

Epilepsy and neurocysticercosis in an Andean community

Marcelo E Cruz,^a Peter M Schantz,^b Ivan Cruz,^a Patricio Espinosa,^a Pierre-Marie Preux,^{c,d} Antonio Cruz,^e Washington Benitez,^a Victor CW Tsang,^b Julio Feroso^e and Michel Dumas^c

Background	<i>Taenia solium</i> neurocysticercosis (NCC) has been documented as one of the major causes of epilepsy in developing countries. However, methodological limitations have hindered the evaluation of the epidemiological relationship between cysticercosis and epilepsy at the community level.
Methods	We used the WHO protocol for epidemiological evaluation of neurological disorders to conduct a door-to-door survey among 2723 residents of San Pablo del Lago, an Ecuadorean rural community in which <i>T. solium</i> taeniasis/cysticercosis was known to be endemic. The WHO protocol was complemented by neuroimaging and immunological tests to confirm the diagnosis of this infection.
Results	In all 31 people suffering from active epilepsy were detected (prevalence 11.4 per 1000, 95% CI: 7.7–15.4); 26 agreed to undergo a computer tomography (CT) examination, and 28 agreed to have blood drawn for serodiagnosis. Fourteen of the 26 (53.8%) had CT changes compatible with NCC and six of the 28 (21.4%) tested positive in the enzyme-linked immunoelectro-transfer blot (EITB) assay. In a seizure-free random sample of this population, 17 of 118 (144 per 1000) subjects examined by CT and 10 out of 96 (104 per 1000) examined by EITB had evidence of this infection. The differences between the epilepsy group and the random sample of the population were statistically significant (OR = 6.93, 95% CI: 2.7–17.5, $P < 0.001$) for CT diagnosis, but not for EITB results (OR = 2.75, 95% CI: 0.8–7.1, $P > 0.12$, NS).
Conclusions	These findings confirm that <i>T. solium</i> NCC is a significant cause of epilepsy at the community level in Andean villages of Ecuador. It is important to initiate effective public health interventions to eliminate this infection, which may be responsible for at least half of the cases of reported epilepsy in Ecuador.
Keywords	Epilepsy, neurocysticercosis, developing countries, neuroepidemiology
Accepted	6 September 1998

Neurocysticercosis (NCC) (infection of the central nervous system by the larval stages of the pork taenia, *Taenia solium*) is a frequent cause of neurological disorders in many developing countries^{1–3} and is increasingly being reported in patients suffering from epilepsy in the US.^{4–6} The disease is common in communities where pigs are allowed to run loose, the residents consume insufficiently cooked pork, and basic sanitary services are lacking.²

^a Institute for Tropical Neurology, Unit of Neurosciences, Central University Schools of Medicine and Veterinary Sciences, Quito, Ecuador.

^b Division of Parasitic Diseases, NCID, Centers for Disease Control and Prevention, Atlanta, GA, USA.

^c Institute of Epidemiology and Tropical Neurology, Limoges, France.

^d Department of Biostatistics and Medical Informatics, School of Medicine, Limoges, France.

^e School of Medicine, University of Salamanca, Salamanca, Spain.

Reprint requests to: Dr Marcelo E Cruz, Instituto de Neurociencias, Av. Amazonas 4430, Quito, Ecuador.

Recently, with the introduction of improved techniques of immunodiagnosis and neuroimaging, a more precise picture of the role of NCC in the pathogenesis of neurological disorders in developing countries is emerging. Although the prevalence of human cysticercosis has been estimated between 3% (based on autopsy studies)⁷ and 9% (based on immunological data),⁸ its true prevalence and association with neurological disorders at the community level in disease-endemic villages is largely unknown. Although *T. solium* NCC has been linked with the high prevalence of epilepsy in developing countries,⁹ most of the reported studies are based on hospital populations.^{9–11} In these studies, up to 50% of epilepsy cases have cysticercosis as a putative factor.

The approach we used to screen a population known to be endemic for *T. solium* taeniasis/cysticercosis was to implement the WHO protocol for epidemiological investigation of neurological disorders.^{12–14} A serodiagnostic test (enzyme-linked immunoelectro-transfer blot, EITB)¹⁵ and a neuroimaging

procedure (computer tomography, CT)¹⁶⁻¹⁸ were added to strengthen the specificity of this instrument for the detection of *T. solium* infection and for quantifying its importance in the aetiology of epilepsy.

The present study is, as far as we know, the first to report on the combined use of neuroepidemiological, immunological and neuroimaging procedures to assess the magnitude and distribution of neurological disorders in a NCC-endemic community-based approach.

Patients and Methods

The study was conducted in San Pablo del Lago, an Andean village located 100 km north of Quito, the capital city of Ecuador. It was chosen because of the presence of known cases of epilepsy and human and porcine cysticercosis, as well as the predisposition of community members to participate in an operational research project.

A door-to-door census was performed by a paramedical team, with the help of the town's educational and civic organizations. A register was set up with one record for each family, containing data on age, gender, civil state, occupation, degree of education, and number of family members. An identification number was assigned to each *de facto* resident in April-May 1992. The application of the WHO protocol¹⁴ was then initiated. This tool, used to quantify headaches, epilepsy, cerebrovascular disorders, extrapyramidal syndromes and peripheral neuropathies, consisted of a structured interview and a task-based selective neurological examination.^{12,13} The interview was conducted by trained paramedical personnel, using a questionnaire translated from the above mentioned instrument, containing 12 questions and 10 tasks for individuals ≥ 7 years. The version for children < 7 years old contained 16 questions and no tasks. The language used was Spanish. For those few older people who did not understand spoken Spanish, a trained bilingual member of the survey team was always available for translation. The sensitivity and the specificity of this protocol had already been tested in different groups of patients and populations in similar geographical areas in Ecuador.⁹

A complete enumeration approach, based on each eligible individual, was adopted for data collection and identification of

affected individuals, and captured using Database III-Plus. The study was completed in three phases. In phase I, each individual eligible for inclusion in the survey was visited by the team. Individuals absent from their homes were asked to visit the local programme office for data completion. Written informed consent for participation in all phases was obtained from each head of household, and members of consenting families were interviewed individually for histories of neurological conditions.

In phase II, all individuals who answered at least one question positively or whose task-based examination yielded clear or doubtful abnormal status, were invited to undergo examination by trained neurologists under standard conditions. If a disease was confirmed, it was classified according to the International Classification of Diseases (ICD-9-NA).²⁰

In phase III, all individuals diagnosed with epilepsy on the grounds of anamnesis and neurological examination by two of the three neurologists ascribed to the programme were given an appointment for further studies. A prevalent case of active epilepsy was defined as a person who has had at least one epileptic seizure in the previous 5 years, regardless of antiepileptic drug (AED) treatment, as established in previous community-based studies,²¹ and conforming with the recommendations of the International League Against Epilepsy.²² The complementary examinations systematically included an awake routine EEG (done at the community clinic), plain CT scanning (done in Quito) and immunodiagnostic testing, EITB, for cysticercosis¹⁵ (done at the Centers for Disease Control and Prevention, Atlanta, Georgia, USA). In selected or doubtful cases, contrast infusion was used after plain CT examination, following the neuroradiologist's criteria.

The CT criteria for the diagnosis of neurocysticercosis was based on the extensive descriptions of this condition in the literature.^{16-18,23} Calcifications were seen on CT as small, round or elongated, high-density lesions in the brain parenchyma; cysts were described as rounded low-density lesions and the encephalitic phase was referred to as a low-density irregular area on plain CT which, after contrast infusion, displayed a nodular uptake.²⁴ Figure 1 depicts the classical tomographic findings in NCC.

Toxoplasmosis was diagnosed based on clinical grounds (presence of chorioretinitis and microcephaly) and on CT findings

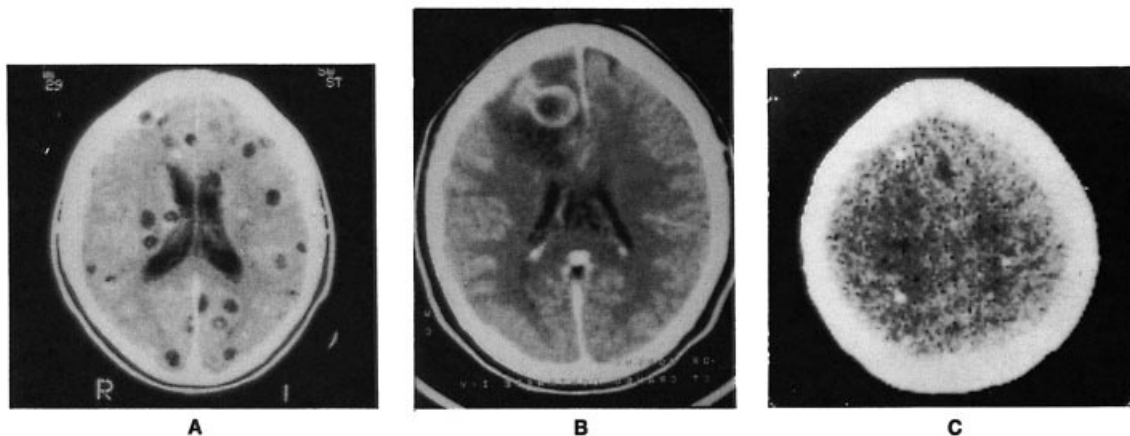


Figure 1 Computed tomographic findings in neurocysticercosis may be diverse according to the developmental phase of the parasite.^{23,24} (A) Multiple, viable intraparenchymatous cysts seen as rounded low-density areas. (B) Encephalitic-phase cysticercus in right frontal region presenting as a low-density area which, after contrast infusion, displays a nodular uptake. (C) Three calcifications of varying size are observed in both hemispheres

Table 1 Distribution by age and sex of the studied sample. San Pablo del Lago, Ecuador, 1994

Age (years)	Males	Females	Both sexes
0-9	324	283	607
10-19	322	341	663
20-29	162	228	390
30-39	138	179	317
40-49	91	138	229
50-59	84	97	181
60+	139	197	336
Total	1260	1463	2723

Table 3 Classification of epileptic seizures. San Pablo del Lago, Ecuador, 1994

	No. cases	%
A Generalized seizures	16	51.6
B Partial seizures	15	48.4
Simple partial	(1)	
Complex partial	(3)	
Partial with secondary generalization	(11)	
Total	31	100.00

Table 2 Prevalence and prevalence ratios (per 1000), by age and sex, of epilepsy, in San Pablo del Lago, Ecuador, 1994

Age (years)	Males		Females		Total	
	No.	Prevalence × 1000	No.	Prevalence × 1000	No.	Prevalence × 1000
0-9	3	9.3	4	14.1	7	11.5
10-19	3	9.3	4	11.7	7	10.6
20-29	3	18.5	1	4.4	4	10.3
30-39	2	14.5	3	16.8	5	15.8
40-49	0	0.0	2	14.5	2	8.7
50-59	1	11.9	2	20.6	3	16.6
60+	1	7.2	2	10.2	3	8.9
Total	13	10.3	18	12.3	31	11.4

(typical distribution of calcifications within the brain parenchyma, especially periventricular). Single calcifications were ascribed to possible neurocysticercosis after other types of granulomatous disease, mainly tuberculosis, were ruled out clinically.²⁵

Prevalence estimates were based on the population measured during the Holy Week of 1992, a well-remembered event in this community. Individuals in the denominator for prevalence calculations fulfilled conditions of eligibility (*de facto* resident in San Pablo del Lago during April–May, 1992 at least one day during the prevalence period), and met diagnostic criteria for epilepsy for at least one day of a similar time interval. A computer-generated random sample of seizure-free members of the population was also examined by CT (118 individuals) and EITB (96 individuals) to serve as control.

Fifty individuals, chosen randomly from the programme's register, unrelated to the computer-generated random sample, who screened negative on phase I where examined by the neurologists. No epilepsy was found in any of them.

Prevalence ratios for epilepsy and cysticercosis were obtained. Ninety-five per cent confidence intervals were calculated.²⁶ Two by two tables were constructed using this information and χ^2 and two-tailed *P* values were obtained using Epi Info 5.²⁷ Differences were considered statistically significant if *P* < 0.05.

Results

The distribution by age and sex of the studied population was typical of developing communities with young people under 20 years of age comprising about 50% of the total (Table 1).

To document the infection in local swine, 80 pigs were slaughtered at the outset of the programme; *T. solium* cysticerci were found in the muscles of seven, giving a prevalence ratio of 8.75% (95% CI: 3.5–18).

Complete information was collected from 2723 individuals living in the urban nucleus of San Pablo del Lago. At the end of phase I, 578 individuals had at least one survey question or one neurological test yielding clear or questionable abnormal status. All these people were evaluated by neurologists at the local programme office or, in a few cases, at their homes. There was no loss of participants between phases I and II. At the end of phase II, 31 individuals were confirmed as suffering from active epilepsy as defined previously.^{21,22} Thus, the point prevalence ratio was 11.4 per 1000 (95% CI: 7.7–15.4). The number of cases and the prevalence ratios by age and sex of the 31 patients are shown in Table 2.

Table 3 shows the classification of these cases according to seizure type. Seizures in 16 patients (51.6%) were classified as generalized and in 15 (48.4%) as partial. All were invited to undergo further examination at the clinical centre in Quito for completion of ancillary investigations for epilepsy, but five refused CT examinations and three refused to have blood drawn for immunological testing. The CT findings in those investigated by this method and their corresponding EITB results are presented in Table 4.

Among 26 people with epilepsy who agreed to be examined, 14 (53.8%) had CT evidence of past or recent NCC infection. Of these 14, 5 (37.5%) had generalized seizures, and 9 (64.3%) had partial seizures.

Table 4 Summary of computer tomography (CT) and enzyme-linked immunoelectro-transfer blot (EITB) findings in 26 patients with epilepsy. San Pablo del Lago, Ecuador, 1994

	CT +	EITB +
Neurocysticercosis	14	5
Single intracranial calcification	(4)	(0)
Multiple intracranial calcifications	(5)	(1)
Single cyst	(1)	(1)
Multiple cysts + calcifications	(2)	(1)
Multiple cysts + single encephalitic phase	(1)	(1)
Multiple encephalitic phases + calcifications	(1)	(1)
Toxoplasmosis	2	0
Cortical atrophy	2	0
Normal	8	1
Total	26	6

Table 5 Age of onset of epilepsy and computer tomography (CT) and enzyme-linked immunoelectro-transfer blot (EITB) findings. San Pablo del Lago, Ecuador, 1994

Age at onset (years)	No.	CT ^a	NCC ^b	% NCC	EITB ^c	EITB +	% +
<1	6	4	1	25%	6	0	0%
1-5	4	4	2	50%	4	0	0%
6-12	5	4	2	50%	4	2	50%
13-19	3	3	2	67%	3	1	33%
20+	13	11	7	64%	11	3	27%
Total	31	26	14	54%	28	6	21%

^a Number who had CT performed.

^b Number with CT evidence for NCC.

^c Number tested by EITB.

In 11 of the 26 with epilepsy, their epilepsy had first begun when they were older than 20 years. In this group with late-onset epilepsy, NCC was found in seven cases (64%). Table 5 shows the correlation of age of onset of seizures with CT and EITB findings.

The EITB was positive in only 6 of 28 (21.4%) people with epilepsy; these included three of six males (50%) and two of eight females (25%) who also had CT findings consistent with the diagnosis of NCC. In total, approximately one-third of epilepsy patients with NCC diagnosed by CT had a concomitant positive EITB (5/14, 35.7%). Four of five people studied by CT and showing single or multiple apparently viable cysts had a positive EITB (80%), whereas only one of nine who displayed single or multiple calcifications had a positive EITB (11%) (Table 4). One other patient with epilepsy had a positive EITB with a negative CT.

Among the seizure-free random sample of the population, 118 CT examinations were completed. Of these, 17 showed findings suggestive of cysticercosis, yielding a prevalence of 144 per 1000 (95% CI : 85-212). In the same sample, it was possible to obtain serum specimens for EITB from 96 people, and 10, or 104 per 1000 (95% CI : 52-167) were positive.

Chi-square analysis was performed to test the association of epilepsy and NCC by comparing results of CT scanning and serological tests in the epilepsy-free random sample of the population with those in the epilepsy cases (Table 6). A statistically

Table 6 Two by two table for epilepsy and computer tomography (CT) diagnosis of neurocysticercosis (NCC)

	Epilepsy +	Epilepsy -	Total
CT + for NCC	14	17	31
CT - for NCC	12	101	113
Total	26	118	144

OR = 6.93, 95% CI : 2.7-17.5, $P < 0.001$.

significant difference (OR = 6.93, 95% CI : 2.7-17.5, $P < 0.001$) was found when comparing the CT diagnosis, but not the EITB diagnosis of NCC (OR = 2.75, 95% CI : 0.8-7.1, $P > 0.12$, NS), using Mantel-Haenzel statistics (Table 6).

Discussion

The WHO protocol for epidemiological studies of neurological disorders^{12-14,19} is a useful tool for screening for the neurological morbidity associated with *T. solium* taeniasis/cysticercosis.²⁸

The present study confirms the high prevalence figures for epilepsy reported in developing countries using the WHO protocol¹⁹ and indicates that *T. solium* NCC accounts for a major fraction of the condition in Ecuador. In such regions up to two-thirds of late onset epilepsy cases may be due to this infection. Although the number of epilepsy cases is not large, it seems that women become ill at an earlier age and in higher proportions, probably due to food handling activities and their relationship with cysticercal infection (Table 2).⁶

If we correct the epilepsy ratio found in this study by eliminating those cases linked to cysticercal infection, the remaining figure (17 patients, i.e. 6.2 per 1000), is almost identical to that reported for Rochester, Minnesota (6 per 1000).²⁹ Analysis of these same data for the association of NCC with migraine-type headaches in this community also confirmed an important causal association.²⁸

Table 4 shows that EITB positivity correlates well with the presence of viable parasitic cysticerci within the central nervous system, but poorly in those with CT findings suggestive of dead cysts (Tables 4 and 6). The EITB, or immunoblot, assay is a specific and sensitive diagnostic assay for infection by larval *T. solium*.¹⁵ In patients with clinical diagnoses of NCC, the most important factors determining sensitivity are numbers and stages of development of cysticerci.³⁰ In patients with multiple viable lesions, the test has 90% sensitivity, whereas in patients with only calcified cysts, sensitivity is reduced. The relatively low sensitivity of EITB for supporting the CT evidence for NCC in this study is most likely explained by the long-standing chronic nature of NCC in these patients, in which pathological changes evident by CT imaging persist after the cysticerci are no longer viable and producing immune stimulation; consequently antibodies were no longer detectable.³¹

Patients with parenchymal NCC typically present with seizures.^{4,9-11} In this study two out of three patients with epilepsy secondary to NCC presented with partial seizures. Although anticonvulsants are routinely prescribed, Latin American clinicians advocate the concomitant use of anti-parasitic drugs and steroids where there is evidence of active NCC.^{23,32,33} In fact, it seems that there is a decrease in seizure frequency in patients so treated.^{34,35} However, more detailed,

longer term, placebo-controlled clinical trials are needed in order to ascertain beyond doubt the clinical value of antiparasitic drugs in parenchymal NCC.

Among a random sample from the community with no history of seizures, approximately 14% had CT findings compatible with NCC and approximately 10% had immunological evidence of larval *T. solium* infection. Although the rate of neuroimaging findings was significantly less than in people with histories of seizures (Table 6), it does emphasize that many people with intracerebral cysticercosis do not experience symptoms from their infection.⁷ These findings illustrate some of the difficulties of interpreting the results of neuroimaging studies and serological tests for cysticercosis in populations highly exposed to infection with this cestode zoonosis.

The radiological and immunodiagnostic data presented confirm that NCC is the major definable risk cause of epilepsy in geographical areas where *T. solium* infection is endemic. *T. solium* NCC is a preventable and treatable infection and all efforts should be made by responsible governments in order to reduce and ultimately eliminate this disease from countries where it is endemic.^{36,37} In doing so, a sizable amount of epilepsy burden and consequent human suffering will be alleviated.

References

- 1 Botero D, Tanowitz HB, Weiss LM, Wittner M. Taeniasis and cysticercosis. *Infect Dis Clin North Am* 1993;**7**:683–97.
- 2 World Health Organization. *Guidelines for Surveillance Prevention and Control of Taeniasis/Cysticercosis*. WHO document VPH/83.49, Geneva, 1983.
- 3 Preux PM, Melaku Z, Druet-Cabanac M *et al*. Cysticercosis and neurocysticercosis in Africa: current status. *Neur Infect Epidemiol* 1996;**1**:63–68.
- 4 Latoviski N, Abrams G, Clark C *et al*. Cerebral cysticercosis. *Neurology* 1978;**28**:838–42.
- 5 White AC. Neurocysticercosis: a major cause of neurological disease worldwide. *Clin Infect Dis* 1997;**24**:101–15.
- 6 Schantz PM, Moore AC, Muñoz JL *et al*. Neurocysticercosis in and Orthodox Jewish Community in New York City. *N Engl J Med* 1992;**327**:692–95.
- 7 Guerrero F. Cisticercosis cerebral: hallazgos necrópsicos. *Rev Ecuat Med Cien Biol* 1965;**3**:142–50.
- 8 Sarti E, Schantz PM, Plancarte A *et al*. Prevalence and risk factors for *Taenia solium* taeniasis and cysticercosis in humans and pigs in a village in Morelos, Mexico. *Am J Trop Med Hyg* 1992;**46**:677–85.
- 9 Medina MT, Rosas E, Rubio-Donnadieu F, Sotelo J. Neurocysticercosis as the main cause of late-onset epilepsy in Mexico. *Arch Intern Med* 1990;**150**:325–27.
- 10 Garcia HH, Gilman R, Martinez M *et al*. Cysticercosis as a major cause of epilepsy in Peru. *Lancet* 1993;**341**:197–200.
- 11 Del Brutto OH, Santibanez R, Noboa CA *et al*. Epilepsy due to neurocysticercosis: analysis of 203 patients. *Neurology* 1992;**42**:389–92.
- 12 Schoenberg BS. Clinical neuroepidemiology in developing countries. *Neuroepidemiology* 1982;**1**:137–42.
- 13 Meneghini F, Rocca WA, Rigoletto F *et al*. Door-to-door prevalence study of neurological diseases in a Sicilian population. *Neuroepidemiology* 1991;**10**:70–85.
- 14 World Health Organization, Neurosciences Programme. *Research Protocol for Measuring the Prevalence of Neurological Disorders in Developing Countries*. Geneva: WHO, 1991.
- 15 Tsang VCW, Brand JA, Boyer AE. An enzyme-linked immunoelectrotransfer blot assay and glycoprotein antigens for diagnosing human cysticercosis (*Taenia solium*). *J Infect Dis* 1989;**159**:50–59.
- 16 Herrera G. Diagnostico por Tomografia Axial Computarizada en neurocysticercosis. In: Garcia HH, Martinez SM (eds). *Taeniasis/cysticercosis por T. solium*. Lima: Ed Universo, 1996, pp.79–84.
- 17 Rodriguez-Carbajal J, Palacios G, Zee CH. Neuroradiology of cysticercosis of the central nervous system. In: Palacios E, Rodriguez-Carbajal J, Taveras J (eds). *Cysticercosis of the Central Nervous System*. Springfield: Charles C Thomas, 1983, pp.101–43.
- 18 Byrd SHE, Locke GE, Biggers S *et al*. The computed tomography appearance of cerebral cysticercosis in adults and children. *Radiology* 1982;**144**:819–23.
- 19 Cruz ME, Schoenberg BS, Ruales J *et al*. Pilot study to detect neurologic disease in Ecuador among a population with a high prevalence of endemic goiter. *Neuroepidemiology* 1985;**4**:108–11.
- 20 World Health Organization. *Application of the International Classification of Diseases to Neurology*. Geneva: WHO, 1987.
- 21 Cruz ME, Barberis P, Schoenberg BS. The epidemiology of epilepsy. In: Poeck K, Freund HJ, Ganshirt H (eds). *Neurology. Proceedings of the XIII World Congress of Neurology*. Berlin: Springer Verlag, 1986, pp.229–39.
- 22 Commission on epidemiology and prognosis, International League Against Epilepsy. Guidelines for epidemiologic studies on epilepsy. *Epilepsia* 1993;**34**:592–96.
- 23 Kramer LD, Locke G, Byrd SE. Cerebral cysticercosis: documentation of natural history by CT. *Radiology* 1989;**171**:459–62.
- 24 Cruz M, Cruz I, Horton J. Albendazole versus praziquantel in the treatment of cerebral cysticercosis: clinical evaluation. *Trans Roy Soc Trop Med Hyg* 1991;**85**:244–47.
- 25 Del Brutto AH, Wadia N, Dumas M *et al*. Proposal of diagnostic criteria for human cysticercosis and neurocysticercosis. *J Neur Sci* 1996;**142**:1–6.
- 26 Schoenberg BS. Calculating confidence intervals for rates and ratios. *Neuroepidemiology* 1983;**2**:257–65.
- 27 Centers for Disease Control. *Epi Info Version 5.01b, July, 1991*. Atlanta, GA.
- 28 Cruz ME, Cruz I, Preux PM *et al*. Headache and cysticercosis in Ecuador, South America. *Headache* 1995;**35**:93–97.
- 29 Hauser WA, Kurland LT. The epidemiology of epilepsy in Rochester, Minnesota, 1935 through 1967. *Epilepsia* 1975;**16**:1–66.
- 30 Wilson M, Bryan RT, Fried JA *et al*. Clinical evaluation of the cysticercosis enzyme-linked immunoelectrotransfer blot in patients with neurocysticercosis. *J Infect Dis* 1991;**164**:1007–09.
- 31 Garcia HH, Gilman RH, Catacora M *et al*. Serologic evolution of neurocysticercosis patients after antiparasitic therapy. *J Infect Dis* 1997;**175**:486–89.
- 32 De Ghetaldi LD, Norman RM, Couville AW. Cerebral cysticercosis treated biphasically with dexamethazone and praziquantel. *Ann Intern Med* 1983;**99**:179–81.
- 33 Cruz I, Cruz ME, Carrasco F, Horton J. Neurocysticercosis: optimal dose treatment with albendazole. *J Neurol Sci* 1995;**133**:152–54.
- 34 Vazquez V, Sotelo J. The course of seizures after treatment for cerebral cysticercosis. *N Engl J Med* 1992;**327**:696–701.
- 35 Del Brutto AH. Prognostic factors for seizure recurrence after withdrawal of antiepileptic drugs in patients with neurocysticercosis. *Neurology* 1994;**44**:1706–09.
- 36 Cruz M, Davis A, Dixon H, Pawlowski ZS, Proano J. Operational studies on the control of *Taenia solium* taeniasis/cysticercosis in Ecuador. *Bull World Health Organ* 1989;**67**:401–07.
- 37 Schantz PM, Cruz M, Sarti E, Pawolwski Z. Potential eradicability of taeniasis and cysticercosis. *Bull Pan Am Health Organ* 1993;**27**:397–401.

