

Correspondence



Left Ventricular Assist Device

To the Editor: Rose et al. (Nov. 15 issue)¹ performed a seminal trial of the use of a left ventricular assist device for end-stage heart failure. However, to gauge the full extent of the effect of invasive therapy with a left ventricular assist device on the health-related quality of life, missing data must be accounted for (i.e., subjects who died between administrations of the questionnaires measuring health-related quality of life or who are alive but did not participate in serial evaluations).² For example, among other statistical techniques, the investigators could assign the “worst possible score” to those who died or, even though it may be difficult, impute a score on the basis of correlations between prior responses and clinical status. Furthermore, the way in which the health-related quality of life is measured should be delineated, since the method and timing of the administration of questionnaires may affect the results.³ Since mortality remains high in the group with the left ventricular assist device (77 percent at two years), the argument for the use of mechanical therapy may very well rest on its effect on the health-related quality of life.

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To the Editor: The value of angiotensin-converting-enzyme (ACE) inhibitors, beta-blockers, and spironolactone has been well established by the results of numerous clinical trials. About 70 percent of the patients described by Rose et al. were treated with ACE inhibitors or angiotensin II-receptor antagonists; 35 to 40 percent received spironolactone, and only about 20 percent received beta-blockers. Thus, this population cannot have been considered to be optimally treated from the point of view of medical therapy.

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To the Editor: In the study by Rose et al., the use of a left ventricular assist device in patients with advanced heart failure resulted in a survival benefit and an improved quality of life. It is noteworthy that the most frequent cause of death among patients in the group with the left ventricular assist device was sepsis. Although the characteristics of these infections are not described, data^{1,2} from the use of a left ventricular assist device as a bridge to transplantation suggest that the incidence and severity of infections in patients receiving support with these devices are proportional to the duration of mechanical circulatory support.

More recently, our report³ of infections in patients with extended support with left ventricular assist devices demonstrates an emerging spectrum of serious infections. The repeated bacteremic episodes in these patients, despite long courses of antibiotics, are suggestive of persistent endovascular infection resembling endocarditis. In addition, the relapsing bacteremia may have arisen from the drive lines of the left ventricular assist devices. The possible contribution of drive-line infections to the development of relapsing bacteremia and endocarditis associated with left ventricular assist devices in the course of extended circulatory support cannot be underestimated. Both conditions are difficult to eradicate with antibiotics and may require a change of the device, increasing morbidity and mortality among these

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patients. Perhaps the recent development of drive lines impregnated with antimicrobial agents may prevent the colonization of the drive lines and facilitate ingrowth of tissue to provide long-term stability and protection for patients receiving extended circulatory support with left ventricular assist devices.⁴

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To the Editor: The randomized study by Rose and colleagues demonstrated that a left ventricular assist device improves survival and quality of life in adults with advanced heart failure. The authors do not mention the use of assist devices in children.

We successfully implanted miniaturized pulsatile devices with stroke volumes of 10 to 60 ml (Berlin Heart, Berlin, Germany) in 45 infants and children from 1990 through 2001^{1,2} and are impressed by the results in patients with myocarditis or cardiomyopathy (Table 1). All children presented with cardiogenic shock and multiorgan failure after

repeated resuscitation, and death was expected within hours. Of the 28 children, 6 received a left ventricular assist device and 22 a biventricular assist device. Although the majority of the children with myocarditis were weaned from ventricular support after recovery of their own heart, in most children with cardiomyopathy such support was used as a bridge to transplantation.

Children often die before transplantation because of the shortage of organs, and frequently no recipient of appropriate size is found when a child's heart becomes available. We expect that children with end-stage heart failure will benefit from mechanical cardiac support and that fewer organs from young donors will be lost.

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The authors reply:

To the Editor: We agree with Hauptman's contention that issues of health-related quality of life are crucial to "the argument for the use of mechanical therapy" for patients with end-stage heart failure. We disagree, however, with his suggestion of assigning a "worst possible score" to those who died, because we believe that the health-related quality of life of a person who has died is not some low number; it is undefined. On the other hand, imputing a low score to truly missing observations from survivors seems sensible, because missing data often connote a poor health-related quality of life. Applying this strategy to the data from the Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure (REMATCH) trial (Table 3 in our article) would accentuate the advantage of left ventricular assist devices, because the fraction of observations missing is larger in the group receiving optimal medical management. Our report did not enumerate all the details of our methods and measurement schedule for assessing the health-related quality of life, which we have described elsewhere.¹

Jiménez-Navarro and colleagues argue that the low frequency of use of beta-blockers, ACE inhibitors, and spironolactone at base line in the medically treated group of the REMATCH trial reflects suboptimal medical therapy, because recent trials reported a survival benefit from these agents in patients with "severe heart failure."²⁻⁴ As Braunwald pointed out, however, the Carvedilol Prospective Randomized Cumulative Survival trial (of beta-blockers) excluded patients with "extremely severe heart failure" — the target population for the REMATCH trial.^{2,5} Similarly, the Randomized Aldactone Evaluation trial (of spironolactone) and the Cooperative North Scandinavian Enalapril Survival Study (of ACE inhibitors) also focused on less severely ill patients

TABLE 1. CHARACTERISTICS OF CHILDREN WITH MYOCARDITIS OR CARDIOMYOPATHY WHO RECEIVED PNEUMATIC PULSATILE VENTRICULAR ASSIST DEVICES.

CHARACTERISTIC	PATIENTS WITH MYOCARDITIS (N=7)	PATIENTS WITH CARDIOMYOPATHY (N=21)
Age (yr)		
Median	1.3	12.2
Range	0.01–14	0.5–15
Body weight (kg)		
Median	10.2	30.0
Range	2–81	5–64
Duration of support (days)		
Median	11	14
Range	2–22	1–98
Outcome (no.)		
Heart transplantation	1	13
Weaning from support	4	1
Death	2	7

than those we studied. In the REMATCH trial, dependency on inotropic therapy was 70 percent at base line, and patients who were not dependent at base line had markedly reduced oxygen consumption (average peak consumption, 9.18 ± 1.98 ml per kilogram of body weight per minute). Few of these patients were subsequently able to tolerate ACE inhibitors, let alone beta-blockers or spironolactone.

Vilchez et al. correctly point out the "emerging spectrum of serious infections" to which patients with left ventricular assist devices are susceptible. We agree that persistent bacteremia may necessitate removal or replacement of the device, whereas the use of drive lines impregnated with antibiotics or the use of fully implantable devices may reduce the incidence of this dreaded complication.

Stiller et al. describe excellent results from the short-term use of assist devices in children as a bridge to transplantation or to recovery from acute myocarditis. Although neither their experience nor the REMATCH trial addresses the long-term use of devices in children, this critical area deserves future investigation.

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Goal-Directed Therapy for Severe Sepsis

To the Editor: Rivers et al. (Nov. 8 issue)¹ report on goal-directed therapy for severe sepsis and septic shock. Although their findings are interesting and provocative, they should be interpreted with caution. The end points of therapy and the treatment approaches used are somewhat troubling. The central venous pressure is a poor indicator of intravascular volume.² Furthermore, mixed venous oxygen saturation (SMVO₂), and, by inference, central venous oxygen saturation (ScVO₂), are poorly correlated with cardiac output and indexes of tissue oxygenation.^{2,3} The use of packed cells as first-line therapy to increase the ScVO₂ in the patients assigned to early goal-directed therapy is worrisome. There is good evidence that packed red cells do not increase oxygen consumption (at least in the first 24 hours) in patients with sepsis.⁴ Paradoxically, "old" units of packed red cells may cause tissue dysoxia in patients with sepsis.⁴ Furthermore, transfusions of packed red cells may increase mortality in critically ill patients with sepsis.^{5,6}

How, then, does one reconcile these facts with the impressive results of the study by Rivers et al.? Clearly, early, aggres-

sive resuscitation of patients with sepsis improves the outcome. The patients in the early-therapy group received, on average, approximately 1500 ml more in total fluids in the first six hours of treatment than did the standard-therapy group and had a significantly higher mean arterial pressure (mean [\pm SD], 95 ± 19 vs. 81 ± 18 mm Hg; $P < 0.001$). These findings may be important in explaining the difference in outcome between the two groups. We wholeheartedly endorse the concept of early, aggressive volume resuscitation in patients with sepsis, but please do not transfuse blood and do not be misled by the SMVO₂ or ScVO₂.

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To the Editor: Rivers et al. report an impressive reduction in in-hospital mortality in the group of patients with sepsis who received early goal-directed therapy, as compared with those who received standard therapy (absolute reduction, 16 percent). We are concerned, however, that the elevated mortality rate in the standard-therapy group (46.5 percent) may account for these results. In two recent trials of the treatment of severe sepsis, reported by Bernard et al.¹ and Warren et al.,² the mortality rate at 28 days was 30.8 percent and 38.7 percent, respectively, in the placebo groups. These studies involved 1690 and 2314 patients, respectively, with scores for disease severity in the control groups that were similar to the scores in the standard-therapy group in the study by Rivers et al. (Acute Physiology and Chronic Health Evaluation [APACHE II] score, 25.0 ± 7.8 and 20.4 ± 7.4 in the studies by Bernard et al. and Rivers et al., respectively; Simplified Acute Physiology Score II, 49 ± 16 and 48.8 ± 11.1 in the studies by Warren et al. and Rivers et al., respectively).

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To the Editor: Rivers et al. report that early goal-directed therapy provided in the emergency department to patients with severe sepsis and septic shock resulted in a significantly lower mortality than that associated with standard therapy. However, there was a statistically significant difference between the two groups in the rate of achievement of the stated hemodynamic goals. The goals were achieved in 86.1 percent of the patients assigned to standard therapy and in 99.2 percent of those assigned to early goal-directed therapy ($P < 0.001$). A potentially major bias in this study is that it was unblinded, since blinding was not feasible, given the study design. Knowledge of the treatment assignments may have led to a difference in the intensity of bedside care between the two groups. A lower level of intensity in the care given to the standard-therapy group would have resulted in an inability to achieve the investigators' stated goals in this group. This explanation may account for the difference in the incidence of sudden cardiovascular collapse between the two groups and, therefore, the decreased survival in the standard-therapy group.

We agree with the authors' conclusion that "benefits arise from the early identification of patients at high risk for cardiovascular collapse" but believe that these tasks of identification and prevention are best served by intensive bedside care and treatment rather than by the use of additional costly monitoring devices of unproved efficacy.

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The authors reply:

To the Editor: In response to Drs. Marik and Varon: our protocol was derived from the guidelines for hemodynamic support in adults with sepsis issued by the Society of Critical Care Medicine,¹ which discuss the variables that Marik and Varon believe warrant caution (central venous pressure and venous oxygen saturation),^{2,3} including red-cell transfusion. A decreased value for $SMVO_2$ or $ScVO_2$ and an increased lactate level (as in our patients), at any given cardiac index, indicate supply dependency, necessitating further resuscitation.¹ Furthermore, in high-risk elderly patients with cardiovascular disease (such as those in our study), a hematocrit of 30 percent is associated with improved survival.^{1,4}

In response to Sarkar et al.: all patients in both treatment groups met the protocol goals for central venous pressure and mean arterial pressure during the first six hours. In 13.7 percent of the patients in the early-therapy group, $ScVO_2$ was low after the central venous pressure, mean arterial pressure, and hematocrit had been normalized, indicating myo-

cardial suppression and necessitating inotropic therapy.¹ With improved myocardial contractility and compliance, the central venous pressure subsequently decreases, triggering additional administration of fluids. This conversion of pressure to a volume interpretation of central venous pressure was not apparent in the control group in the absence of continuous monitoring of $ScVO_2$. This early recognition of myocardial dysfunction is important because it approximates the mortality benefit.

Venous oximetry is not new and has been extensively studied for more than two decades. Our early use of it as an adjunct in the resuscitation of patients with sepsis is novel. Contrary to the claim by Sarkar et al. that our approach is costly and unproved, it resulted in four fewer days of hospitalization among survivors and decreased the rate of use of pulmonary-artery catheterization by 13.9 percentage points.

In response to Abroug et al.: the APACHE II scores, when used to predict mortality, are computed on the basis of the most abnormal variables in a 24-hour period after admission to the intensive care unit. Our intent was to assess and compare the severity of illness (not mortality) at "nontraditional" time points for scoring (starting within one hour after arrival at the hospital). Because a significant cause of early death was sudden cardiovascular collapse in seemingly stable patients before the onset of serious organ failure, the scores in our study were similar to those in the two cited at base line, but in our study, in-hospital mortality was higher. To reflect mortality adequately, scoring systems must be recalibrated to better weigh coexisting conditions, assess the severity of shock, and reflect events before admission to the intensive care unit. As another reference population, the control group in the study by Gattinoni et al. had a mortality rate of 48.4 percent.³

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Protease Inhibitors and Mortality among Children and Adolescents Infected with HIV-1

To the Editor: The article by Gortmaker et al. about combination therapy including protease inhibitors in children with human immunodeficiency virus type 1 (HIV-1) infection (Nov. 22 issue)¹ includes no data about the side effects of protease inhibitors. The homology of the catalytic region of HIV protease with cytoplasmic retinoic acid-binding protein 1 and low-density lipoprotein receptor-related protein

may allow protease inhibitors to interfere with these proteins, which may be the cause of the metabolic and somatic alterations that develop in patients treated with protease inhibitors (dyslipidemia, insulin resistance, and lipodystrophy).² There is also evidence that protease inhibitors directly inhibit the uptake of glucose in insulin-sensitive tissues, such as skeletal muscle.² Moreover, the bone mineral loss induced by protease inhibitors may retard the growth of HIV-infected children and adolescents.

An attractive approach to the management of the somatic and metabolic alterations induced by protease inhibitors is to switch to triple-therapy regimens that do not include a protease inhibitor. The switch from regimens containing protease inhibitors to regimens containing nonnucleoside reverse-transcriptase inhibitors may result in some benefit in terms of the levels of circulating lipids, bone mineral density, and lipodystrophy without causing substantial viral rebound.³ Unfortunately, Gortmaker et al. include no data on switching from protease inhibitors or related follow-up data. Despite the reduction in mortality from AIDS-related diseases, the side effects of protease inhibitors could influence the growth of HIV-infected children and adolescents and increase both the incidence of metabolic and cardiovascular complications⁴ and related costs for care and rehabilitation.

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The authors reply:

To the Editor: We agree with Dr. Barbaro that there are potentially substantial adverse effects associated with all antiretroviral therapies, including protease inhibitors, as we tried to point out. The Pediatric AIDS Clinical Trials Group Protocol 219 (PACTG 219) is a prospective cohort study designed to examine not only the benefits of antiretroviral therapy, but also the potential adverse effects of such treatment. We agree with Dr. Barbaro that these effects require close scrutiny, now that it has been demonstrated that combination antiretroviral therapy can significantly reduce the previously high mortality associated with HIV disease in children. Further study is clearly needed to identify adverse outcomes related to specific drugs and to determine whether such outcomes are related to the disease or to other factors. However, we must recognize that, if it were not for the success of these highly active antiretroviral therapies, concern about the long-term consequences of therapy might not be relevant.

Considerable disagreement remains as to whether switching regimens can reverse all the changes in metabolism and body composition that have been observed in patients receiving regimens that include protease inhibitors. An important issue is whether some of these changes, such as the loss of bone mineral density or changes in body composition, may be directly related to the underlying disease and not just to the therapeutic interventions used to treat HIV.

The results of another study by the PACTG 219 team indicated that the use of combination therapies containing protease inhibitors was not associated with retardation of growth in height or weight,¹ although another report provides evidence of osteonecrosis.² Most of the metabolic abnormalities cited by Dr. Barbaro have been described in adult populations. We share his concern that the same abnormalities could adversely affect the quality of life for HIV-infected children. These long-term issues related to toxic effects will be a focus of future analyses of the data being generated by PACTG 219.

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An Outbreak of Primary Pneumonic Tularemia

To the Editor: We disagree with Feldman et al. and Hornick (Nov. 29 issue)^{1,2} about establishing a diagnosis of primary pneumonic tularemia on the basis of clinical symptoms and a single high titer of anti-*Francisella tularensis* antibody alone, even during an outbreak of tularemia. A single high titer may reflect a previous infection with *F. tularensis*, either symptomatic or asymptomatic. High titers of both IgG and IgM antibodies, determined by agglutination assay or enzyme-linked immunosorbent assay, can persist for more than 10 years.³ Asymptomatic infection is not uncommon.⁴ Unlike the ulceroglandular form of tularemia, the pneumonic form has no clinical characteristics to distinguish it from pneumonia of other causes. Therefore, before *F. tularensis* is considered the cause of a pneumonic infection in a patient with a single high titer of antibody, other causes should be ruled out. Furthermore, contrary to what is stated in the editorial, a positive titer after one week of illness is more likely to denote a previous infection than a present infection, since antibodies often develop late in the course of tularemia.³

During a recent outbreak in Sweden, 26 patients with tularemia were seen in our department. All nine patients with

paired serum samples initially had negative titers by agglutination assay, and six of those samples had been obtained more than a week after the onset of illness (on days 8, 10, 10, 10, 12, and 18).

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To the Editor: In his editorial, Hornick suggests that fluoroquinolones be considered in the treatment of tularemia, although only a few patients have been reported to have been cured with these drugs. In an outbreak in 1997 and 1998, ciprofloxacin was used in 66 patients, with better results than with the usual therapeutic regimens. It was used as a second-line treatment after the failure of other treatments in 34 patients, 30 of whom were cured.¹ Fluoroquinolones should probably be considered first-line treatment because they are effective, have fewer side effects than other drugs, and are easy to administer.

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The authors reply:

To the Editor: We agree with Strålin et al., in that a fourfold or greater change in the titer of anti-*F. tularensis* antibody is necessary to confirm a diagnosis of tularemia serologically. We also concur that in patients with suspected pneumonic tularemia, other causes of community-acquired pneumonia and atypical pneumonia must be considered. In our epidemiologic investigation, we used clinical findings supported by elevated serologic titers to identify cases; paired serum samples were sought for this purpose but not always obtained.

Of the 11 patients with primary pneumonic tularemia in the 2000 outbreak, 7 had at least a fourfold rise in the serum titer of anti-*F. tularensis* antibody, and 1 had a positive culture for *F. tularensis*. The three additional patients meeting our case definition of pneumonic tularemia had single ti-

ters of 1:1280, 1:256, and 1:128. Both patients with the more easily distinguished ulceroglandular form had single titers of 1:1024. Of the two patients with a typhoidal presentation, one (in whom the first sample was collected two months after the onset of illness) had a titer that decreased by a factor of eight; the other had a threefold increase, but it was not possible to obtain an additional serum sample.

Any nondifferential misclassification, in which the case status of study participants is misclassified independently of their exposure status, results in bias toward the null. If the three patients with pneumonic tularemia who had single titers are reclassified as controls, the odds ratio associated with use of a lawn mower or brush cutter in the two weeks before illness is 13.1 (95 percent confidence interval, 1.5 to 612.1), as compared with the reported odds ratio of 9.2 (95 percent confidence interval, 1.6 to 68.0).

Our experiences with microagglutination titers agree with those of others. The patient with pneumonic tularemia who died in the summer of 2000 was seronegative 11 days after the onset of illness; tularemia was confirmed by isolation of *F. tularensis*. In 2001 we detected antibody in serum specimens from three patients from the 1978 outbreak on Martha's Vineyard¹ (two with titers of 1:128 and one with a titer of 1:256); a fourth patient from the 1978 outbreak was seronegative.

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The editorialist replies:

To the Editor: Strålin et al. question my statements regarding the elements used in diagnosing pneumonitis caused by *F. tularensis*. This form of tularemia is rare in the United States. Most patients are infected with the virulent type A organisms and are usually quite ill; appropriate, prompt administration of an antibiotic will prevent death and reduce the severity of illness. Making a diagnosis in these patients is difficult; in the editorial I cite a report of a fatal case that was not identified as tularemia disease.¹ Obviously, culture of sputum, gastric, and throat specimens is the ideal method of making a diagnosis. This is a risk for laboratory workers unless they are notified about the possibility of *F. tularensis* in the specimens. Because it is a rare infection, few physicians request a culture for the organisms. Therefore, serologic studies are obtained, frequently after the patient has been ill for a week. If a serum sample is screened for antibodies to *F. tularensis* and the patient has a titer of 1:160, it would be prudent to treat the patient with an antibiotic known to be effective in the treatment of tularemia. The classic fourfold rise in antibody titer would confirm the diagnosis, even if no culture had been obtained.

Strålin and colleagues are correct in noting that few pa-

tients have a titer of 1:160 at one week. However, in an outbreak involving type A organisms, that titer would be considered diagnostic of tularemia, because few serologic surveys have been performed in areas where infection with the type A strain is endemic and in persons who have no history of tularemia. Patients who have had type A pneumonitis do have persistent agglutinating-antibody titers for several years and should be immune to reinfection, if they are immunocompetent.

Pérez-Castrillón et al. have reported the use of fluoroquinolones in the treatment of patients with tularemia. I was aware of their report and should have acknowledged their work. In their study, the less virulent form of *F. tularensis* was the causative microbe. Presumably, disease caused by type A organisms will be as responsive as the less virulent form. However, only a few patients with type A disease have been successfully treated.

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Case 38-2001: Paraneoplastic Encephalomyelitis and Sensory Ganglioneuropathy

To the Editor: In discussing the case of paraneoplastic ganglioneuropathy with encephalopathy (Dec. 13 issue),¹ Amato and Anderson focused on the differential diagnosis and the causes of sensory ganglioneuropathies. Because of the primary impairment of T-shaped sensory neurons, ganglioneuropathies are characterized by a specific pattern of joint, muscle, and skin denervation involving myelinated and unmyelinated axons in a fashion that is not length-dependent, leading to distinct clinical, neurophysiological, and neuropathological findings. Typically, skin biopsy demonstrates similar decreases in fiber density at distal and proximal sites.² Moreover, evidence of involvement of the central sensory pathway can be used to localize the disease process to the dorsal-root ganglia and differentiate ganglioneuropathies from axonal neuropathies. Magnetic resonance imaging (MRI) of the spinal cord can show high signal intensity on T₂-weighted images throughout the length of the posterior columns.³ In this case, the linear hyperintensity on the posterior aspect of the cervical spine probably reflected the degeneration of central sensory projections observed on neuropathological examination and would have been better demonstrated by axial MRI scanning at different levels.

The authors report a number of disorders associated with sensory ganglioneuropathies, but additional disorders with such associations include chronic autoimmune hepatitis,³ among autoimmune diseases, and infection with Epstein-Barr virus,⁴ varicella-zoster virus,⁵ or measles virus, among infectious diseases. Finally, sensory neurons of dorsal-root ganglia are primarily affected in many genetic diseases, including hereditary sensory autonomic neuropathies, Fabry's disease, vi-

tamin E deficiency, Friedreich's ataxia, and other types of spinocerebellar degeneration.^{3,6}

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Dr. Amato replies:

To the Editor: Lauria and colleagues have performed elegant studies of epidermal nerve fiber density¹ and spinal MRI findings² in patients with sensory ganglioneuropathies. With regard to the case of paraneoplastic encephalomyelitis and sensory ganglioneuropathy, they suggest that the linear abnormality noted on the MRI scan of the cervical spine may be secondary to degeneration of the posterior columns related to the sensory ganglioneuropathy. However, I chose not to overinterpret this subtle abnormality, which was present only on the sagittal image, because concurrent signal changes were not noted on the axial images. Skin-biopsy specimens were not readily available, and more important, would not have provided any more information that would have been useful for localization than was already available from the clinical history and examination.

The time constraints of the clinicopathological conference did not allow me to present a differential diagnosis of every reported disease associated with damage to the sensory ganglia. Instead, I tried to review the pertinent aspects of the patient's history, neurologic examination, and workup that would allow for a reasonable differential diagnosis and specification of further diagnostic testing required to make the correct diagnosis. In a patient with known small-cell lung cancer, an altered mental status, pseudoathetosis, myoclonus, and sensory ataxia, there was no differential diagnosis. Nevertheless, I tried to discuss common neuropathies that occur in the setting of cancer. I would never consider chronic autoimmune hepatitis infection with Epstein-Barr virus, varicella-zoster virus, or measles virus or any of the inherited neuropathies listed by Lauria and colleagues as possible causes of this patient's neurologic deterioration. The patient had an obvious paraneoplastic disorder, which was eventually confirmed by the demonstration of anti-Hu antibodies in the serum and cerebrospinal fluid and by the autopsy findings.

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1. Lauria G, Sghirlanzoni A, Lombardi R, Pareyson D. Epidermal nerve fiber density in sensory ganglionopathies: clinical and neurophysiologic correlations. *Muscle Nerve* 2001;24:1034-9.
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Will Parity in Coverage Result in Better Mental Health Care?

To the Editor: Frank et al. (Dec. 6 issue)¹ make the case that managed care enhances the affordability and feasibility of parity in coverage for mental health care because of controls implemented by managed-care organizations on the utilization and costs of psychiatric treatment. The authors point out, however, that parity with managed care will not cover many cost-effective services, such as day hospitals, psychosocial rehabilitation, case management, or residential treatment programs. It is this model of acute care in the event of "medical necessity" that has led, in my view, to a downside of managed care that requires attention.

Managed care for behavioral health that is "carved out" of the health benefits provided by an employer — a practice that is ubiquitous — has led to psychiatric hospital stays of ever decreasing duration, so that stays of three, four, or five days are commonplace and multiple readmissions are standard practice. Managed behavioral health care has shifted many people from private insurance to the public sector for care. Parity with managed care has done nothing to reduce the number of mentally ill persons who are homeless or of those who are incarcerated; this situation represents a public health crisis and a failure of public policy.² Before we think that we have solved the problem of financial discrimination against the mentally ill and the problems associated with their treatment through parity with managed care, we must evaluate more carefully the effects of managed mental health care.

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1. Frank RG, Goldman HH, McGuire TG. Will parity in coverage result in better mental health care? *N Engl J Med* 2001;345:1701-4.
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To the Editor: As a pediatrician in a community with abundant resources for mental health care, I am appalled by the inaccessibility of mental health care providers to my patients. Psychiatrists and psychologists have opted out of insurance panels, simply because they cannot afford to stay on them. When one of my patients with severe learning disabilities and behavioral problems required neuropsychological testing, the health plan covering the patient reimbursed the psychologist \$53 per hour for the appropriate consultations, testing, and follow-up.

To improve access to appropriate mental health care services, we need to do more than improve insurance coverage.

We have to address the problem of inadequate reimbursement for mental health care providers.

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The authors reply:

To the Editor: Drs. Sharfstein and Benjamin highlight two points. First, parity is necessary but not sufficient to ensure access to high-quality mental health care services. Second, parity does not provide for many services that are needed to support patients with severe and persistent mental disorders.

Dr. Benjamin is frustrated by the difficulty of finding a child psychiatrist who will accept referrals, despite the fact that he practices in one of the most psychiatrist-rich areas of the country — upper-income communities in suburban Boston. In spite of a seemingly generous supply of therapists, networks can be organized and reimbursed in such a fashion as to create barriers to access that are every bit as substantial as those created by limits on coverage and high levels of cost sharing.

Dr. Sharfstein notes that even in the presence of a benefit providing parity in mental health coverage, the focus of health insurance on acute care limits coverage for cost-effective services such as psychosocial rehabilitation. He also claims that parity and managed care have resulted in a shifting of costs to the public mental health system. Although this effect is plausible, at this point his argument is speculative.

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West Nile Encephalitis in Massachusetts

To the Editor: We describe one of the two sentinel cases of West Nile encephalitis that have occurred in humans in Massachusetts. Both cases involved elderly Massachusetts residents, one of whom died from West Nile virus infection in mid-October, and both were diagnosed simultaneously.

An 89-year-old man presented to a community hospital in October 2001 with fever and confusion. Except for recurrent hematuria, he had been in good health until the day before admission. After going to sleep unusually early, he awoke confused at 12:30 a.m. and slumped to the floor. His initial evaluation was notable for fever (oral temperature, 38.6°C), disorientation, and difficulty following commands.

There was no clear weakness, and the remainder of his physical examination was unrevealing. The white-cell count was 6.9×10^3 per cubic millimeter (78 percent polymorphonuclear leukocytes, 13 percent lymphocytes, and 8 percent monocytes). Cerebrospinal fluid contained 1785 red cells and 120 white cells per cubic millimeter (75 percent neutrophils); a polymerase-chain-reaction assay for herpes simplex virus was negative. The patient's temperature subsequently rose to 39.4°C. At the time of his transfer to our hospital four days after presentation, he was mute and did not respond to oral commands; the score on the Glasgow Coma Scale was 7 (a score of 1 on the eye-opening category, a score of 1 on the best-verbal-response category, and a score of 5 on the best-motor-response category). Repeated lumbar puncture revealed 39 white cells per cubic millimeter (29 percent neutrophils and 51 percent lymphocytes). Electroencephalography revealed diffuse slowing with triphasic waves. Magnetic resonance imaging showed a small acute infarct in the right cerebellar hemisphere and the absence of meningeal or parenchymal enhancement. The patient had worked on his farm until the week before he became ill. Both his home and farm are located in a town where West Nile virus had been isolated from mosquitoes and horses. Serologic analysis of serum for West Nile virus on the fifth hospital day revealed an IgM titer of at least 1:12,800; the results of a confirmatory plaque-reduction neutralization assay were positive. Measurement of IgG titers in serum samples obtained four days apart showed an increase from 1:1600 to 1:6400. After receiving supportive care for one month, the patient is slowly recovering at this writing. He remains disoriented but can engage in short conversations.

West Nile virus is a flavivirus that can be transmitted by mosquitoes from its host species (birds) to humans. Since the first U.S. cases were described in 1999, human infection has been documented in 10 states, with an additional 17 states reporting the isolation of West Nile virus from mosquitoes, birds, or horses.^{1,2} Older age is a risk factor for severe neurologic illness and death from West Nile virus infection.³ Currently, there is no specific treatment for West Nile encephalitis; vaccines are being developed.

This case report serves to remind physicians of the importance of screening for endemic arboviruses in the proper clinical setting and to underscore the importance of public health initiatives to reduce the prevalence of mosquito-borne illnesses.

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