

have been discussed elsewhere¹ and will be further defined as this promising procedure is subjected to clinical trials that take into account important variables such as the extent of disease, imaging signs of instability, the location of the metastasis within the spinal canal, neurologic function, and tumor type.

Dr. Charrette correctly emphasizes rehabilitation. I regret that space limitation prevented me from discussing this important topic.

In patients with presumed spinal cord involvement from metastases, Drs. Newman and Bonheim recommend unenhanced CT without myelography, followed by radiation therapy if the CT demonstrates a lesion consistent with the clinical situation (when MRI is unavailable). Many epidural metastases may be missed if this protocol is used in patients with clinical manifestations of metastatic epidural compression, since imaging the entire spinal axis is not routinely feasible with CT² and, unless an intrathecal contrast agent is used, definitive visualization of the contents of the spinal canal is not achieved.³ In a study of the sensitivity of spinal CT without an intrathecal contrast agent for the diagnosis of epidural tumor, Weissman et al.⁴ found that radiation-therapy ports were often inadequate if treatment was based on the results of CT without myelography. This finding, along with a 38 percent incidence of synchronous noncontiguous epidural metastases, led these authors to emphasize "the need for careful myelographic documentation of the extent of tumor before instituting treatment." Redmond and colleagues⁵ have similarly concluded that when spinal CT reveals cortical-bone discontinuity around the neural canal or soft-tissue mass, CT with myelography should be performed. In patients with clinical manifestations of metastatic epidural compression, unless it is otherwise contraindicated, imaging of the entire spinal axis by MRI or myelography is necessary to optimize radiotherapy and planning for surgical treatment, because symptomatic epidural metastases at other unsuspected levels may go unrecognized.

THOMAS N. BYRNE, M.D.
Yale University
School of Medicine
New Haven, CT 06511

1. Siegal T, Siegal T. Current considerations in the management of neoplastic spinal cord compression. *Spine* 1989;14:223-8.
2. Kambholtz R, Sze G. Current imaging in spinal metastatic disease. *Semin Oncol* 1991;18:158-69.
3. Beltran J, Noto AM, Chakeres DW, Christoforidis AJ. Tumors of the osseous spine: staging with MR imaging versus CT. *Radiology* 1987;162:565-9.
4. Weissman DE, Gilbert M, Wang H, Grossman SA. The use of computed tomography of the spine to identify patients at high risk for epidural metastases. *J Clin Oncol* 1985;3:1541-4.
5. Redmond J III, Friedl KE, Cornett P, Stone M, O'Rourke T, George CB. Clinical usefulness of an algorithm for the early diagnosis of spinal metastatic disease. *J Clin Oncol* 1988;6:154-7.

of endemic disease, we know that an asymptomatic interval (mean, 4.8 years) precedes symptomatic disease.² This asymptomatic period corresponds to the expected life span of tissue cysts. Only on degeneration or death of the cyst do symptoms appear. Pathological studies, primarily from Mexico, have confirmed that cysts from subjects with symptoms are surrounded by an inflammatory infiltrate; cysts from patients who died from other causes do not show inflammation.^{3,4} Our experience in Houston has been that seizures are largely confined to patients who have cysts with associated inflammation or residual calcifications from previous infection.⁵ One of the few cases of a parenchymal cyst without inflammation involved a patient with an asymptomatic cyst found during an evaluation of metastatic cancer. Furthermore, laboratory studies confirm that taenia cysts have elaborate mechanisms to suppress the host inflammatory response.⁴ Thus, for patients in groups 1 and 2, the cysts would probably have died within a few years regardless of cysticidal therapy.

Second, we are concerned about the validity of the comparisons between the treatment and control groups. Vazquez and Sotelo compared patients enrolled in open-label trials with those excluded. Extensive experience from clinical trials has demonstrated that regardless of therapy, enrollment in trials is associated with a better prognosis than routine care. Enrollment selects for motivated patients and increases the patient's compliance and the physician's attentiveness. Variation in compliance with anticonvulsant therapy may explain the differences in the groups noted by the authors. This is of special concern with cysticercosis in the light of the benign natural history and prognosis of parenchymal disease (as opposed to the more severe morbidity in ventricular and cisternal disease).^{2,5,6} Praziquantel and albendazole are clearly effective at killing cysts. We think, however, that controlled trials are still needed to clarify which patients benefit clinically from cysticidal therapy.

A. CLINTON WHITE, JR., M.D.
WAYNE X. SHANDERA, M.D.
Baylor College of Medicine
Houston, TX 77030

1. Vazquez V, Sotelo J. The course of seizures after treatment for cerebral cysticercosis. *N Engl J Med* 1992;327:696-701.
2. Dixon HB, Lipscomb FM. Cysticercosis: an analysis and follow-up of 450 cases. *Med Res Spec Rep (Lond)* 1961;299:1-58.
3. Ridausa Sanz C. Host response in childhood neurocysticercosis: some pathological aspects. *Childs Nerv Syst* 1987;3:206-7.
4. White AC Jr, Tatro P, Molinari JL. Host-parasite interactions in *Taenia solium* cysticercosis. *Infect Agents Dis* 1992;1:185-93.
5. Shandera WX, Armstrong R, Tan J, White AC Jr. Cysticercosis in Harris County, Texas. Presented at the 30th Annual Meeting of the Infectious Diseases Society of America, Anaheim, California, 1992. abstract.
6. Bandres J, White AC Jr, Samo T, Murphy E, Harris R. Extraparenchymal neurocysticercosis: report of five cases and review of the literature on management. *Clin Infect Dis* 1992;15:799-811.

Dr. Sotelo replies:

To the Editor: The classic study by Dixon and Lipscomb published in 1961 reported a mean period between infection and symptoms.¹ The assumption that this period corresponds to the life span of the parasite is merely speculative, since only with imaging studies, unavailable at that time, could this span be determined.² Cysticercosis may become symptomatic at any stage of the disease³; otherwise, patients receiving therapy for noninflammatory cysts could not be found, whereas in fact these patients do enter therapeutic trials.⁴ It is possible that a cysticercus dies spontaneously

SEIZURES AND TREATMENT FOR CEREBRAL CYSTICERCOSIS

To the Editor: We have two areas of concern about the data on cysticercosis presented by Vazquez and Sotelo (Sept. 3 issue).¹ First, the findings they report for groups 1 and 2 (patients with lesions without inflammation) are inconsistent with other data on the pathogenesis and natural history of the disease. Cysts within the brain parenchyma have a limited life expectancy. From natural-history studies among British expatriates who have spent a limited time in an area

within a few years, but there is no reason to let an infectious disease follow its natural course when an effective therapy exists. Our study showed that the long-term persistence of the parasite in the brain increases the chances of persisting epilepsy. Our results indicated that the reduction in the frequency of seizures was related to cysticidal therapy, rather than to anticonvulsant medication.⁵

JULIO SOTELO, M.D.

14269, Mexico City, National Institute of Neurology
Mexico and Neurosurgery

1. Dixon HB, Lipscomb FM. Cysticercosis: an analysis and follow-up of 450 cases. *Med Res Spec Rep (Lond)* 1961;299:1-58.
2. Rodríguez-Carbajal J, Saigado P, Gutiérrez-Alvarado R, Escobar-Izquierdo A, Aruffo C, Palacios E. The acute encephalitic phase of neurocysticercosis: computed tomographic manifestations. *AJNR Am J Neuroradiol* 1983;4:51-5.
3. Del Brutto OH, Sotelo J. Neurocysticercosis: an update. *Rev Infect Dis* 1988; 10:1075-87.
4. Takayanagui OM, Jardim E. Therapy for neurocysticercosis: comparison between albendazole and praziquantel. *Arch Neurol* 1992;49:290-4.
5. Vazquez V, Sotelo J. The course of seizures after treatment for cerebral cysticercosis. *N Engl J Med* 1992;327:696-701.

HORMONE-REPLACEMENT THERAPY AND PULMONARY LEIOMYOMATOSIS

To the Editor: I wish to describe how recurrent pneumothorax led to the diagnosis of pulmonary leiomyomatosis in a young woman receiving hormone-replacement therapy. Uterine leiomyomas found in the lung were first reported by Steiner.¹ The phenomenon is rarely reported, but its true incidence is not known. Decreased lung function and spontaneous pneumothorax have been attributed to pulmonary leiomyomas associated with the use of oral contraceptives,^{2,3} but no association with hormone-replacement therapy has been reported.

A 32-year-old woman presented to her physician to obtain refills of her prescriptions for antihypertensive medication (a combination of hydrochlorothiazide and triamterene) and conjugated estrogens. She had undergone a hysterectomy and bilateral oophorectomy in 1987 after earlier operations for infertility and pelvic pain. Uterine fibroids had been noted incidentally at the time of her hysterectomy, and treatment with conjugated estrogens (Premarin, 0.625 mg) was started at that time. The patient stated that a "breast cyst" was removed in 1981. Her hormone-replacement regimen was changed to conjugated estrogens (0.625 mg per day) on days 1 to 25 of the month and medroxyprogesterone (10 mg per day) on days 16 to 25 of the month, to address concern about unopposed estrogen stimulation of her breast tissue.

Starting with the very next cycle and during the next 26 months, five unexplained episodes of pneumothorax occurred. In April 1991 a thoracotomy and wedge biopsy of the lung were performed. Multiple gray nodules 1 to 3 mm in diameter were found close to the parietal surfaces of the upper and lower lobes of the lung; Figure 1 shows that the tumors were very close to the parietal surface. On pathological examination, these nodules were interpreted as benign pulmonary fibroleiomyomas.

The patient was advised to stop her hormone-replacement therapy. Remission of this condition has been reported after oophorectomy.⁴ The patient had subjective improvement within three months, and to date there has been no further pneumothorax.



Figure 1. Wedge-Biopsy Specimen of Lung, Showing the Pleural Surface (Arrow), a Leiomyoma (A), and Normal Tissue (B).

This patient had recurrent pneumothorax presumably due to hormonal stimulation of latent pulmonary leiomyomas. These appear to have been discovered after progesterone was added to her treatment or cyclic hormone-replacement therapy was begun. Pulmonary leiomyomas and recurrent pneumothorax may be complications of hormone-replacement therapy.

JOHN MARON THOMAS, M.D.

Cleveland, OH 44111

Fairview General Hospital

1. Steiner PE. Metastasizing fibroleiomyoma of the uterus: report of a case and review of the literature. *Am J Pathol* 1939;15:89-109.
2. Stephenson CA, Henley FT, Goldstein AR. Benign metastasizing leiomyoma. *Ala J Med Sci* 1984;21:78-81.
3. Clark DH, Weed JC. Metastasizing leiomyoma: a case report. *Am J Obstet Gynecol* 1977;127:672-3.
4. Banner AS, Carrington CB, Emory WB, et al. Efficacy of oophorectomy in lymphangioliomyomatosis and benign metastasizing leiomyoma. *N Engl J Med* 1981;305:204-9.

THE AMERICAN HEALTH CARE SYSTEM — MANAGED CARE

To the Editor: In his article on managed care (Sept. 3 issue),^{*} Iglehart discusses the challenge of identifying cost-effective physicians. Whereas many debate the appropriateness of selecting physicians on the basis of the numbers, managed-care entities, out of necessity, are using such criteria to refine their provider networks.

Selecting cost-efficient providers requires data from which payers can make informed decisions. There are two sources of data that would make the compilation of statistically significant averages possible: statewide discharge data on hospital inpatients and data on claims history from payers. Although the latter can include extensive data about inpatient and outpatient activities, most payers cannot use this

*Iglehart JK. The American health care system — managed care. *N Engl J Med* 1992;327:742-7.