Letters

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Injury, Death, and Cholesterol

To the Editor: In 1990, my colleagues and I reported that the mortality outcomes in randomized, primary prevention trials of cholesterol-lowering measures, when aggregated with the results of a meta-analysis, showed an excess of suicide and traumatic deaths (that is, "non-illness mortality") in the treated men (odds ratio, 1.76; $P = 0.004$) (1). Cummings and Psaty (2) examined this issue by analyzing non-illness mortality occurring in multiple risk factor intervention trials; however, these trials, by their design, preclude attributing any outcome to one particular intervention.

Cummings and Psaty also redid the meta-analysis of the primary prevention trials of non-illness mortality and found that this treatment effect is not statistically significant (odds ratio, 1.42; $P > 0.05$). The difference between our findings and theirs is due to Cummings and Psaty's use of recently reported "intention-to-treat" outcomes from the World Health Organization (WHO) trial, their exclusion of the Los Angeles Veterans Affairs trial because of incomplete data, and their inclusion of the Expanded Clinical Evaluation of Lovastatin (EXCEL) study. Reasonable arguments can be made against the latter two decisions.

Although complete data from the Veterans Affairs trial would be preferred, excluding this trial from analysis is neither satisfying nor free of potential bias. Inclusion of the EXCEL study is dubious because no traumatic deaths occurred, and therefore its consideration only dilutes a treatment effect apparent in trials in which at least one non–illness-related death occurred. Moreover, it was not designed as a primary prevention trial, and, of the deaths not caused by trauma, 70% occurred in persons known to have heart disease at baseline (3).

Beyond specific methodologic arguments, it is important to note that primary prevention trials have been repeatedly analyzed because one of the outcomes—increased mortality in the treatment groups—is unexpected and worrisome. Iterative analyses, however, may tempt us to believe the particular analysis that produces the desired finding. One way to safeguard against this is to present the results from all analytic permutations and to compare these results with a "favorable" outcome. For example, when non-illness mortality and cardiac mortality were directly compared in various groupings of the eight largest single-intervention, randomized trials, we noted that the increase in non-illness mortality with cholesterol interventions appears to be more robust than the cardiac mortality reduction (4) (Table 1).

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2. **Cummings P, Psaty BM.** The association between cholesterol and death from injury. Ann Intern Med. 1994;120:848-55.
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4. **Muldoon MF, Rossouw JE, Manuck SB, Glueck CJ, Kaplan JR, Kauf-**

Table 1. Meta-analytic Findings for Non-Illness and Coronary Heart Disease Mortality in Randomized Trials of Cholesterol Reduction*

Trial	Non-Illness Mortality		CHD Mortality	
	Odds Ratio† (95% CI)	*P* Value	Odds Ratio (95% CI)	*P* Value
All trials‡ ($n = 8$)	1.55 (1.11 to 2.17)	0.010	0.89 (0.80 to 1.00)	0.051
All trials (new WHO data§) ($n = 8$)	1.40 (1.02 to 1.93)	0.026	0.92 (0.83 to 1.02)	0.118
Primary prevention trials ($n = 6$)	1.76 (1.19 to 2.59)	0.004	0.85 (0.69 to 1.05)	0.125
Secondary prevention trials ($n = 2$)	1.07 (0.55 to 2.11)	>0.2	0.92 (0.80 to 1.13)	0.179
Dietary trials ($n = 2$)	1.76 (0.94 to 3.31)	0.077	0.95 (0.72 to 1.30)	>0.2
Drug trials ($n = 5$)	1.52 (1.01 to 2.29)	0.047	0.90 (0.80 to 1.02)	0.102
Excluding fibric acid trials‖ ($n = 6$)	1.76 (1.14 to 2.73)	0.011	0.87 (0.76 to 1.00)	0.042
Long trials¶ ($n = 6$)	1.53 (1.03 to 2.26)	0.035	0.90 (0.80 to 1.01)	0.073
Long trials excluding fibric acid trials ($n = 4$)	1.88 (1.04 to 3.41)	0.037	0.87 (0.76 to 1.01)	0.061

* CHD = coronary heart disease; WHO = World Health Organization. Adapted from reference 4.
† Odds ratios for mortality across trials calculated according to the modified Mantel-Haenszel procedure.
‡ Includes the six primary prevention trials (1) and the major secondary prevention trials that have reported non-illness mortality (Coronary Drug Project and Program on the Surgical Control of Hyperlipidemia study).
§ Uses WHO trial "intention-to-treat" mortality data.
‖ Excludes Helsinki Heart Study (gemfibrozil) and WHO trial (clofibrate) and includes only the niacin treatment arm from the Coronary Drug Project.
¶ Long trials defined as those having intervention periods of at least 5 years.

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In response: Our meta-analysis of primary prevention trials of cholesterol-lowering interventions in men showed the relative risk for death from injury among treated men compared with that of controls to be 1.42 (95% CI, 0.94 to 2.15) (1). Although Dr. Muldoon disagrees with our inclusion of the EXCEL trial (2) and exclusion of the Veterans Affairs trial (3), these choices are of little practical importance. Including the Veterans Affairs trial and excluding the EXCEL trial produces essentially the same estimate of effect, as reported in the second line of Dr. Muldoon's Table 1; that is, our findings are substantially in agreement with those of Dr. Muldoon.

Dr. Muldoon seems to agree that the best data from the WHO clofibrate trial are those reported from the intention-to-treat analysis (4). When Muldoon and colleagues (5) published their meta-analysis in 1990, the intention-to-treat results were not available. It is not clear why he continues to use the older WHO data in some of the calculations. We believe that the results most free of bias are based on intention-to-treat analyses.

We share Dr. Muldoon's concern about the possible adverse effects of cholesterol-lowering therapy and state in our review (1) that his meta-analysis (5) helped to focus attention on this topic. Although we found a slightly smaller estimate of effect (and a wider confidence interval) than he did, we concluded that cholesterol-lowering interventions in primary prevention trials are associated with an increase in the risk for death from injury among men. We are not certain whether this association is due to chance or to an adverse effect of cholesterol-lowering therapy. The results of several large trials of cholesterol-lowering interventions will be presented before the turn of the century, and, if deaths from injury are adequately reported, this question may soon be clarified.

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Case Management after Acute Myocardial Infarction

To the Editor: The recent study by DeBusk and colleagues (1) is as important for its implications as for its findings. Their experiment confirms and extends observations repeatedly made over the past two decades: Nurses are vastly more successful at protocol-driven health promotion than are primary care physicians (1). This finding should not be surprising, given that doctors are trained exclusively to diagnose and treat disease. Health promotion and prevention require a different set of skills and interests.

What is new in their work is the conclusion that the old message applies to "managed care." The Kaiser-Permanente Medical Clinics are widely recognized to be among the best practitioners of managed care in the country. Yet, at least in patients who have had myocardial infarction, the nurse case-management system proved superior to the preventive care provided by physicians. Further, it is not unreasonable to speculate that in other areas of prevention, such as the control of hypertension or hyperlipidemia, health maintenance organizations might be found equally wanting compared with categorical programs placed in the hands of nurses.

Health reform seems destined to make managed care its chosen model of service. The challenge for policymakers is to build on the lessons of the study by DeBusk and colleagues and to find a better way to deliver to all patients those valuable prevention protocols that have been shown to improve health. It is time to abandon our reliance on doctors and "the sick care system" for these measures. Instead, we should consider developing a complementary approach that brings prevention to the entire population and leaves physicians to do what they do best, that is, to serve the sick.

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In response: We appreciate and agree with the comments of Drs. Alderman and Shenson regarding the complementary nature of nurse case management for prevention and physician management for acute illness. In our study, the nurse case-managment system provided better preventive care than physicians provided alone. However, the authors interpreted our study as suggesting that the preventive care provided in this managed care organization was "wanting." We disagree with this interpretation. The Kaiser-Permanente Medical Care Program has recognized the value of the improved care provided by the nurse case managers. Nurse case management has been incorporated into the routine preventive care provided to patients with ischemic heart disease in 10 Kaiser-Permanente hospitals in northern California, with plans for future expansion to more hospitals.

Our nurse case-management system was developed in response to the fact that management of acute illness leaves physicians little time for direct involvement in preventive care. Incorporating prevention and rehabilitation into medical practice necessitates a complementary approach that uses both physicians and nurses to address the aspects of care for which they are best suited.

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Persistently Perplexing Purpuras: Thrombotic Thrombocytopenic Purpura and Immune Thrombocytopenic Purpura

To the Editor: We read with interest Olenich and Schattner's report (1) on postpartum thrombotic thrombocytopenic purpura (TTP) complicating immune thrombocytopenic purpura (ITP) of pregnancy and agree wholeheartedly with their emphasis on examining the peripheral smear in cases of thrombocytopenia. In a high-technology era, it is rewarding to see the value of a low-technology procedure. Although the authors asserted that coexistence of the two disorders had not been documented, we previously reported a series of three patients with TTP who developed immune thrombocytopenia while their TTP was in remission (2). Patient 1 in our series initially presented with postpartum TTP before developing ITP. Since that report was published, we have observed an additional case of ITP that occurred after treatment of TTP and that responded to splenectomy. An additional case has been reported to us by a colleague at another institution. The association of TTP and ITP may not be that rare but clearly will not be noted if physicians fail to examine the peripheral smear.

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To the Editor: Olenich and Schattner (1) report a case of postpartum TTP complicating pregnancy-associated ITP. The authors state that "the coexistence of these two disorders has not been previously reported in the English-language literature."

Although coexistent TTP and ITP may not have been reported during pregnancy, the association between these two diseases has previously been described in various settings. Elevated levels of platelet-associated antibodies have been noted in patients with TTP (2, 3), and the sequential development of TTP and ITP has been described in several cases, including one after pregnancy (4). Further, the association between TTP and ITP in patients infected with the human immunodeficiency virus has recently been recognized (5), as predicted by the authors. Thus, TTP and ITP may coexist in the susceptible host, and the role of immunologic factors in TTP remains an enigma.

Olenich and Schattner make the important point that the diagnosis of TTP may be overlooked if pertinent data are not carefully evaluated. Perhaps another lesson is that, given the vicissitudes of searching the medical database, caution should be exercised when making statements about the "English-language literature" because pertinent articles may be overlooked.

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To the Editor: The recent report by Olenich and Schattner (1) of a case considered to represent postpartum TTP did not provide sufficient data to permit this diagnosis. Of the usual manifestations of TTP—thrombocytopenia, microangiopathic hemolytic anemia, neurologic symptoms, fever, and renal dysfunction—the patient in question seemed to have only two. Apparently, no hypertension, neurologic symptoms, or evidence of consumption coagulopathy were noted. A description of a skin or mucosal biopsy specimen confirming the clinical impression by showing aneurysmal dilatation at arteriocapillary junctions and hyaline deposits was also absent (2). Unquestionably, this patient had thrombocytopenia and hemolytic anemia. We previously showed that nearly all patients with chronic ITP have subclinical erythrocyte fragmentation (3). When hemolysis becomes clinically significant, as in Olenich and Schattner's patient, it is usually referred to as the Evan syndrome (4). Despite some improvement in the management of TTP, the prognosis of the syndrome remains much more grave than that of acute or chronic ITP, with or without overt hemolysis.

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In response: We appreciate the letter by Drs. Stein and Flexner, who have observed and reported cases similar to ours (1). As emphasized by Dr. Stricker, the association of ITP and TTP may not be rare and may instead be underdiagnosed.

In response to Dr. Zucker-Franklin, we stand by our diagnosis. The classic pentad for TTP is present in only few cases. Of the patients reviewed by Ridolfi and Bell (2), 74% had only microangiopathic hemolytic anemia, thrombocytopenia, and neurologic symptoms (3). Our patient had personality changes (which were admittedly difficult to distinguish from postpartum depression) and was receiving steroids, which may have suppressed her fever, as we addressed in our original report. Although Zucker-Franklin and Karpatkin (4) have reported erythrocyte and platelet fragmentation in patients with ITP, as noted by electron microscopy, they and others have not described a fulminant schistocytosis that is visible on routine peripheral blood smear and is accompanied by marked lactate dehydrogenase elevation, as occurred in our patient and which would be most unusual for ITP. Although the Evan syndrome is a possible diagnosis for any patient with ITP and acquired hemolytic anemia, it is defined in part by the presence of autoantibodies directed at erythrocytes (5). Our patient did not have microspherocytes and did not have anti-erythrocyte antibodies. (Both direct and indirect test results were negative.)

We agree that a skin or mucosal skin biopsy might have been useful to support the diagnosis of TTP but did not believe it necessary in this case. Instead, the sudden onset of a Coombs-negative, microangiopathic, hemolytic anemia and thrombocytopenia in the postpartum period, without hypertension or significant proteinuria, suggested TTP. In addition, our patient's dramatic response to plasmapheresis, with complete normalization of her smear results and lactate dehydrogenase level, further supports the diagnosis.

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Cocaine-induced Acute Renal Failure without Rhabdomyolysis

To the Editor: Several cases of acute renal failure following cocaine abuse, all associated with rhabdomyolysis, have been reported (1, 2). We report a case of acute renal failure occurring

Table 1. Serum Biochemical Findings during Hospitalization*

Variable	Days after Admission							
	0	1	2	3	4	6	8	9
Urea, *mmol/L*	15.7	17.1	22.2	21.4	17.1	10.7	8.6	8.6
Creatinine, *μmol/L*	424	583	760	725	530	292	168	141
K+, *mmol/L*			4.4	4.1	3.8	3.7	4.4	4.3
Cl−, *mmol/L*			97	106		108	103	
HCO_3−, *mmol/L*				19		20	27	
Uric acid, *μmol/L*				589				
Total calcium, *mmol/L*				2.08		2.15		
Phosphorus, *mmol/L*				1.74		1.65		
Albumin, *g/L*				36				
Creatine kinase, *U/L*	33			26				
AST, *U/L*	28							
LDH, *U/L*	83							

* AST = aspartate aminotransferase; LDH = lactate dehydrogenase.

secondary to cocaine abuse but without evidence of rhabdomyolysis.

A 16-year-old girl with no contributory medical history except for oral contraceptive use was transferred to our hospital for acute renal failure in November 1993. She admitted having inhaled cocaine 3 days before admission. Findings from her physical examination were unremarkable. Her blood pressure was 110/60 mm Hg without orthostatic change, her pulse rate was 72 beats per minute, and she was afebrile. Her urine output remained at 1 to 1.5 L/d with a sodium fractional excretion greater than 1%. Her weight was stable during hospitalization. Complete blood count and routine coagulation test results were normal. Her serum biochemistry results are summarized in Table 1.

Serum levels of complement, antistreptolysin, and antinuclear antibodies were all within normal limits, and a test result for hepatitis B surface antigen was negative. Urinalysis showed a pH of 5.0, positivity for blood, and traces of protein. Microscopic examination showed 3 to 4 erythrocytes, 4 to 6 leukocytes, and 1 granular cast per high-power field. Her kidneys appeared normal in size and slightly hyperechogenic on ultrasound examination. A kidney biopsy specimen obtained on day 2 showed 10 glomeruli of normal appearance. Vessels, interstitium, and tubules were unremarkable except for rare dilatations of tubular lumens, in which a few granular casts were seen. Immunofluorescence staining was nonspecific. The patient recovered renal function spontaneously.

In this case, cocaine was strongly suspected as the only cause of the acute renal failure. To our knowledge, however, all previously reported cases of cocaine-associated acute renal failure were related to an induced rhabdomyolysis. Because the half-life of serum creatine kinase is 17 hours in normal persons and is prolonged in patients with renal insufficiency (3), it seems improbable that rhabdomyolysis was the initiating event of acute renal failure in this patient. Of note, however, the vasoconstrictive properties of cocaine have been previously implicated in cardiovascular toxicity (4). Vasoconstriction caused by cocaine relates to the blockade of norepinephrine reuptake and to the release of adrenal catecholamines (5). Ischemia caused by intense intrarenal vasoconstriction may have resulted in medullary hypoxia and tubular dysfunction in our patient. We suggest that acute renal failure following cocaine abuse can supervene in the absence of concomitant rhabdomyolysis.

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Clozapine-related Pancreatitis

To the Editor: Clozapine, an atypical antipsychotic drug, has some well-known adverse reactions (1). The most important is agranulocytosis, which necessitates regular monitoring of leukocyte counts. This agent has also been associated with gastrointestinal complications, but, to our knowledge, only two cases of pancreatitis have been reported (2, 3). We report a new case of pancreatitis related to an acute overdosage of clozapine.

A 63-year-old woman with a history of dysthymic disorder and a personality disorder with histrionic traits attempted suicide by ingesting more than 1000 mg of clozapine—a drug that had been used to treat chronic schizophrenia in her son. She had no history of alcohol or drug intake.

She arrived at the hospital in a coma (Glasgow coma score of 7) and was intubated for mechanical ventilation. Results of blood screening for common toxic drugs (benzodiazepines, cocaine, opiates, cyclic antidepressants) were negative. Abnormal laboratory determinations included a leukocyte count of 2000 cells/mm^3, a serum amylase level of 7000 UI/L (normal, <200 UI/L), a lipase level of 459 UI/L (normal, <190 UI/L), a total bilirubin level of 1.5 mg/dL (normal, <1.1 mg/dL), and a direct bilirubin level of 1.1 mg/dL. No electrocardiographic changes were detected.

On the second day, her neurologic status improved to a Glasgow coma score of 11, but she developed delirium (probably caused by the anticholinergic effect of clozapine). On the fourth day, she was conscious and well oriented and was extubated. Her leukocyte count recovered in 5 days to 5200 cells/mm^3. Her amylase level progressively decreased to normal within a week. The patient did not report abdominal pain.

An echographic study done on the third day showed a decrease in echogenicity through the pancreatic parenchyma, which is consistent with an inflammatory process (Figure 1), and showed no evidence of gallstones. A new study repeated before the patient was discharged showed a normal echogenicity of the pancreatic parenchyma (Figure 1).

The diagnosis of pancreatitis in our patient is supported by the hyperamylasemia and hyperamylasuria, elevated serum lipase level (specificity approaching 90%), and ultrasonographic images. Hypersalivation is a common and unexplained effect of clozapine (1, 4). This hypersalivation can produce mild hyperamylase levels, but the range of amylase levels and the associated increase in lipase levels in this case favor a diagnosis of acute pancreatitis.

The temporal association of clozapine intake and the diagnosis

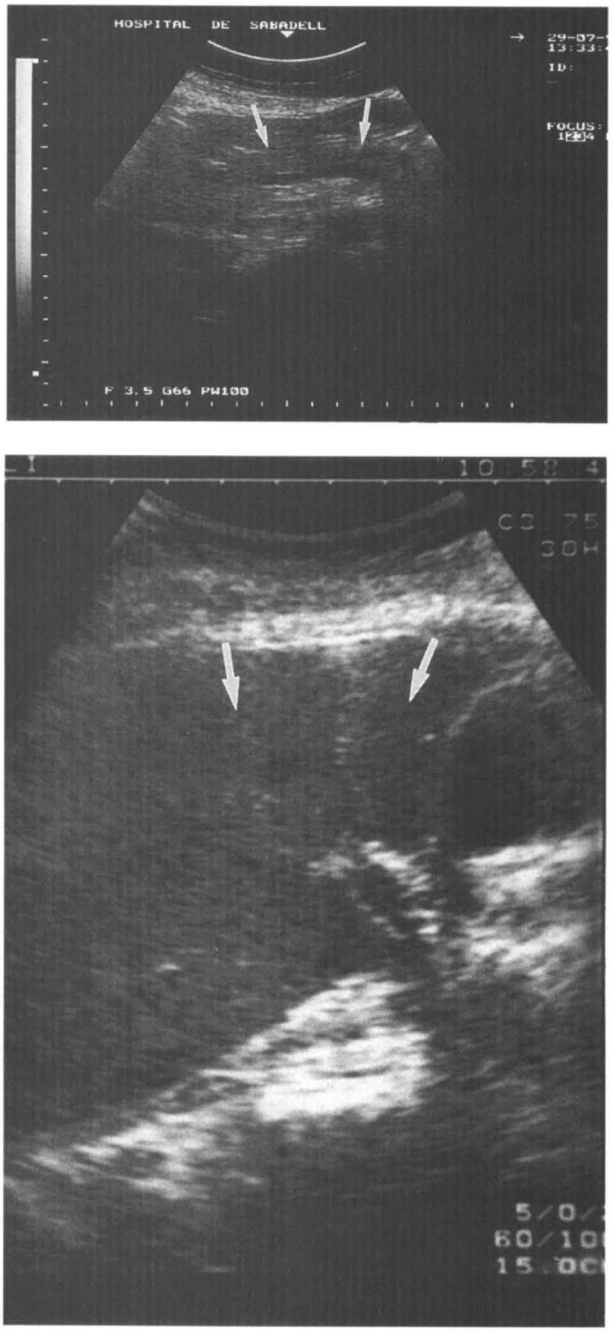

Figure 1. Ultrasonographic transverse scan at the epigastric level. Top. Low ecogenicity of the pancreatic parenchyma (*arrows*). **Bottom.** Normal ecogenicity of the pancreatic parenchyma (*arrows*).

of pancreatitis, excluding other causes of pancreatitis, allows us to suggest a drug-related event.

The two patients previously described had clinical, overt pancreatitis diagnosed from abdominal pain and abnormal laboratory and echographic findings. In our patient, routine amylase monitoring detected a subclinical asymptomatic pancreatitis. Routine amylase level monitoring in patients treated with high doses of clozapine is advisable and will probably show a more frequent pancreatic involvement than is currently reported.

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Hepatic Injury after Interferon-α Therapy for Chronic Hepatitis C

To the Editor: We describe a patient with chronic hepatitis C virus (HCV) infection who had severe hepatic injury during interferon-α therapy and who was successfully treated with prostaglandin E_1 and prednisone.

A 54-year-old man with chronic active HCV infection received intramuscular interferon-α2a (Canferon A, Takeda Pharmaceutical Company, Japan), 9×10^6/U three times a week, in March 1992. The patient's serum alanine aminotransferase (ALT) level returned to normal in April. In May, he reported an itchy eruption, and his serum ALT level began to increase. On 15 June, his serum ALT level reached 811 kU, and interferon therapy was discontinued. On hospitalization, he showed severe hepatic injury: His total bilirubin level was 4.9 mg/dL; his conjugated bilirubin level was 4.3 mg/dL; his ALT level was 885 kU; his asparate aminotransferase level was 940 kU (normal, 10 to 26 kU); and his prothrombin time was 32% of normal. Test results were negative for hepatitis B virus, hepatitis A virus, cytomegalovirus, and Epstein-Barr virus. Autoantibodies, such as antinuclear, anti–smooth muscle, antimitochondrial, and anti–liver-kidney microsomal antibodies, as well as anti–interferon-α antibody, were not detected in the serum. The serum IgG concentration was 1850 mg/dL. Test results showed that HCV-RNA in the serum obtained on 4 June 1992 was negative. Continuous infusion of prostaglandin E_1 (1) was begun, and the patient's prothrombin time improved rapidly. However, his serum ALT levels remained high, and prednisone therapy was initiated. His serum ALT level decreased to 103 kU after 2 weeks of prednisone administration.

Patients with fatal liver failure induced by interferon administration have been described (2, 3). Our patient showed no autoantibodies, and superinfection with other viruses was excluded. Negativity of serum HCV-RNA at the time of exacerbation makes it unlikely that HCV caused severe hepatic injury. Itchy eruptions appeared before the increase in the ALT level, and the rapid decrease in serum ALT levels after prednisone administration may suggest an immunologic mechanism induced by interferon, given that he had not received any concurrent drugs or allergens. Close monitoring of liver function test results during interferon therapy is recommended, and combination therapy with prostaglandin E_1 and prednisone at an early phase of severe hepatic injury should be considered, even when no autoantibodies are detected in the serum.

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Hispanics with End-Stage Renal Disease

To the Editor: The recent National Institutes of Health consensus conference statement on the morbidity and mortality of renal

dialysis noted that whereas the incidence of treated end-stage renal disease is dramatically higher for African-Americans and Native Americans than for other racial groups (1), a clinical impression of greater treated incidence in Hispanics could not be confirmed because of the unavailability of data on ethnicity in the United States Renal Data System (2). National data from our 1980s dialysis practice study show that Hispanics accounted for 7.6% of the population of treated patients with end-stage renal disease at that time (3) compared with 6.4% of the resident population (1980 U.S. Census) (4). Among end-stage renal disease subgroups, Hispanics have a 10.0% rate for diabetic nephropathy and a 4.1% rate for hypertensive nephropathy (3), rates consistent with overall ethnic patterns in mortality associated with these underlying diseases (5). Although the subsequent increase in the number of Hispanics in the U.S. resident population (4) will have increased these figures, the findings corroborate the clinical impression cited above (1) by showing a slight over-representation of Hispanics among treated patients with end-stage renal disease.

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In response: On behalf of the members of the National Institutes of Health Consensus Panel that addressed morbidity and mortality in dialysis, I thank Drs. Radecki and Nissenson for drawing our attention to published data that support the clinical impression referred to in the consensus statement that Hispanics are somewhat over-represented among treated patients with end-stage renal disease.

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OBRA Regulations for Nursing Homes—Enhancing Paperwork or Patient Care?

To the Editor: In their commendable review (1), Ouslander and Osterweil describe extensive physician responsibilities but confess that the cost-effectiveness and efficiency of these practices for preventing morbidity and death in nursing homes have never been tested. Instead, the authors rely on guidelines of federal rules for nursing homes, particularly the Omnibus Budget Reconciliation Act (OBRA) of 1987 (2). The authors do acknowledge that OBRA regulations will be difficult to implement in light of the negative "nature of the nursing home environment" and inadequate Medicare reimbursement for physician care.

Medical practice cannot be neatly regulated by government, nor will extensive regulation be an asset in attracting the best physicians to the field of geriatrics. I believe that we should maximize nursing home residents' potential for returning to the community and should prevent unnecessary hospitalizations. Development of a medical unit (3) for the care of acute medical problems within nursing homes might accomplish the latter goal. All systemic infections (short of overwhelming sepsis), uncomplicated cardiac and cerebral vascular events, deep venous thrombosis, and many disorders that traditionally require hospitalization could be treated in such a unit. It would be particularly appropriate for patients with do-not-resuscitate orders. Just treating acute urinary tract infections and pneumonia in nursing homes could prevent more than 2000 hospitalizations nationwide, with savings of more than $1 billion (4).

The diagnosis-related group regulations have freed physicians to prematurely discharge hospitalized patients. As a result, many acutely ill elderly are transferred to nursing and home care facilities that are poorly prepared to care for them. These regulations have exacerbated problems with continuity in our antiquated health care system for the elderly (5). The nursing facility is only one of many components of an integrated, multilevel system that includes day care, day hospitals, home health care, hospice, respite care, geriatric assessment clinics, and rehabilitation units. A multidisciplinary geriatric medical team working in concert would assure continuity of care within the system.

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To the Editor: The excellent review by Ouslander and Osterweil (1) and the accompanying editorial by Besdine and colleagues (2) encourage internists to expand their nursing home practice. The OBRA act is specifically revered by the authors as a "gift" to nursing home patients.

As an internist who devotes half of his practice to nursing home patients, I believe OBRA is responsible for physician reticence to practice in nursing homes. To my mind, OBRA stands for "Oppressive Bureaucratic Requirement Act" because it coerces physicians to devote far more time to completing paperwork and attending meetings than to caring for patients. The motto seems to be "paperwork first, patients last." About 75% of my nursing home time is spent on paperwork, all of which is nonreimbursable. I donate considerable time to charitable causes but do not consider Medicare to be a charitable organization. Contrary to the authors' endorsement, OBRA is not a gift to nursing homes from Santa Claus but is instead a gargantuan Grinch.

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In response: Both Drs. Portnoi and Jameson view OBRA 1987 as an unnecessary and counterproductive bureaucratic intrusion into the practice of geriatric care in nursing homes. Although promoting OBRA was not a primary objective of our review, we do believe that when viewed in proper context, these regulations will foster improved care for nursing home residents. We recognize that many caring physicians like Drs. Portnoi and Jameson struggle to provide high-quality, cost-effective care to nursing home residents in the face of seemingly endless paperwork, needless regulations enforced by often overzealous and sometimes adversarial inspectors, and limited resources.

The OBRA legislation was not intended to regulate physician practice or to create unnecessary paperwork. In responding to

the 1986 Institute of Medicine's report on improving the quality of care in nursing homes, the new regulations focused on residents' rights, quality of care, and the achievement among residents of the highest practicable level of functioning.

We agree with Dr. Portnoi that unnecessary hospitalizations among nursing home residents should be prevented. This is a complicated issue (1). Establishing a medical unit within a nursing home is conceptually attractive but may be financially difficult given current incentives, may detract from the care of chronically ill residents, and may require substantial training and resources for facilities that Dr. Portnoi states are "poorly prepared" to care for subacutely ill patients. We strongly agree with Dr. Portnoi's recommendations for integrated multilevel systems of geriatric care with multidisciplinary approaches to care.

We also believe that basic clinical and health services research in the nursing home (2) on ways to translate research findings into practice in nursing homes (3) is critical to improving care. Only through the cooperative efforts of all geriatricians in creating models of practice, carrying out productive research, summarizing the "state of the art," and engaging in constructive discourse will we meet the challenge of providing optimal medical care to our aging population.

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Ethics Committees

To the Editor: Contrary to Fleetwood and Unger's claims (1), the New York State Task Force on Life and the Law does not propose that physicians receive special or automatic immunity for following the advice of an ethics committee. As part of a broad proposal covering surrogate decisions for patients without decision-making capacity who have not provided treatment directives (2), the Task Force has recommended legal protection for health care professionals who in good faith honor decisions made in accord with the proposal. Such decisions will rarely involve ethics committees, which play only a limited role in the proposed policy and have little if any decision-making authority. Under the proposal, responsibility for health care decisions rests with the patient's surrogate and attending physician. A physician who in good faith honors a surrogate's decision would equally receive legal protection, whether or not an ethics committee is involved.

The Task Force proposal calls for ethics committee participation in some cases of unresolved dispute and in special circumstances, such as those in which a physician judges that life-sustaining treatment should be foregone for an incapacitated patient who has no surrogate. Specific provisions to protect the rights of patients and others address the concerns raised by Fleetwood and Unger. For example, the proposal requires committees to notify all involved parties that a case is being considered, to inform them about the committee and its procedures, and to permit them to present their views to the committee, with the option of being accompanied by an advisor (2).

Fleetwood and Unger note important concerns and rightly emphasize that procedures for ethics committees should be formulated to advance patients' interests. The Task Force's proposal for surrogate decisions, including its specific provisions on ethics committees, addresses these concerns in promoting our shared goal.

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In response: Miller and Mackler's letter illuminates the intent of the New York State Task Force on Life and the Law regarding the authority of institutional ethics committees. Although we are encouraged that the Task Force did not mean to propose giving physicians "special or automatic immunity for following the advice of an ethics committee," we remain concerned about the clarity of their 1992 document (1).

Appendix A of section 11, Bioethics review committees, part C, page 262, states: "Recommendations and advice by the bioethics review committee shall be advisory and nonbinding, except for committee approvals or disapprovals of the withdrawal or withholding of life-sustaining treatment from an emancipated minor patient, from an adult patient without a surrogate, or from any patient who is neither terminally ill nor permanently unconscious." We believe it fair to interpret this statement to mean that advice from a committee is binding when it involves foregoing treatment for the aforementioned patient categories. Our interpretation was echoed in an article in *Hospital Ethics* (2) that stated: "In cases involving life-sustaining treatment decisions, the proposed legislation would make the forgoing of such treatment subject to the review and approval of multidisciplinary institutional ethics committees."

In Appendix A of section 13, Immunity, part 2, page 266, the New York Task Force states, "No health care provider or employee thereof shall be subjected to criminal or civil liability, or be deemed to have engaged in unprofessional conduct, for honoring in good faith a health care decision made pursuant to this article or for other actions taken in good faith pursuant to this article." We infer from this statement that professionals who follow a committee's recommendation receive immunity.

We maintain that the current document implies that immunity is provided for those who follow committee advice in cases involving foregoing life-sustaining treatment for patients without decision-making capacity and without surrogate decision makers. Immunity is not conferred on those who consult a committee about other types of decisions.

Consequently, the "broad protection" that concerns us is conferred in the most troubling cases, including cases in which no one can speak for the patient. We hope the Task Force will clarify this ambiguity.

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Attitudes About Guidelines

To the Editor: The study by Tunis and colleagues (1) and the accompanying editorial (2) shed much light on the increasing popularity of clinical guidelines (particularly in managed care) and on the ambivalent attitudes of physicians who have access to them.

Our profession must prepare for the "Brave New World" of medical practice that is rapidly encroaching on us. In the near future, physicians may conduct all encounters with patients carrying a pocket-sized computer clipboard. The computerized patient records may all be housed in the servers of the payers. When the patient's record is entered into the clipboard, the physician will be connected with the payer's computer system. As data and orders are entered, algorithms, guidelines, and criteria will be applied that, when possible, will alert the physician to

what the payer deems appropriate. The physician may also receive cues related to health maintenance and drug interactions.

The ramifications of this transformation are complex. Many issues of concern are arising, such as quality, privacy, and the physician–patient relationship. On the one hand, the decision support may allow us more time for our patients and may reduce the time required for managing paperwork, data interpretation, and communication. On the other hand, payers may use the "increased efficiency" to push our profession further into assembly-line medicine.

Physicians must become more involved in directing this transformation. More research should focus on studying the effects of this new paradigm on our profession and job satisfaction. It will no longer be merely necessary to be computer literate. Our colleagues will need tools that enable them to effectively interpret the implications of this transformation and negotiate for their interests and those of their patients.

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In response: Dr. Hongsermeier's letter shows the ambivalence of many physicians toward clinical practice guidelines. She presents two contrasting visions of the future: One anticipates that insurers' algorithms will be enforced through pocket computers, and the other envisions clinician-developed practice guidelines being used to improve decisions, reduce paperwork, and enhance patient–provider interactions. These contrasts are also prominent in our survey, in which physicians perceived guidelines as potentially educational and coercive, quality-driven and economically motivated, and convenient and disciplinary (1).

The "Brave New World" envisioned by Dr. Hongsermeier assumes that health insurers will control the development and enforcement of clinical guidelines. In our survey, 94% of respondents reported low confidence in guidelines issued by a major national insurer, possibly because of concerns about the economic bias of insurers and a loss of autonomy in clinical decision making.

We doubt that the quality of a guideline and the validity of its recommendations can be discerned solely by knowing who developed it. Many guidelines developed by professional societies are based on weak methods and incomplete literature reviews. Some insurers, on the other hand, have facilitated the development of solid, evidence-based guidelines. In fact, the Blue Cross and Blue Shield Association has sponsored guidelines developed by the American College of Physicians, an organization whose guidelines inspired confidence in 80% of physicians in our survey.

This suggests that negative attitudes toward guidelines partially reflect a mistrust of insurers' having a role in determining clinical appropriateness. But this shift in who controls medicine is a phenomenon that goes well beyond guidelines. Guidelines are not transforming the organization, financing, and management of American medicine, although this transformation has probably fueled interest in guidelines. Indeed, much of the ambivalence toward guidelines may reflect tensions arising from a new resource allocation imperative in clinical decision making.

Whatever the cause of concern regarding guidelines, we agree that physician participation in guideline development and implementation is vital. Given that resource allocation considerations will become ever more prominent, physician (and patient) participation in guideline development is necessary to ensure that clinical policies are grounded in valid clinical evidence and are responsive to the needs and preferences of patients.

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