Neurocysticercosis has been diagnosed increasingly in the United States as a consequence of increased immigration from and travel to areas of endemic cysticercosis. We report a retrospective series of 47 pediatric cases of neurocysticercosis in our large children’s hospital in Chicago, which has a large immigrant population. Neurocysticercosis was diagnosed on the basis of any of the following three criteria: (1) surgical biopsy findings, (2) radiographic findings consistent with neurocysticercosis as well as diagnostic serum and/or cerebrospinal fluid titers, or (3) consistent radiographic findings and a compatible epidemiologic history (without diagnostic serological findings). Epidemiologic, clinical, laboratory, and radiographic data were analyzed. Neurocysticercosis is a relatively common cause of afebrile seizures in children who present to our emergency department in Chicago. Computed tomography and magnetic resonance imaging are both important modalities in evaluation of children with neurocysticercosis. Laboratory studies are neither sensitive for nor predictive of the diagnosis of neurocysticercosis. Therapy is well tolerated. The long-term prognosis for treated patients appears to be excellent.

In the last decade, neurocysticercosis has been diagnosed with increasing frequency in some areas of the United States (U.S.). Factors contributing to the increased incidence of diagnoses of neurocysticercosis include the large number of Hispanic immigrants from areas of endemia, increased case of travel to and from such regions throughout the world, and improved diagnostic radiographic modalities such as CT and MRI. The increased number of recently reported cases of neurocysticercosis involving persons living in the U.S. should heighten physicians’ awareness of neurocysticercosis as a diagnostic entity in the U.S.

The most recently reported series of pediatric neurocysticercosis in the U.S. included patients from the period of 1980 through 1986 [1]. None of the children in that report had been treated with anthelmintic agents, and MRI scanning results were not reported. In this article we review cases involving children with neurocysticercosis treated in recent years at our Chicago hospital and address the issues of treatment and MRI findings.

Methods

We performed a retrospective review of all cases of neurocysticercosis that were diagnosed at Children’s Memorial Hospital (Chicago) from 1986 through 1994. Patients were identified by reviews of (1) medical records of all inpatients and outpatients whose discharge diagnosis was cysticercosis or neurocysticercosis, (2) laboratory serology records, (3) pathology records, and (4) the Division of Infectious Diseases’ outpatient clinical and consultation records.

Patients were included in this series on the basis of any of the following three criteria: (1) surgical biopsy findings confirming the diagnosis of neurocysticercosis, (2) radiographic (MRI and/or CT scan) findings consistent with neurocysticercosis as well as diagnostic serum and/or CSF titers, or (3) consistent radiographic findings and a compatible epidemiologic history (without diagnostic serological findings). A compatible epidemiologic history was defined as immigration from or travel to areas of high endemicity, the hosting of a household visitor from an area of endemicity, or the use of a child-care provider from such an area.

Consistent radiographic features included single or multiple focal parenchymal, intraventricular, spinal, or subarachnoid lesions that were contrast-enhancing, cystic, or calcified. Patients with a compatible epidemiologic history (with or without diagnostic serological titers) but whose radiographic findings were not consistent were excluded from study. The following information was extracted from the records: epidemiologic history; clinical features of illness; laboratory, radiographic, and pathologic data; and features of treatment and follow-up.

Serum and CSF titers of antibodies to Taenia solium were determined by various methods during the time period of the study, including immunoblotting and ELISA. Serological results were considered diagnostic when the serum titer was $\geq 1:160$ and the CSF titer was $\geq 1:8$ or when an immunoblot was reported to be positive.

Statistical analysis was performed with use of Epi-Info Version 6 software (Centers for Disease Control and Prevention, Atlanta). Categorical data were analyzed by $\chi^2$ test with Yates’
Table 1. Signs and symptoms of the 47 patients with neurocysticercosis at presentation.

<table>
<thead>
<tr>
<th>Sign or symptom</th>
<th>No. (%) of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seizure</td>
<td>41 (87)</td>
</tr>
<tr>
<td>Focal seizure</td>
<td>32 (78)</td>
</tr>
<tr>
<td>Headache</td>
<td>15 (32)</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>15 (32)</td>
</tr>
<tr>
<td>Altered mental status</td>
<td>6 (13)</td>
</tr>
<tr>
<td>Cranial nerve palsy</td>
<td>4 (9)</td>
</tr>
<tr>
<td>Gait abnormality</td>
<td>2 (4)</td>
</tr>
<tr>
<td>Papilledema</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Decreased visual acuity</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Sensory changes</td>
<td>0</td>
</tr>
<tr>
<td>Fever</td>
<td>0</td>
</tr>
</tbody>
</table>

correction factor for small numbers, when applicable. Continuous data were analyzed with the Student’s t-test. Significance was defined as $P < .05$.

Results

A total of 74 records were evaluated for inclusion. Twenty-seven patients were excluded from study, for the following reasons: an alternative neurological diagnosis was established (6 patients), the condition was diagnosed elsewhere (1 patient), or the criteria for diagnosis of neurocysticercosis were not met because of incomplete radiographic evaluation (20 patients).

Forty-seven patients met our criteria for inclusion in the study. The mean age of these patients was 8.4 years (median, 8.9 years; range, 1.2–15.4 years); 29 (62%) were female and 45 (96%) were Hispanic. Seventeen (38%) of the Hispanic patients were born in Mexico, 7 (16%) in Central America, and 19 (42%) in the U.S. Of the two non-Hispanic patients, one was a native of India and the other was a U.S.-born white child.

The mean length of time for which the foreign-born patients lived in the U.S. was 32.5 months (range, 1–132 months). Ten (50%) of the 20 U.S.-born patients had most recently traveled to Mexico or Central America, a mean of 24 months (median, 6.5 months; range, 2–120 months) prior to their presentation. Four (20%) of the 20 U.S.-born patients had not traveled, but these four had recently had a visitor in their home from Mexico or Central America. The country of origin and travel and visitor history of two Hispanic patients were unknown. Four of the U.S.-born patients, three of whom were Hispanic, reported neither recent travel nor a foreign visitor.

All patients presented to the emergency room for initial evaluation. Presenting signs and symptoms are shown in table 1. Patients were symptomatic for <1 day to 8.75 years (median, 2 days) prior to seeking medical care. There was no significant difference in the duration of symptoms prior to the seeking of medical care by those who presented with seizure (median, 2 days) vs. headache (median, 4 days). In addition, there was no difference in the age of the patients who presented with seizures (mean, 8.2 years) vs. headaches (mean, 9 years).

Neither leukocytosis nor eosinophilia was noted in the 21 patients whose complete blood cell counts were determined. None of the 15 stool samples evaluated for the presence of ova and parasites was diagnostic. The cell count, differential cell count, and chemistry profile were all within the normal range for all 15 CSF samples evaluated.

Positive titers diagnostic for neurocysticercosis were demonstrated in the serum of 16 (44%) of 36 patients and in the CSF of two (25%) of eight patients tested. Nine (56%) of the 16 patients whose serological tests were diagnostic had a single demonstrable lesion, 6 (38%) had multiple lesions, and 1 (6%) had an intraventricular lesion. One patient whose CSF antibody titer was diagnostic had extensive lesions throughout the cerebral cortex, thalamus, and optic radiations, and the second was the child with an intraventricular cystic lesion.

Eleven (23%) of 47 patients had a pathological specimen obtained during their neurological evaluation, nine of whom underwent craniotomy and brain biopsy for diagnostic purposes. One patient underwent biopsy of a cervical lymph node, and the other underwent biopsy of a subcutaneous nodule. Eight (89%) of the nine brain biopsies yielded histologic findings consistent with neurocysticercosis (cysts with varying degrees of fibrosis, granulomata, and calcification). One brain biopsy demonstrated gliosis without distinguishing characteristics. The biopsied subcutaneous nodule revealed cysticercosis. The lymph node specimen demonstrated necrotizing granulomata.

Of the 9 patients with pathologically confirmed neurocysticercosis, only 1 underwent CSF serology (of which the result was diagnostic) and only 5 had serum titers determined (1 [20%] was diagnostic). The reasons for which some patients underwent biopsy without prior serological testing—and two underwent biopsy after negative serological results had been obtained—could not be ascertained from the medical records in this retrospective study, but they likely were related to concern about tumors or granulomatous disease.

Twenty-eight (60%) of the 47 patients were treated with praziquantel (mean, 14 days) and prednisone (mean, 5 days). The praziquantel dosage was 50 mg/(kg·d), and the prednisone dosage was 0.2–0.5 mg/(kg·d). Specific indications for antihelminthic therapy could not be extracted from the medical records. One patient was treated with steroids for increased intracranial pressure but never received antihelminthic therapy. Steroids were given to the other 28 patients in an effort to prevent the development of cerebral edema as a result of antihelminthic therapy.

Patients who did not undergo a surgical procedure were hospitalized for a mean of 2.5 days; the majority of the medical treatment was administered at home. There were no adverse reactions to therapy. Forty-two (90%) of the 47 patients began receiving anticonvulsant therapy during their initial evaluation in the emergency department.
CT and MRI scans were performed on 45 (96%) and 33 (70%) of the 47 patients, respectively, shortly after presentation. Overall, a single lesion with surrounding edema was identified on 25 (56%) of the 45 CT scans and on 17 (52%) of the 33 MRI scans. A scolex could be visualized only infrequently. Most lesions were demonstrated in the parietal lobe, and few were demonstrated in the occipital lobe.

Single nonenhancing lesions were demonstrated by CT and MRI scanning in three (7%) of 45 and one (3%) of 33 patients, respectively. A total of 15 CT scans (33%) and 12 MRI scans (36%) demonstrated multiple enhancing and/or nonenhancing lesions. Multiple (≥2) enhancing lesions were seen on 4 CT scans (9%) and 6 MRI scans (18%). Multiple lesions that did not enhance were seen on the CT scans of three patients (7%) but were not seen on MRI scans. A combination of enhancing and nonenhancing lesions was seen on eight (18%) of the 45 CT scans and six (18%) of the 33 MRI scans. A total of 28 CT scans (62%) and 18 MRI scans (55%) demonstrated some kind of single intraparenchymal lesion.

Thirty patients had both an MRI and CT scan performed within 24 hours following presentation, thus allowing comparison of these two modalities for detection of lesions. In four patients, the MRI scan revealed more extensive parenchymal enhancing lesions than did the CT scan, obtained at approximately the same time. One MRI scan revealed an intraventricular lesion that was not evident on the CT scan. Two MRI scans showed additional calcified lesions not visible on the CT scan, and two CT scans showed calcified lesions not seen on the MRI scan. One CT scan revealed an enhancing lesion that was not apparent on the accompanying MRI scan.

Twenty-six patients had at least one follow-up neuroradiographic imaging study after their initial scans. Follow-up scans were performed a mean of 3 months (range, 0.25–15 months) after the first CT scan and 6 months (range, 1–31 months) after the first MRI scan. In the six patients who had follow-up scanning after surgery, absence of the lesion was demonstrated. Eleven (85%) of the 13 follow-up scans for patients who had received praziquantel demonstrated decreased edema and diminution in the size of lesions, while two (15%) revealed greater edema and an increased number of lesions. One of the latter patients had a large number of lesions at presentation.

Of the 7 follow-up scans for patients who had not undergone surgical intervention or received praziquantel, 3 (43%) showed no change, 3 (43%) showed a decrease in edema and in the size of lesions, and 1 (14%) showed increased edema and an increased number of lesions (evident on subsequent scans).

There was no association between presenting signs and symptoms and the demonstration by scanning of single vs. multiple enhancing or nonenhancing lesions. One of the three patients whose scan demonstrated an intraventricular lesion presented with signs and symptoms of obstructive hydrocephalus. One patient with extensive cerebral, thalamic, and optic radiation involvement that was evident on scanning had sought medical attention for worsening visual acuity, hemiparesis, and seizures.

Another unusual patient presented initially with a subcutaneous nodule of the chest wall that had been present for 2 years. Biopsy of the lesion revealed cysticercosis. At that time the patient's head CT scan was normal. The patient returned for medical attention 15 months later because of seizures and reported no travel or new exposure during this period. At this time the CT and MRI scans revealed a single enhancing parietal lesion.

Outcome data are presented in table 2. The mean length of follow-up for the 36 patients who returned for at least one follow-up visit was 15.3 months. Twenty patients were followed for <3 months. The range of follow-up was 0–64 months. At their last known evaluation, 42 (89%) of the 47 patients were healthy and had no neurological deficits. Seventeen (40%) of 42 were not taking any medications, while the remaining 25 (60%) were receiving anticonvulsants.

The relatively minor neurological abnormalities in four patients (9%) included a seizure disorder poorly controlled by anticonvulsant therapy (2), hemiparesis (1), and motor and speech delay (1). The single patient with major neurological sequelae was an 11-year-old girl with hemiparesis, seizure disorder, and blindness, associated with dozens of lesions.

We assessed outcome by mode of therapy. Twenty-two (88%) of 25 patients treated only medically were completely healthy, although 13 (52%) required anticonvulsants. The other three patients included two with the above-noted minor abnormalities and the patient with blindness. All 13 patients who received no antihelminthic therapy were completely healthy, although 9 (69%) required anticonvulsants. Seven of the nine patients who underwent craniotomy were completely healthy, while the other two had minor abnormalities.

Discussion

The overwhelming majority of persons in the U.S. with neurocysticercosis have emigrated from or traveled to regions of the world, particularly Latin America, where cysticercosis is endemic [2–4]. Data from the 1990 census in Chicago indicate that the population was 19.6% Hispanic and that 28% of the children <16 years old were Hispanic [5]. Of the adults in Chicago who identified themselves as Hispanic, 68% reported that they were from Mexico, 22% from Puerto Rico, 2% from Cuba, and 12% from other Latin American areas [5]. The number of travelers to Latin America from Chicago and of foreign visitors to Chicago from these regions appears to be considerable.

The prevalence of neurocysticercosis in some regions of Mexico has been reported to be as high as 3.6% of the population [6]. In an autopsy series in Mexico, the prevalence of cerebral cysticercosis was 1.9% [7]. The duration of residence
in the U.S. by our own patients varied, although most of our cases involved recent immigrants.

Included in our series of 47 patients were one white child and three U.S.-born Hispanic children who lacked apparent risk factors for neurocysticercosis. A few previously reported cases involved no identifiable risk factor [8-10]. Some unusual means of transmission have also been reported, and these emphasize the need for careful history-taking [11].

One must consider the possibility of potential transmission in regions of nonendemicity. A retrospective chart review such as ours is limited by the inability to obtain further historical data, beyond that already recorded. It is possible that with more careful solicitation of the patients’ history, additional risk factors might have been elicited in this and other studies.

Patients included in this review were those with radiographically and clinically evident CNS involvement and compatible epidemiologic histories and/or whose serological tests were diagnostic (except for those who did not have radiographic features of neurocysticercosis). A high rate of false-positive diagnosis may have resulted from inclusion of patients from regions of endemicity, solely on the basis of serological findings (which can be diagnostic for as much as 3.6% of the Mexican population) [6]. The possible exclusion of patients with neurocysticercosis who have CNS lesions beneath the threshold of resolution of MRI or CT should be acknowledged.

Our series is consistent with previous studies that demonstrated that no signs or symptoms are pathognomonic for neurocysticercosis. An extensive review of 753 patients showed that the most common presenting symptoms of neurocysticercosis were seizures (52%) and those reflecting increased intracranial pressure (headache, nausea, and vomiting; 72%) [12]. A review of 131 children with neurocysticercosis in Mexico indicated that >70% presented with seizures and 50% with increased intracranial pressure [13].

We found less frequent papilledema than revealed in other studies, although these studies included few pediatric cases [12]. We did not identify any cases of neurocysticercosis encephalitis, a particularly severe form of the disease (with extensive inflammatory changes), in our series [14].

It is significant to note that all of our patients were afebrile. This suggests that physicians must consider neurocysticercosis in the differential diagnosis for previously healthy Hispanic children who have a history of afebrile focal seizure (unrelated to traumatic injury). We were unable to determine the percentage of patients who presented to our emergency department with afebrile seizures who were ultimately found to have neurocysticercosis. The apparently increasing prevalence of neurocysticercosis in the U.S. suggests that this percentage is probably increasing.

Our finding of normal peripheral WBC counts, without evidence of eosinophilia, is consistent with previous reports [15]. This is probably due to the lack of extensive soft-tissue migration by T. solium. None of our patients had the pleocytosis, increased protein level, or decreased glucose level that other investigators have reported. When Wright’s or Giemsa staining is done, CSF eosinophilia is reported in 10%-77% of cases of neurocysticercosis [2,12]. None of our CSF specimens were processed in this manner, so we cannot confirm this.

The finding of ova of T. solium in the stool of patients with neurocysticercosis is reported with wide variability. Richards and Schantz suggested that a single stool examination is positive for 50%-78% of tapeworm carriers [15]. Others have reported that the stool examination demonstrates ova in only zero to 4% of cases of neurocysticercosis [2]. Sorvillo et al. reported that the possibility of finding a tapeworm carrier among family contacts in locally acquired cases is significant (22.2%) [16].

A recently published recommendation regarding the diagnosis of neurocysticercosis in children suggests that stools from patients as well as from their family members should be examined in an effort to identify the source of infection [17]. This may be an important public health measure, although it is unclear whether the yield of such extensive examination is sufficient to justify the considerable effort and expense involved.

During the years covered by this retrospective review, serum and CSF were not consistently evaluated serologically for evidence of neurocysticercosis, and specimens were sent to a vari-

### Table 2. Outcome of neurocysticercosis, as established at patients’ last follow-up evaluations.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>All (n = 47)</th>
<th>Medically treated (n = 25)</th>
<th>Surgically treated (n = 9)</th>
<th>Not treated (n = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy; no medications prescribed</td>
<td>17 (36)</td>
<td>9 (36)</td>
<td>4 (44)</td>
<td>4 (31)</td>
</tr>
<tr>
<td>Healthy; anticonvulsants prescribed</td>
<td>25 (53)</td>
<td>13 (52)</td>
<td>3 (33)</td>
<td>9 (69)</td>
</tr>
<tr>
<td>Minor abnormalities*</td>
<td>4 (9)</td>
<td>2 (8)</td>
<td>2 (22)</td>
<td>0</td>
</tr>
<tr>
<td>Major abnormalities†</td>
<td>1 (2)</td>
<td>1 (4)</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

* Seizure disorder, speech/motor delay, or hemiparesis.
† Blindness.
ety of different laboratories that utilized widely varying methods. Lack of a reliably sensitive and specific immunologic assay has been a major hindrance to diagnosis of neurocysticercosis. Mitchell et al. found an elevated serum titer in five (31%) of 16 and an elevated CSF titer in only one (4%) of 24 children with neurocysticercosis [1].

Serum CF and indirect hemagglutination tests were reported to be diagnostic for ~50% of patients with seizures or intracerebral calcified lesions, 70% of those with meningitis, and 95% of those with increased intracranial pressure [18]. CSF titers determined by these methods are more often diagnostic for patients with meningitis or increased intracranial pressure due to neurocysticercosis [19]. It has been suggested that CSF titers are more likely to be positive in the rare cases of neurocysticercosis with CSF pleocytosis. Serological studies using these methods have been negative in ~50% of patients [18].

The ELISA method is more sensitive and specific than CF testing. However, its sensitivity is dependent on the clinical presentation. Overall, sensitivities of 65%–87% in serum and 62%–90% in CSF have been demonstrated [2]. Sensitivity of 75%–80% has been reported for patients with few or calcified cysts, viable intraparenchymal lesions, or relatively benign disease. Higher sensitivity has been noted for those with intraventricular lesions, meningitis, diffuse edema, or relatively malignant disease [15].

Most recently, the enzyme-linked immunoelectrotransfer blot (EITB) assay has been determined to be the serological test of choice, with reported 100% specificity and ≥90% sensitivity. However, these figures are achieved only in cases with at least 2 lesions. This method has been found to be more sensitive for testing of serum than of CSF, and paired specimens provide no additional sensitivity than serum testing alone [2, 15]. Because children most often present with a single lesion, the utility of this methodology for children may be limited. None of our patients had an EITB assay performed.

CT and MRI have dramatically improved the ability to detect neurocysticercosis lesions. Several studies have compared the efficacy of these two imaging methodologies [20, 21]. CT is generally superior to MRI in identifying parenchymal calcifications associated with neurocysticercosis. However, CT scanning is not sensitive in visualizing intraventricular cysts because of the similarity of the densities of cyst fluid and CSF. MRI is more sensitive than CT in demonstrating the presence of intraventricular cysts and in showing the extent of pericystic gliotic/edematous change that indicates activity, i.e., that the cyst is involving [20, 21] (figure 1A and 1B).

The results of our imaging studies indicate that both CT and MRI are necessary in the initial evaluation of children with neurocysticercosis. CT is more readily available than MRI and can be performed on an emergency basis. The CT should be performed with and without contrast. The noncontrast CT scan superbly delineates calcification (as small as 1 mm) and is the best radiologic modality to demonstrate homogeneously calcified lesions as well as nonenhancing lesions with eccentric and/or rim calcification [22]. Nonenhancing lesions with and without calcification were demonstrated in our patients at presentation, on 14 (31%) of 45 CT scans and seven (21%) of 33 MRI scans.

The contrast CT demonstrates ring or homogeneous enhancement of viable lesions. However, MRI provides better resolution than CT and is more sensitive in delineating viable lesions [23, 24]. Enhancing lesions were demonstrated in our patients at presentation on 37 (82%) of 45 CT scans and 29 (88%) of 33 MRI scans.

The MRI study should be performed as a complementary study to the CT. MRI demonstrates the intraventricular and subarachnoid (cisternal) lesions better than CT does [23]. An intraventricular lesion was demonstrated by MRI but not by CT in one of our patients.

Although greater than one-half of our patients were treated with praziquantel and dexamethasone, the use of this therapy is debatable. Many reports have addressed the controversial issue of whether or not patients with neurocysticercosis benefit from antihelminthic therapy [25–29]. Antihelminthic therapy is not indicated for patients with calcified lesions, as these lesions represent nonviable cysts that have already been destroyed by the host's immune response. Patients with viable, nonenhancing cysts should receive antihelminthic therapy because their immune system is not reacting to the parasite. Antihelminthic therapy does not appear to be beneficial for intraventricular cysts; surgery still appears to be the most effective treatment for these lesions [30].

The controversy regarding treatment concerns the correct approach to viable, enhancing cystic parenchymal lesions. Some authors believe that the use of antihelminthic agents does not modify the disease course, while others believe that treatment may actually be harmful to the patient. Mitchell and Crawford reported on 52 children with intraparenchymal lesions whose long-term outcomes were excellent despite the fact that they received no antihelminthic therapy [1].

However, a more recently reported study by Vasquez and Sotelo demonstrated clear benefit for those epileptic patients with neurocysticercosis who were treated with praziquantel [31]. The delayed regression of untreated lesions resulted in a higher incidence of granuloma formation, which was associated with a higher incidence of seizure activity. In addition, they reported fewer residual seizures in medically treated vs. surgically treated patients with intraparenchymal lesions. Del Brutto reported that a significant prognostic factor for seizure recurrence (after withdrawal of antiepileptic drugs) in patients with neurocysticercosis who were treated with albendazole was residual calcification [32].

None of the patients in this study were treated with albendazole, which is not currently approved by the U.S. Food and Drug Administration. Studies comparing the efficacy and tolerability of albendazole vs. praziquantel suggest that albendazole
is more effective than praziquantel in reducing the total number of cysts, as demonstrated on CT scanning, and results in an improved neurological outcome [33–35]. Adverse reactions secondary to the destruction of cysticerci can be seen with the use of albendazole, as with that of praziquantel.

Although none of our patients experienced any adverse reactions to therapy with praziquantel, it is important to acknowledge the limitations inherent in collecting that type of data in a retrospective study. The reactions that have been reported with use of albendazole and praziquantel are usually controlled by administration of dexamethasone, although there have been reports of death due to uncontrolled increased intracranial pressure. While the increased intracranial pressure is most often a consequence of the inflammatory reaction to a very large burden of cysts, this can be seen with as few as two lesions.

The concurrent administration of praziquantel and dexamethasone reduces plasma levels of praziquantel but does not appear to reduce levels of albendazole. It has been suggested, therefore, that the decision to use dexamethasone with praziquantel should be made only when moderate adverse reactions to therapy are noted [33–35]. In the case of treatment of giant subarachnoid cysticerci, corticosteroids must be used to avoid the hazard of the severe inflammatory reaction one can anticipate in the destruction of such lesions.

We were limited in our ability to assess whether there was a difference in long-term outcome for those patients treated with praziquantel and those who were not by the small sample size of the study, by their nonrandom management, and by the lack of follow-up for a sizeable number of these patients. However, we noted that seven children underwent craniotomy and lesional biopsy for intraparenchymal lesions that may have responded to antihelminthic medical therapy—or perhaps even without any medical therapy at all.

Establishing accurate diagnostic criteria for neurocysticercosis is somewhat difficult. To determine the sensitivity and specificity of various criteria requires availability of a “gold standard” with which to compare. Definitive diagnosis of neurocysticercosis can be made only with pathological confirma-
tion, but only in a minority of cases is the obtaining of a pathological specimen justified, primarily because of concern about alternative diagnoses. More sensitive and specific serological assays would increase the usefulness of serological results in the diagnosis of neurocysticercosis.

With these limitations, we evaluated the ability of various combinations of characteristics to identify patients in this study. A history of emigration from or travel to a region of endemicity, consistent radiographic findings, and afebrile seizures would have identified 29 (64%) of 45 patients. The addition of those who had recently had a visitor in the home from a region of endemicity would have included 35 (78%) of 45. A history of emigration from or travel to a region of endemicity, consistent radiographic findings, and afebrile focal seizures would have identified 25 (56%) of 45 of our patients, and adding the recent-visitor criterion would have identified 29 (64%) of 45.

Neurocysticercosis has become an important cause of afebrile seizures in children in the U.S. Both CT studies and limited-MRI studies are important modalities in the evaluation of children with neurocysticercosis. No readily available laboratory studies are sensitive for or predictive of the diagnosis of neurocysticercosis. The continued development of sensitive and specific diagnostic tests is very important.

Therapy appears to be well tolerated by children and results in an excellent long-term outcome. Larger prospective studies are needed to evaluate further the efficacy of antihelminthic therapy in children with neurocysticercosis.

References