10

Headaches During Childhood and Adolescence

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Headaches are common in children and teenagers. By 7 years of age, 40% of children have had headaches, with 2.5% having frequent nonmigraine types and 1.4% having migraine (1). By 15 years of age, 75% have had headaches: 15.7% with frequent tension type, 5.3% with migraines, and 54% with infrequent nonmigraine headaches.

The evaluation of the child with headaches is similar to the basic approach in adults, described in Chapter 1. A complete history is essential. Information from parents and other family members or caregivers, especially for younger children, is vital. In some adolescents, it is helpful to obtain the history from the child without family members present and then from family members. A psychosocial history is also important. For recurring or chronic headaches, what is the impact of the headaches on the child's life? Has school been missed, has homework gone undone, or have other activities been curtailed? Because of the frequent familial nature of migraine, family history is also important. When you ask the parents about their own headaches, often you will diagnose their migraines for the first time. A general physical and neurological examination is also necessary. General indications for diagnostic testing are discussed in Chapter 1: Table 1-8 focuses on indications in children and Table 1-9 gives general indications for neuroimaging.

This chapter covers the following topics: migraine, episodic tension-type headaches, chronic nonprogressive headaches, acute headaches, and chronic progressive headaches. Cluster headaches, the subject of Chapter 7, are not separately discussed because the onset usually occurs after 20 years of age and these headaches are rare in children. Some other causes of secondary headaches in children and adolescents, such as posttraumatic, ophthalmological causes, and pseudotumor cerebri, are also reviewed in other chapters.

MIGRAINE

Introduction

A variety of migraine types can occur in children and teenagers (2). Individuals may have just one type or they may have different types. In addition to obtaining the usual history, ask about possible triggers; it is also helpful to ask about a history of motion sickness or somnambulism (sleepwalking), both of which occur much more often in migraineurs. Motion sickness is reported by 45% of children with migraine and 5% of controls (3). Sleepwalking occurs in 28% of children with migraine and 5% of controls (4).

Table 10-1. The peak incidence of migraine with and without aura in males and females

Gender	Incidence (per 1000 person-years)	Peak age (years)
Male		
With aura	6.6	5
Without aura	10	10–11
Females		
With aura	14.1	12–13
Without aura	18.9	14–17

Epidemiology

The onset of migraine frequently occurs during childhood: 20% before 10 years of age and 45% before 20 years of age. Until puberty, the prevalence of migraine is the same in boys and girls. After puberty, the ratio of females to males is 3:1. The peak of new cases of migraine, the incidence, occurs during childhood (Table 10-1) (5). The onset of migraine with aura is earlier than that of migraine without aura. Migraine also begins earlier in males than in females. By 15 years of age, 5% of children have had migraines. Of these, about 1.5% have migraine with aura. The risk of a child developing migraine is 70% when both parents have migraine and 45% when one parent is affected.

Clinical Manifestations

Migraine without Aura

As in adults, migraine without aura is the most common type. However, childhood migraine can be somewhat different. The duration of headache is often much less and can last as little as 30 minutes. Pediatric migraine is more often bilateral, frontal, and temporal than unilateral (35% vs. 60% in adults). Finally, there is a higher incidence of light or noise sensitivity alone than is found in adults. Table 10-2 presents the IHS 2nd edition criteria (6).

Migraine with Aura

About 20% of children report the gradual onset of a visual aura before or during the onset of the headache. Descriptions include spots, colors, dots, or lights, which are usually found in both eyes but occasionally occur only in one eye. The aura usually lasts less than 30 minutes. Table 10-2 provides criteria for diagnosis.

"Alice in Wonderland" syndrome is a rare migraine aura in which patients experience distortion in body image characterized by enlargement, diminution, or distortion of part of or the whole body that they know is not real. The syndrome can occur at any age but is more common in children. The etiology may be migrainous ischemia of the nondominant posterior parietal lobule. Other rare visual hallucinations, distortions, and illusions

Table 10-2. IHS 2nd edition classification for migraine without aura and with typical aura

Pediatric migraine without aura (footnotes incorporated into adult classification)

A. At least five attacks fulfilling B–D
B. Headache attacks lasting 1 to 72 hours (untreated or unsuccessfully treated)
C. Headache has at least two of the following characteristics:
   1. Unilateral location. (Migraine headache is commonly bilateral in young children; an adult pattern of unilateral pain usually emerges in late adolescence or early adult life. Migraine headache is usually frontotemporal. Occipital headache in children, whether unilateral or bilateral, is rare and calls for diagnostic caution; many cases are attributable to structural lesions.)
   2. Pulsating quality
   3. Moderate or severe pain intensity
   4. Aggravation by or causing avoidance of routine physical activity (e.g. walking or climbing stairs)
D. During headache, at least one of the following:
   1. Nausea and/or vomiting
   2. Photophobia and phonophobia (may be inferred from the behavior of young children)

Typical aura with migraine headache

A. At least 2 attacks fulfilling criteria B–D
B. Aura consisting of at least one of the following, but no motor weakness: fully reversible visual symptoms including positive features (e.g., flickering lights, spots or lines) and/or negative features (i.e., loss of vision); fully reversible sensory symptoms including positive features (i.e., pins and needles) and/or negative features (i.e., numbness); fully reversible dysphasic speech disturbance
C. At least two of the following:
   1. homonymous visual symptoms and/or unilateral sensory symptoms
   2. at least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over (5 minutes
   3. each symptom lasts ≥5 and ≥60 minutes
D. Headache fulfilling criteria B–D for 1.1 Migraine without aura begins during the aura or follows aura within 60 minutes
E. Not attributed to another disorder

that have been reported in migraine include the following: zoopsia (visual hallucinations containing complex objects such as people and animals); achromatopsia (no perception of color); prosopagnosia (inability to recognize faces); visual agnosia (inability to recognize objects); akinetopsia (loss of ability to perceive visual motion); metamorphopsia (distortion of the shapes

of objects); micropsia (objects appear too small); macropsia (objects appear too big); teleopsia (objects seem too far away); lilliputianism (people appear too small); multiple images; persistent positive visual phenomena (diffuse small particles such as TV static or dots in the entire visual field lasting months to years); palinopsia (the persistence or recurrence of visual images after the exciting stimulus object has been removed); cerebral polyopia (the perception of multiple images); and tilted and upside-down vision (7).

Rarely, benign occipital epilepsy can mimic migraine with aura in children and adolescents. Visual symptoms, such as amaurosis, phosphenes (flashes of light), illusions, and visual hallucinations, may be followed by usually hemiclonic movements. Other types of seizure activity can occur, such as simple partial, complex partial, and partial with secondary generalization. Headache, nausea, vomiting, and vertigo can be pre- and postictal symptoms. The electroencephalogram (EEG) usually shows occipital discharges.

Familial Hemiplegic Migraine

Familial hemiplegic migraine (8), a rare variant, is migraine with aura that includes hemiplegia or hemiparesis. At least one first- or second-degree relative has migraine aura including motor weakness. The inheritance is autosomal dominant. Familial hemiplegic migraine type 1 (FHM1) is localized to the gene CACNA1A on chromosome 19. CACNA1A encodes the alpha 1A subunit of voltage-gated P/Q-type calcium channels in neurons. Thus FHM1 is a calcium channelopathy. Familial hemiplegic migraine type 2 (FHM2) is localized to chromosome 1 where mutations occur in the ATP1A2 gene (the Na+, K+-ATPase pump gene). In approximately 50% of FHM1 families, chronic progressive cerebellar ataxia occurs independently of the migraine attacks.

The attack onset is usually in childhood, virtually always before 30 years of age, with a mean age of onset of 12 years. The frequency of attacks range from 1 per day to less than 5 in a lifetime with a mean of 2 or 3 per year. Long attack-free intervals from 2 to 37 years have been reported. The two most common triggers are emotional stress and minor head trauma.

Attacks may occur on the same or different side from prior episodes. The face, arm, and leg typically become paretic with a slow, spreading progression. There may be an associated alteration in consciousness that ranges from confusion to coma. When the dominant hemisphere is involved, aphasia may also be present. The headache can be ipsilateral to the paresis in one third of cases. The hemiparesis may last from less than 1 hour to days or weeks. Complete recovery usually occurs. Triptans and dihydroergotamine (DHE) should not be used during the neurological deficit because of the potential for vasoconstriction and stroke. Beta-blocker medications might be avoided because of anecdotal reports of migraine-induced stroke. Verapamil, divalproex, and topiramate can be used as preventive agents.

Depending on the availability of a family history, the evaluation is similar to that of stroke in the young and may include magnetic resonance imaging (MRI) with magnetic resonance

angiography (MRA) and sometimes magnetic resonance venography, blood studies (complete blood count, with platelets, anticardiolipin antibodies, lupus anticoagulant, antithrombin III, protein S and C, factor V mutation, and others, depending on the clinical context), and cardiac evaluation, such as two-dimensional echocardiography. There are numerous causes of hemiparesis and headache, including partial seizures, congenital heart disease, acquired heart disease, infectious/inflammatory disease (e.g., HIV and varicella encephalitis), systemic vascular dysfunction (e.g., venous sinus thrombosis and hypertension), vascular disorders (e.g., carotid artery dissection; homocystinuria; mitochondrial encephalomyopathy, lactic acidosis, and strokelike episodes [MELAS] syndrome; and connective tissue disorders), hematological disorders (e.g., hemoglobinopathies, disseminated intravascular coagulation, and antiphospholipid antibody syndrome), cerebrovascular malformations (e.g., arteriovenous malformations, aneurysms, and Sturge-Weber syndrome), and head trauma.

Basilar-Type Migraine

Migraine with aura symptoms originating from the brain stem or from both occipital lobes is known as basilar-type migraine (Table 10-3) (9). The aura of basilar-type migraine usually lasts from 5 to 60 minutes but can last up to 3 days. Visual

Table 10-3. IHS 2nd edition diagnostic criteria for basilar-type migraine (Migraine with aura symptoms clearly originating from the brain stem and/or from both hemispheres simultaneously affected, but no motor weakness.)

A. At least 2 attacks fulfilling criteria B–D
B. Aura consisting of at least two of the following fully reversible symptoms, but no motor weakness:
   1. dysarthria
   2. vertigo
   3. tinnitus
   4. hypacusia
   5. diplopia
   6. visual symptoms simultaneously in both temporal and nasal fields of both eyes
   7. ataxia
   8. decreased level of consciousness
   9. simultaneously bilateral paraesthesias
C. At least one of the following:
   1. at least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over (5 minutes
   2. each aura symptom lasts (5 and (60 minutes
D. Headache fulfilling criteria B–D for $$Migraine without aura begins during the aura or follows aura within 60 minutes
E. Not attributed to another disorder

symptoms–including blurred vision, teichopsia (shimmering colored lights accompanied by blank spots in the visual field), scintillating scotoma, graying of vision, or total loss of vision-may start in one visual field and spread to become bilateral. Diplopia may be present in up to 16% of cases. Vertigo (which can be present with tinnitus), dysarthria, gait ataxia, and paresthesias (usually bilateral but may alternate sides with a hemidistribution) may be present alone or in various combinations. In 50% of cases, bilateral motor weakness occurs. Impairment of consciousness often occurs, including obtundation, amnesia, syncope, and, rarely, prolonged coma.

A severe throbbing headache is present in 96% of cases, usually with a bilateral occipital location. Nausea and vomiting typically occur, with light and noise sensitivity occurring in up to 50%.

The differential diagnosis and diagnostic evaluation are similar to those in hemiplegic migraine. Partial seizures, especially of occipital and temporal lobe origin, can have features similar to basilar migraine. An EEG study may be considered part of the evaluation. EEG abnormalities are found in less than 20% of cases. Children and adolescents may have interictal occipital spike-slow-wave or spike-wave activity.

Basilar-type migraine is an uncommon disorder. The onset is typically before 30 years of age (although the first attack occasionally occurs in those over 50 years of age) with a female preponderance following puberty of 3:1, as in other forms of migraine. The age of onset peaks during adolescence. Children may also have this migraine type. Those with basilar-type migraine may also have other types of migraine, although the basilar type is the predominant one in 75% of cases.

The frequency of basilar migraine decreases as patients enter their 20s and 30s. Stroke is a rare complication. As with other forms of migraine, triggers should be avoided if possible. Analgesics or nonsteroidal antiinflammatory drugs (NSAIDs) can be used for the pain. The package inserts for triptans, ergotamine, and DHE list basilar and hemiplegic migraine as contraindications to use based on the concern over vasoconstrictive properties. Based on a small case series in which triptans were safely used in basilar-type and familial hemiplegic migraine, Klapper et al. (10) argue that triptans may actually be a safe and effective treatment for headache in this circumstance because of a neuronal mechanism of action. Beta-blockers should be avoided because of a theoretical potential for stroke (based on a small case series), but verapamil, divalproex, topiramate, and antidepressants such as amitriptyline can be tried as preventive agents.

Ophthalmoplegic Migraine

Patients with ophthalmoplegic migraine present complaining of migraine headache and diplopia. This is a rare condition. Onset is often during adolescence, although it may occur during infancy. MRI studies have been reported as demonstrating thickening and enhancement of the oculomotor nerve at its exit from the midbrain. Speculation on the pathophysiology includes a trigeminovascular migraine epiphenomenon and a recurrent demyelinating neuropathy (11).

As the intensity of an ipsilateral severe headache subsides after a day or more, paresis of one or more of cranial nerves III, IV, and VI occurs. The third cranial nerve is involved in about 80% of cases, initially with ptosis and then with oculomotor paresis that is usually complete but may be partial. Dilation of the pupil, mydriasis, is present in more than 50% of cases. Recovery of nerve function may occur in a week to 4 to 6 weeks. Recovery may be incomplete after multiple attacks. Early high-dose corticosteroid treatment may be beneficial.

The diagnosis is made by excluding, as appropriate, such conditions as Tolosa-Hunt syndrome (granulomatous inflammation in the cavernous sinus), parasellar lesions, diabetic cranial neuropathy, collagen vascular disease, and orbital pseudotumor (an idiopathic infiltration of orbital structures with chronic inflammatory cells). MRI with MRA is usually adequate, although a cerebral arteriogram may be necessary in some cases.

Benign Paroxysmal Vertigo of Childhood

The onset of benign paroxysmal vertigo of childhood (12) usually occurs between 2 and 5 years of age but can be before 1 year of age or as late as 12 years of age. Unprovoked stereotypical episodes of true vertigo (with a sensation of movement as described by verbal children) usually last for seconds or minutes but may last for hours. The child becomes pale, cannot maintain an upright posture, and wishes to remain absolutely still. There is no complaint of headache or alteration of consciousness, although nausea or other abdominal discomfort may follow the vertigo. Because the episodes are so brief, treatment is not usually needed.

As the child becomes older, the episodes of vertigo may be associated with migraine headache or may become less severe and disappear. Other types of migraine may then occur in 21% of these patients (13).

Other causes of vertigo in children should be considered. A single prolonged episode could be due to infection of the labyrinth or vestibular nerve. Partial seizures can also produce true vertigo.

Abdominal Migraine

According to the IHS 2nd edition criteria (6), the diagnostic criteria for abdominal migraine require at least five attacks of abdominal pain lasting 1 to 72 hours (untreated or unsuccessfully treated) fulfilling the following criteria. The abdominal pain has all of the following characteristics: midline location, periumbilical or poorly localized, dull or "just sore" quality, and moderate or severe intensity. During abdominal pain, at least two of the four symptoms of anorexia, nausea, vomiting, and pallor are present and not attributed to another disorder.

The prevalence peaks at ages 5 to 9 years. As with all migraine types, this is a diagnosis of exclusion. If there is alteration of consciousness, a seizure disorder should be considered. Other disorders in the differential diagnosis include urogenital disorders, ornithine transcarbamylase deficiency, peptic ulcer disease, cholecystitis, Meckel's diverticulum, partial duodenal obstruction, gastroesophageal reflux, Crohn's disease, and irritable

bowel syndrome. Drugs used for migraine prevention and symptomatic treatment may be helpful.

Confusional Migraine

Confusional migraine is migraine with a headache, which can be minimal, associated with a confusional state that can last from 10 minutes to 2 days. The patient may be agitated and have impaired memory. There may be inattention, distractibility, and difficulty maintaining coherent speech or action. The diagnosis is made by excluding, as appropriate, the numerous causes of an acute encephalopathy, including partial complex seizures, metabolic disorders, infection, and subarachnoid hemorrhage (SAH).

"Footballer's" Migraine

As discussed in Chapter 9, acute minor head trauma can trigger migraine in children and adolescents. In a study of children with a mean age of 7.4 years who had mild head injuries, a history of motion sickness, migraines, and migraine in other family members is highly predictive of vomiting after a mild head injury (14).

MELAS Syndrome

AQ1 Mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes (MELAS), a rare disorder, can present as episodic migraine early in the course of the disease (17). The following features must be present: strokelike episodes typically before 40 years of age; encephalopathy with seizures, dementia, or both; and evidence of a mitochondrial myopathy with lactic acidosis, ragged-red fibers, or both. At least two of the following should be present: normal early development, recurrent headache, or recurrent vomiting. Most patients have exercise intolerance, limb weakness, short stature, hearing loss, and elevated cerebrospinal fluid (CSF) protein.

The cause of 80% of cases is an A-to-G point mutation in the mitochondrial gene encoding for $tRNA^{[Leu(UUR)]}$ at nucleotide position 3243. The other 20% are due to 14 other mitochondrial DNA point mutations. All children of mothers with MELAS are affected because of maternal transmission of mitochondrial DNA.

Management

Nonmedication Approaches

Identification and avoidance of migraine triggers are important. Missing meals, stress, and sleep deprivation can be particularly important triggers. Elimination of caffeinated beverages can be helpful in some cases. Biofeedback, stress management, and progressive relaxation training may be beneficial (16). Education about migraine for the patient and parents or caretakers is well worthwhile.

Pharmacological Treatments

Acute or symptomatic and preventive medications are available. Many of the recommendations in this section are anecdotal because of the lack of well-designed studies of treatment of childhood and adolescent migraine.

ACUTE HEADACHES. Aspirin should be avoided before 15 years of age because of the potential for Reye's syndrome. Headaches in children 6 years of age and younger are typically brief and resolve with acetaminophen and/or sleep. For children over 6 years of age, acetaminophen (10 to 15 mg/kg) may be effective. Other options (Table 10-4) include NSAIDs such as ibuprofen (10 mg/kg) (17), naproxen sodium (10 mg/kg); isometheptene mucate (65 mg), dichloralphenazone (100 mg), and acetaminophen (325 mg) (Midrin, one capsule); butalbital (50 mg), acetaminophen (325 mg), and caffeine (40 mg) (Fioricet, 6 to 9 years, one-half tablet; 9 to 12 years, three-fourths tablet; and more than 12 years, one tablet).

Children with significant nausea and/or vomiting may benefit from the use of metoclopramide 0.2 mg/kg orally or promethazine 0.5 mg/kg orally or in suppository form. Some children and adolescents also benefit from a combination of codeine and acetaminophen or acetaminophen, butalbital, and caffeine.

Total weekly doses of symptomatic medications and caffeine should be carefully monitored because of the potential for causing rebound headaches. There are restrictions on the use of certain acute and preventive medications in hemiplegic and basilar migraine as detailed earlier.

Additional treatments are available for severe migraines in children and adolescents ages 6 and over who do not respond to these medications. In a study of 50 children between the ages 6 and 18, Linder administered sumatriptan (Imitrex) subcutaneously (dose of 0.06 mg/kg) and reported efficacy in 78% (18). In responders, the migraine recurrence rate was 6%. Adverse events, usually transient and mild, occur in 80%.

Oral triptans are often effective in adolescents of 12 years of age and over. Sumatriptan nasal spray (ages 4–7 years, 5 mg; older than 7 years, 20 mg) and zolmitriptan nasal spray (older than 12 years, 5 mg) can also be effective. Anecdotally, triptans may be effective in children over 6 years of age. Side effects of triptans are discussed in Chapter 2. Table 10-4 provides dosages of triptans commonly used in children. Triptans are not approved by the Food and Drug Administration (FDA) for those under 18 years of age.

Alternatively, as in adults, children and adolescents with prolonged migraine often respond to an inpatient protocol of an antiemetic, metoclopramide, and DHE (Table 10-5), as reported by Linder (19). The oral metoclopramide and intravenous (IV) DHE can be given every 6 hours for a maximum of eight doses. When the headache ceases, one additional dose can be given. The dose of DHE may be increased by 0.05 mg/dose up to the point at which the patient has mild abdominal discomfort. The protocol should be continued at the dose prior to the onset of the abdominal discomfort. Triptans and DHE should not be given within less than 24 hours of each other.

These medications can have significant side effects. If metoclopramide causes an extrapyramidal syndrome, diphenhydramine can be given (1 mg/kg, maximum dose of 50 mg) orally, intramuscularly, or IV. Metoclopramide can also cause nausea and vomiting. For subsequent DHE doses, ondansetron 0.15 mg/kg IV 30 minutes prior to the DHE dose can be given as an

Table 10-4. Symptomatic treatment for migraine in children and adolescents

Medication	Dosage
Acetaminophen	10–15 mg/kg orally (po)
Pseudoephedrine HCL	30 mg po
Ibuprofen	10 mg/kg po
Naproxen sodium	10 mg/kg po
Butalbital 50 mg, acetaminophen 325 mg, caffeine 40 mg	6–9 yr, $^1/_2$ tablet
	9–12 yr, $^3/_4$ tablet
	>12 yr, 1 tablet
Isometheptene mucate, dichloralphenazone 100 mg and acetaminophen 325 mg	6–12 yr, 1 capsule
	>12 yr, 1-2 capsules
Sumatriptan	0.06 mg/kg SC
	25–mg for wt <50 lb
	25–50 mg for wt 50–100 lb
	50–100 mg for wt >100 lb
	5 mg NS 4–7 yr
	20 mg NS >7 yr
Zolmitriptan	6–8 yr, 1.25 mg po or 2.5 mg NS
	9–11 yr, 2.5 mg po or 2.5 mg NS
	>12 yr, 5 mg po or 5 mg NS
Rizatriptan	6–8 yr, 2.5 mg
	9–11 yr, 5 mg
	>12 yr, 10 mg
Almotriptan	>12 yr, 12.5 mg
Eletriptan	20 mg for wt ≤100 lb
	40 mg for wt ≤100 lb
Naratriptan	>12 yr, 2.5 mg
Frovatriptan	>12 yr, 2.5 mg
DHE IV	(see Table 10-6)

alternative, if necessary, to prevent nausea and vomiting, which can be a side effect of DHE or part of the migraine. Side effects of DHE include a flushed feeling, tingling in the extremities, leg cramping, and a transient increase in headache. DHE is not FDA approved for use in those under 18 years of age.

PREVENTIVE MEDICATIONS. Preventive medications should be considered for children and adolescents with frequent migraines that are not responsive to symptomatic medications or that significantly interfere with school or home activities (Table 10-6). In

Table 10-5. Dosing of metoclopramide and DHE for severe intractable migraine

Age in years	Metoclopramide[a]	DHE
6–9	0.2 mg/kg	0.1 mg/dose
9–12	0.2 mg/kg	0.15 mg/dose
12–16	0.2 mg/kg	0.2 mg/dose

[a]Administered orally 30 minutes prior to administration of IV DHE. Maximum dose of 20 mg.

Table 10-6. Preventive medications for migraine in children and adolescents

Medication	Dosage
Propanolol	<14 yr, initial dose 10 mg po bid. May increase by 10 mg/day/each week to 20 mg tid maximum >14 yr, initial dose 20 mg po bid. May increase by 20 mg/day each week up to 240 mg/day. Equivalent long-acting doses may be used.
Cyproheptadine HCl	>6 yr, 4 mg po hs. May be slowly increased to 12 mg po hs or 8 mg po hs and 4 mg po q a.m.
Amitriptyline or nortriptyline	10 mg po hs. May be increased every 2 weeks to 50 mg po hs <12 yr and 100 mg po hs >12 yr
Topiramate	<12 years: week 1, 25 mg evening; week 2, 25 mg am, 25 mg evening; week 3, 25 mg am, 50 mg evening; week 4 and maintenance, 50 mg every 12 h (slow titration if side effects)
	>12 years: week 1, .25–1 mg/kg/d (no more than 25 mg); increase by initial dose each week in divided doses up to 50–100 mg total/day (slow titration if side effects)
Divalproex sodium	125–250 mg po hs, slowly increase to 500–1,000 mg in two divided doses (or once a day with ER formulation)

general, preventive medications are started at low doses and are increased slowly.

Beta-blockers such as propranolol may be effective. The initial dose for children 8 years of age or older is 10 mg two times a day, which can be increased, depending on response, by 10 mg per week or slower to a maximum of 20 mg three times a day for

children under 14 years of age. Adult doses (Table 10-6) can be given to those 14 years of age and older. Those on higher doses of propranolol can be switched to or started on the long-acting preparation. Among the numerous possible side effects, beta-blockers can occasionally exacerbate asthma, cause hypotension, and cause depression. Propranolol given to diabetic children on insulin can mask symptoms of hypoglycemia. Congestive heart failure, atrioventricular conduction defects, and renal insufficiency are also contraindications to use. In some patients, nadolol or atenolol may be better tolerated, with fewer side effects, such as depression or asthenia, than propranolol.

Cyproheptadine (Periactin), an antihistamine, can also be an effective preventive medication in single doses of from 4 to 12 mg at bedtime (hs) or in divided doses, such as morning and evening for children 6 years and older (e.g., starting at 4 mg hs and, depending on effect, slowly increasing after several weeks to 8 mg hs, and later, if necessary, to 12 mg hs or 8 mg hs and 4 mg in the morning). The dose for children under 6 years of age is 1 mg hs, slowly increasing to 2 mg hs and 2 mg in the morning. Common side effects include weight gain and drowsiness. Cyproheptadine may be the preventive of first choice for those with frequent migraines and atopic allergies or sinus disease.

Tricyclic antidepressants, such as amitriptyline (Elavil) and nortriptyline (Pamelor), can also be used. For children over 8 years of age, the starting dose of either is 10 mg/day hs. Depending on the response and side effects, the total daily dose can be increased by 10 mg every 1 to 2 weeks or slower. Effective daily dosage is typically 50 mg or less in younger children and 100 mg or less in adolescents. Common side effects include sedation, weight gain, and dry mouth. Rarely, cardiac conduction abnormalities can occur with prolongation of the P-R, QRS, and Q-T intervals. Nortriptyline is less sedating than amitriptyline. The tricylics are often the preventive of choice for those with frequent migraine and tension-type headaches, chronic daily headaches, or associated depression or sleep disturbance. If a tricyclic is ineffective, a trial of trazadone (1 mg/kg a day divided into three doses) might be considered (20).

As discussed in Chapter 2, large clinical trials have demonstrated that topiramate is an effective medication for prevention for migraineurs 12 years of age or older. An open-label study also showed efficacy in younger children (21). For children 12 and older, the medication is started at 25 mg in the evening for the first week, 25 mg in the morning and 25 mg in the evening for the second week, 25 mg in the morning and 50 mg evening for the third week, and 50 mg in the morning and 50 mg in the evening (50 mg twice a day) starting for the fourth week, which is the maintenance dose. If side effects such as sleepiness, speech, or concentration problems occur, the patient may benefit from reducing the dose (e.g., starting at 12.5 mg or 15 mg) and slowing the increase of dose in the titration schedule to every 2 weeks. For children younger than 12 years, the initial dose is .25 to 1 mg mg/kg/day for a maximum of 25 mg hs increased by the initial dose once weekly to 50 to 100 mg. The total daily dose is given twice a day. Adverse events associated with topiramate are discussed in Chapter 2. Oligohydrosis (decreased sweating)

and hyperthermia have rarely been reported in children on topiramate. Toparamate may be preferred for treatment of migraineurs with comorbidity such as epilepsy, bipolar disease, essential tremor, or who are overweight.

Valproic acid may also be effective for migraine prevention in children and adolescents. Divalproex sodium (Depakote), the enteric coated form, is commonly used to minimize gastrointestinal side effects. According to data from adult studies, there may be efficacy for migraine prevention at doses lower than those used for epilepsy. A starting dose of 125 to 250 mg given at bedtime may be used and then slowly increased at 2-week intervals or slower. The total daily effective dose, given at bedtime and in the morning, is often 500 mg a day or less in younger children and 500 to 1,000 mg a day in adolescents. Equivalent once-daily doses may be used if the Depakote ER formulation is prescribed. This total daily dose may be less than that used for the treatment of epilepsy. There are numerous side effects, as described in Chapter 2, most commonly weight gain, tremor, hair loss, and nausea. Tremor and hair loss are fully reversible after discontinuing the medication. If nausea is persistent, the sprinkle formulation may be better tolerated. The rare complication of fatal hepatotoxicity almost always occurs in children under 10 years of age, most under the age of 2 years. (This complication has been reported for children taking the medication for epilepsy. The risk for those on polytherapy is 1:8,307 and on monotherapy, 1:16,317 (22). This drug may be especially considered for those with migraine and comorbidity of epilepsy and bipolar disease.

Prognosis

Migraine with onset before 7 years of age more commonly remits in boys than in girls. By 22 years of age, 50% of men and 60% of women still have migraine. In those with severe migraine beginning between the ages of 7 and 15, 20% are migraine free by 25 years of age and 50% continue to have it into their 50s and 60s (23).

EPISODIC TENSION-TYPE HEADACHES

The most common recurrent headache in children and adolescents is episodic tension type. Tension-type headaches typically have the following characteristics: duration of 30 minutes to many days; bilateral with a pressing or tightening quality; mild to moderate intensity; and not worsened by routine physical activity. Although nausea is absent, light or noise sensitivity may be present.

When symptomatic medication is necessary, acetaminophen, ibuprofen, or naproxen sodium may be effective. Frequent use of these or other symptomatic drugs or caffeine can lead to medication rebound headaches.

If the headaches are frequent, adequate sleep, regular exercise, and avoidance of caffeine may be beneficial. Biofeedback, stress management, and progressive relaxation training may also be helpful. Psychological or psychiatric evaluation may be worthwhile in some cases in which school or family problems, stress, depression, or anxiety is prominent. If there is a significant muscle contraction contribution, treatments such as a

short-term course of muscle relaxants, NSAIDs, physical therapy, and a trial of a transcutaneous nerve stimulator unit may be warranted. Preventive medications such as amitriptyline, nortriptyline, and paroxetine (Paxil 10 to 20 mg daily) can be effective.

CHRONIC NONPROGRESSIVE HEADACHES (24)

Frequent nonmigrainous headaches occur in 2.5% of children by 7 years of age and in 15.7% by 15 years of age. The headache may be present on awakening and last all day. The history, examination, and neuroimaging, as indicated, exclude secondary headaches. Chronic pansinusitis should be considered as the cause of chronic headaches even when sinus symptoms are not present.

Depending on the specifics, the headache may be classified as chronic tension type, mixed (both distinct migraine and tension headaches or headaches with features of both), transformed migraine (a history of episodic migraine transforming into daily or near-daily headaches), or caused by medication rebound . The headache may be difficult to classify, and, in many cases, it will paroxymally worsen and accrue migrainous features (25).

Medication rebound headache is important to identify because discontinuing the analgesics alone can produce great improvement (26). These patients typically have a history of migraine and/or tension-type headache. Without a definite precipitating event or following an injury or illness, the headaches may increase in frequency; then a pattern develops of daily or almost daily analgesic use. The daily analgesic use, in susceptible individuals, can cause daily bilateral or unilateral headaches with tension and migraine features. Frequent use of caffeine may also cause or contribute to daily headaches. Episodic migraine headaches may also occur. The headaches may persist for months or years. Preventive medications may be less effective in this setting.

Medications that can cause rebound headaches include the following: acetaminophen; ibuprofen, and other NSAIDs; combination drugs with such agents as butalbital, acetaminophen, caffeine, aspirin, and codeine; propoxyphene; and ergotamine. In some cases, frequent triptan use can also cause rebound headaches. The number of doses of analgesics taken per week, in the largest study with patients from ages 5 to 17 years, ranged from 8 to 84 (27). Acetaminophen and ibuprofen were the most commonly overused medications. Discontinuing daily analgesics and taking amitriptyline 10 mg orally daily reduced the frequency of headaches by 80%.

If headaches persist despite medication withdrawal or if chronic tension-type or mixed headaches are present, a psychological or psychiatric evaluation should be considered to evaluate the presence of home and school problems, other stressors, or depression, which may contribute to headaches. Biofeedback, stress management training, relaxation training, and behavioral contingency management may be helpful in reducing the headaches. The preceding section on tension-type headaches describes preventive medications that may be effective. Divalproex sodium can also be helpful in some cases. A 3-day IV

DHE protocol administered every 8 days can be indicated for some refractory cases (Table 10-5).

New daily, persistent headaches can also occur without a history of increasingly frequent tension- and/or migraine-type headaches. The headache develops over less than 3 days. In many cases, the etiology is unknown. Other cases may reflect a postviral syndrome.

Acute infectious mononucleosis should be considered, especially when there are accompanying complaints of sore throat and findings of cervical adenopathy. (The term *infectious mononucleosis* was first used in 1920 to describe medical students at Johns Hopkins with the condition who were found to have atypical mononuclear cells.) The typical picture is a 7-day prodromal illness followed by a 4-day to 3-week acute illness with fever, headache, malaise, pharyngitis, cervical lymphadenopathy, and mononuclear leukocytosis with atypical lymphocytes (28). Transient hepatic dysfunction and splenic and hepatomegaly may be present. The diagnosis is based on a positive heterophil antibody test, the appearance of atypical lymphocytes in the blood at 1 to 4 weeks after onset of disease, and/or by changes in EBV-specific antibodies. Longer duration chronic headaches can be present with persistent Epstein-Barr infection (29).

ACUTE HEADACHES

Epidemiology

Chapter 8 reviews first or worst headaches in adults. In children and adolescents, the epidemiology is different. In a study of 150 consecutive children presenting to the emergency department with a chief complaint of acute headache, the diagnoses were as follows: viral upper respiratory infection, 39%; migraine, 18%; sinusitis, 9%; streptococcal pharyngitis, viral meningitis, and undetermined cause, each 7%; posterior fossa tumor, 2.6%; ventriculoperitoneal shunt malfunction, 2%; intracranial hemorrhage and seizure, each 1.5%; postlumbar puncture and postconcussion, each 1% (30). Upper respiratory infections with fever accounted for 54% of the cases. Viral meningitis can present without fever and with a supple neck and normal neurological examination.

In children and adolescents, aneurysmal SAH is uncommon: fewer than 2% of cases occur in those under 18 years of age. SAH is more likely due to ruptured arteriovenous malformations, which outnumber aneurysms by nearly 10 to 1 in childhood.

Brain Abscess

Brain abscess in children has a peak incidence of 4 to 7 years. Twenty-five percent of these children have cyanotic congenital heart disease with a right-to-left shunt resulting in hematogenous spread of infection. Otogenic abscesses also occur in children. Brain abscesses due to frontal or sphenoid sinusitis occur in children ages 10 and older because of the late development of these sinuses (31). Emissary veins spread infection into the brain from the paranasal sinuses, mastoids, and middle ear.

About 75% of patients with brain abscess present with symptoms of less than 2 weeks' duration. The classic clinical triad of headache, fever, and focal neurological signs occurs in only a minority of patients. Headache is present in about 75% of patients, and nausea and vomiting are found in about 50%. Signs are present as follows: fever, less than 50%; seizures, 33%; nuchal rigidity, 25%; and papilledema, 25% (32).

CHRONIC PROGRESSIVE HEADACHES

The epidemiology of chronic progressive headaches is also different in children and adolescents from that in adults (Table 10-7) (23). Causes include brain tumors, hydrocephalus, brain abscess, hematomas, pseudotumor cerebri, malformation, hypertension, and medication rebound. Pseudotumor cerebri (Chapter 14) may be present without papilledema. The diagnosis can only be made with lumbar puncture and measurement of the opening pressure. This section reviews brain tumors and hydrocephalus. The remaining causes are discussed elsewhere in this book.

Brain Tumors

Although parents and children often fear their headache is due to a brain tumor, brain tumors occur uncommonly. The clinical presentation depends on the type and location of tumor. Posterior fossa tumors that result in hydrocephalus can produce the classic brain tumor headaches with nausea, early morning vomiting, and headaches. Headaches from supratentorial tumors are less specific.

Table 10-7. Causes of chronic progressive headaches in children and adolescents

Neoplasms
Medulloblastoma
Cerebellar astrocytoma
Brain stem glioma
Ependymoma
Pineal region tumors
Craniopharyngioma
Supratentorial astrocytoma
Hydrocephalus
Obstructive
Communicating
Brain abscess
Chronic subdural and epidural hematomas
Pseudotumor cerebri
Malformations
Chiari malformation
Dandy-Walker cyst
Hypertension
Medication rebound

In a study of 74 children with primary brain tumors from England, headache was present in 64%, vomiting in 65%, and changes in personality in 47% (33). Only 34% of headaches were always associated with vomiting, and only 28% occurred in the early morning. Misdiagnosis was common: migraine was diagnosed in 24% and a psychological etiology was found in 15%. Additional features of headaches due to brain tumors are discussed in Chapter 14.

Brain metastases in children and adolescents most often arise from sarcomas and germ cell tumors. A variety of primary brain tumors can occur. About 60% of primary brain tumors are infratentorial (posterior fossa) and 40% supratentorial. The annual incidence of pediatric primary brain tumors is about 2 or 3 out of 100,000.

Medulloblastoma

Medulloblastomas (primitive neuroectodermal tumors), the most common, account for 20% of childhood brain tumors and 30% to 40% of posterior fossa childhood tumors. The tumor may occur at any time of life, including adulthood, but it is most common in the first decade, with peaks at 3 to 4 years of age and 8 to 10 years. Medulloblastomas usually arise from the cerebellar vermis. Symptoms and signs are due to obstruction of the fourth ventricle and hydrocephalus, infiltration of cerebellar tissue, and leptomeningeal spread. By the time of diagnosis, 90% of patients have papilledema, headaches, vomiting (especially morning vomiting), and lethargy. Ataxia is often present early in the course of the disease.

Cerebellar Astrocytoma

The classic, or juvenile, pilocytic cerebellar astrocytoma is a slow-growing lesion arising from the lateral cerebellar hemispheres. This astrocytoma is the second most common tumor of the posterior fossa, accounting for 30% to 40% of cases, and it comprises 10% to 20% of all childhood brain tumors. The peak ages of incidence are the latter half of the first decade and the first half of the second decade of life. Initially, appendicular cerebellar symptoms may be present for weeks to months. As the tumor extends to the midline and obstructs the fourth ventricle resulting in hydrocephalus, the classic brain tumor symptoms of early morning vomiting and headaches may be present.

Brain Stem Glioma

Brain stem gliomas constitute 10% to 20% of all childhood brain tumors and are the third most common posterior fossa tumor. The median age of occurrence is between 5 and 9 years. The neurological presentation depends on the location of the tumor. The classic present is a triad of cranial neuropathies, ataxia, and long tract signs. About one third of patients have headache, nausea, and vomiting.

Ependymoma

Ependymomas comprise 5% to 10% of childhood primary brain tumors. Two thirds arise in the posterior fossa and are usually benign, whereas one third arise supratentorially and are

usually malignant. Infratentorial ependymomas arise from the floor, roof, or lateral recesses of the fourth ventricle. By the time of diagnosis, most of the infratentorial tumors have blocked the third or fourth ventricle, producing hydrocephalus. Headaches, nausea, and vomiting will then occur. Depending on location of the tumor, supratentorial ependymomas can produce focal neurological findings and seizures. By the time of diagnosis, most patients have headaches and other signs and symptoms of increased intracranial pressure.

Pineal Region Lesions

Pineal region lesions include germinomas (1% of childhood primaries), pineoblastomas, glial neoplasms, meningiomas, lymphomas, and pineal cysts. Growth of the tumor causes compression of the aqueduct of Sylvius and hydrocephalus. The typical picture of hydrocephalus can occur with headaches, nausea, and vomiting. Involvement of the superior colliculus can lead to Parinaud's syndrome with paralysis of upgaze, near-light dissociation, and convergence-retraction nystagmus.

CRANIOPHARYNGIOMA. Craniopharyngiomas are benign tumors located in the parasellar region. Although they can occur at any age, the onset is before 15 years of age in 50% of cases. These are the third most common primary in children after medulloblastomas and gliomas. Growth failure is the most common sign at presentation. Visual dysfunction is present in up to 70% of patients at the time of presentation because of the prechiasmal location. Fifty percent of patients complain of severe headaches.

ASTROCYTOMA. Supratentorial astrocytomas can produce focal neurological findings and seizures (in about 25% of cases). Low-grade gliomas can produce a very gradual onset of symptoms, including headache and/or subtle neurobehavioral changes. Increased difficulty with schoolwork can be blamed on social or psychological factors. Malignant astrocytomas are more commonly seen in adults.

Hydrocephalus (34)

There are two categories of hydrocephalus, which is a heterogenous disorder. Obstructive, or noncommunicating, hydrocephalus is due to a blockage of CSF pathways at or proximal to the outlet foramina of the fourth ventricle, the foraminas of Luschka and Magendie. Communicating hydrocephalus is due to a blockage of CSF in the basal subarachnoid cisterns, in the subarachnoid spaces over the brain surface, or within the arachnoid granulations.

Table 10-8 lists the various causes of hydrocephalus in children and adults. Diagnostic testing helps define the features of hydrocephalus, including the site of blockage of CSF, the etiology, and whether the condition is arrested or progressive.

Clinical Manifestations

Small children may present with symptoms and signs of raised intracranial pressure, including headaches, vomiting, irritability, lethargy, and poor feeding. Acute hydrocephalus in older children can result in headaches, often worse in the morning; vomiting; cranial nerve VI palsies; papilledema; and altered

Table 10-8. Causes of hydrocephalus

Noncommunicating	Communicating
Aqueductal stenosis	Chiari malformation
Chiari malformation	Dandy-Walker malformation
Dandy-Walker malformation	Encephalocele
Atresia of the foramen of Monroe	Benign cysts
Skull bases anomalies	Incompetent arachnoid villi
Neoplasms	Leptomeningeal inflammation
Benign intracranial cysts	Viral infection
Inflammatory ventriculitis	Bacterial infection
Hemorrhage	Subarachnoid hemorrhage
Infection	Chemical arachnoiditis
Chemical meningitis	Carcinomatous meningitis
Ruptured arachnoid cyst	

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levels of consciousness. Headaches due to hydrocephalus are often bilateral and are made worse by coughing, sneezing, straining, or head movement.

Ventriculoperitoneal shunts are appropriate treatment for many cases of hydrocephalus. Acute hydrocephalus due to shunt failure can result in headaches, vomiting, altered consciousness, and seizures. Physicians who care for children with shunts should be aware of the rare complication of slit ventricle syndrome.

Symptoms and signs of intermittent intracranial hypertension develop in a patient who has been stable for months to years with a shunt. A scan of the brain shows a smaller than normal ventricular system that could be due to acquired rigidity of the ventricular system. There are numerous other complications of shunts, including infections, subdural hematomas, and seizures.

Colloid Cysts of the Third Ventricle

Colloid cysts of the third ventricle are benign cysts that can move in and out of the foramen of Monro on its pedicle, producing intermittent obstruction of CSF. Colloid cysts are rarely diagnosed during childhood. The cysts can produce severe paroxysmal headaches that can be mistaken for migraine and can lead to sudden death in about 5% of cases (35).

Dandy-Walker Malformation

Dandy-Walker malformation is a developmental disorder characterized by partial or complete absence of the cerebellar vermis and cystlike dilatation of the fourth ventricle. Other features often present include hydrocephalus; enlargement of the posterior fossa; elevation of the tentorium, transverse sinus, or

both; and lack of patency of the foramina of Luschka, Magendie, or both. The incidence is about 1 in 30,000 live births. There are a variety of clinical presentations, including mental retardation in about 50%, ataxia, brain stem dysfunction, and symptoms and signs due to hydrocephalus, which is present in about 80% of cases by 1 year of age. The initial presentation can occur as late as adulthood, with such complaints as headache, cerebellar ataxia, and progressive spastic weakness of all four extremities.

REFERENCES

1. Bille B. Migraine in school children. *Acta Pediatr Scand* 1962;51[Suppl 136]:1–151.
2. Kandt RS. Childhood migraine. In: Gilman S, ed. *MedLink neurology*. San Diego: MedLink Corp. Available at www.medlink.com., 2004.
3. Jan MMS. History of motion sickness is predictive of childhood migraine. *J Paediatr Child Health* 1998;34:483–484.
4. Giroud M, Nivelon JL, Dumas R. [Somnambulism and migraine in children: a non-fortuitous association]. *Arch Fr Pediatr* 1987;44:263–265.
5. Stewart WF, Linet MS, Celentano DD, et al. Age and sex-specific incidence rates of migraine with and without visual aura. *Am J Epidemiol* 1993:34:1111–1120.
6. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders, 2nd ed. *Cephalalgia* 2004;24 (Suppl 1):59:1–160.
7. Liu GT, Volpe NJ, Galetta SL. Visual hallucinations and illusions. In: *Neuro-ophthalmology. Diagnosis and management*. Philadelphia: WB Saunders, 2001:401–424.
8. Peres MFP. Hemiplegic migraine. In: Gilman S, ed. *MedLink neurology*. San Diego: MedLink Corp. Available at www.medlink.com., 2004.
9. Siow HC, Welch KMA. Basilar migraine. In: Gilman S, ed. *MedLink neurology*. San Diego: MedLink Corp. Available at www.medlink.com., 2004.
10. Klapper J, Mathew N, Nett R. Triptans in the treatment of basilar migraine and migraine with prolonged aura. *Headache* 2001;41:981–984.
11. Carlow TJ. Oculomotor ophthalmoplegic migraine: is it really migraine? *J Neuroophthalmol* 2002;22:215–221.
12. Davidoff RA. Benign paroxysmal vertigo of childhood. In: Gilman S, ed. *MedLink neurology*. San Diego: MedLink Corp. Available at www.medlink.com., 2004.
13. Lindskog U, Ödkvist L, Noaksson L, et al. Benign paroxysmal vertigo in childhood: a long-term follow-up. *Headache* 1999;39:33–37.
14. Jan MM, Camfield PR, Gordon K, et al. Vomiting after mild head injury is related to migraine. *J Pediatr* 1997;130:134–137.
15. Hirano M. MELAS. In: Gilman S, ed. *MedLink neurology*. San Diego: MedLink Corp. Available at www.medlink.com., 2004.
16. Sartory G, Muller B, Metsch J, et al. A comparison of psychological and pharmacological treatment of pediatric migraine. *Behav Res Ther* 1998;36:1155–1170.
17. Hamalainen ML, Hoppo K, Valkeila E, et al. Ibuprofen or acetaminophen for the acute treatment of migraine in children: a

double-blind, randomized, placebo-controlled crossover study. *Neurology* 1997;48:103–107.
18. Linder S. Subcutaneous sumatriptan in the clinical setting: the first fifty consecutive patients with acute migraine in a pediatric neurology office practice. *Headache* 1996;36:419–422.
19. Linder S. Treatment of childhood headache with dihydroergotamine mesylate. *Headache* 1994;34:578–580.
20. Battistella PA, Ruffilli R, Cernetti R, et al. A placebo-controlled crossover trial using trazadone in pediatric migraine. *Headache* 1993;33:36–39.
21. Hershey AD, Powers SW, Vockell AL, et al. Effectiveness of topiramate in the prevention of childhood headaches. *Headache* 2002;42: 810–818.
22. Silberstein SD. Divalproex sodium in headache: literature review and clinical guidelines. *Headache* 1996;36:547–555.
23. Bille B. A 40-year follow-up of school children with migraine. *Cephalalgia* 1997;17:488–491.
24. Rothner AD, Winner P. Headaches in children and adolescents. In Silberstein SD, Lipton RB, Dalessio DJ, eds. *Wolff's headache and other head pain,* 7th ed. New York: Oxford University Press, 2001:539–561.
25. Koenig MA, Gladstein J, McCarter RJ, et al. Chronic daily headache in children and adolescents presenting to tertiary headache clinics. *Headache* 2002;42: 491–500.
26. Symon DN. Twelve cases of analgesic headache. *Arch Dis Child* 1998;73:555–556.
27. Vasconcellos E, Piña-Garza JE, Millan EJ, et al. Analgesic rebound headache in children and adolescents. *J Child Neurol* 1998;13:443–447.
28. Roos KL. Chapter 41. Viral infections. In: Goetz CG, ed. *Textbook of clinical neurology,* 2nd ed. Philadelphia: WB Saunders, 2003: 895–918.
29. Vanast WJ. New daily persistent headaches: definition of a benign syndrome. *Headache* 1987;26:318.
30. Lewis DW, Qureshi F. Acute headache in children and adolescents presenting to the emergency department. *Headache* 2000; 40:200–203.
31. Giannoni C, Sulek M, Friedman EM. Intracranial complications of sinusitis: a pediatric series. *Am J Rhinol* 1998;12:173–178.
32. Greenlee JE. Brain abscess. In: Gilman S, ed. *MedLink neurology.* San Diego: MedLink Corp. Available at www.medlink.com., 2004.
33. Edgeworth J, Bullock P, Bailey A, et al. Why are brain tumours still being missed? *Arch Dis Child* 1996;74:148–151.
34. Kinsman SL. Hydrocephalus. In: Gilman S, ed. *MedLink neurology.* San Diego: MedLink Corp. Available at www.medlink.com., 2004.
35. Aronica PA, Ahdab-Barmada M, Rozin L, et al. Sudden death in an adolescent boy due to a colloid cyst of the third ventricle. *Am J Forensic Med Pathol* 1998;19:119–122.

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