics with new ones
6. Absence of other medical explanation for tics
7. Duration for more than 1 year

Treatment Options

The pharmacologic treatment of tic disorders is strictly symptomatic and not curative. Treatment should be reserved for those children with significant tics causing psychosocial or functionally disabling problems. The goal is to reduce the tics to a tolerable level, realizing that eradication may not be possible. While alternative nonpharmacologic treatments have been used, including conditioning techniques, relaxation, biofeedback and hypnosis, pharmacotherapy is most frequently employed.

At present there are two main classes of medications to suppress tics: alpha-adrenergic agonists and neuroleptics (Table 4). Other agents including benzodiazepines, calcium channel blockers, catecholamine-depleting agents and opiate

Table 4
Treatment of Tics and Comorbid Problems

Tics

Alpha-Adrenergic agonists

Clonidine

Guanfacine (Tenex)

Neuroleptics

Pimozide (Orap)

Fluphenazine (Prolixin)

Haloperidol (Haldol)

Attention-Deficit Hyperactivity Disorder

Behavioral and educational interventions

Pharmacologic treatment

Desipramine

Alpha-Adrenergic agonists

Clonidine

Guanfacine (Tenex)

Central stimulants

Ritalin

Dexedrine

Pemoline (Cylert)

Obsessive-Compulsive Disorder

Fluoxetine (Prozac)

Clomipramine (Anafranil)

antagonists have been used with variable success. Clonidine is effective in treating tics in approximately 50 percent to a somewhat overly optimistic 70 percent of patients with Tourette's⁹.

While its efficacy is less than the neuroleptics (80 percent), it is frequently the initial medication prescribed due to a relatively benign side-effect profile. It can be administered as a tablet or trans-dermal patch. An alternative alpha-adrenergic agonists, guanfacine (Tenex), may be tried if behavioral problems are prominent. Neuroleptics are the most effective tic-suppressing agents although their side-effect profile may limit their use. The most commonly used agents include: haloperidol, pimozide and fluphenazine. They all appear to be approximately equipotent in suppressing the tics.

In children, generally pimozide and fluphenazine are used before haloperidol because they have a slightly lower incidence of side-effects and are tolerated somewhat better.

Treatment of any comorbid problems needs to be undertaken with careful consideration to the total patient care. The approach to the attention-deficit hyperactive disorder is somewhat controversial because psychostimulant medications may provoke or intensify tics. If behavioral and educational approaches fail, desipramine and alpha-adrenergic agonists may be tried. If necessary a brief trial of central stimulants can be undertaken with careful attention to tic exacerbation.

In Tourette patients with disabling obsessive-compulsive disorder, additional psychiatric intervention and behavioral modification techniques should be considered. Two psychotropic drugs, clomipramine and fluoxetine, have been helpful in treating severe obsessive-compulsive manifestations.

Conclusion

Physician awareness is the key to properly diagnosing and treating pediatric

tic disorders. The spectrum is wide, ranging from benign transient simple motor tics to Tourette syndrome. Recognition of this spectrum and familiarity with tic classification and tic syndrome classification allows the care provider to properly educate the family and ultimately determine whether the major source of distress is related directly to tics or associated behaviors.

Once this has been done, then an individualized multimodal treatment plan can be developed. A valuable resource for additional information for care providers, parents and patients is the nonprofit voluntary Tourette Syndrome Association, 42-40 Bell Blvd., Bayside, New York, 11361-2874; (718) 224-2999; and on the Internet at tourette@ix.netcom.com.

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