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- Type with double-spacing
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The 28th Amendment

To the Editor: I was intrigued by Davidoff and Reinecke's editorial proposing a constitutional amendment that states "All citizens and other residents of the United States shall have equal access to basic and essential health care." I think discussion and possible passage of such an amendment would indeed help to clarify the debate about how to restructure our health care delivery system.

I offer a slight modification to the wording of the proposed amendment: "All citizens and other residents of the United States shall have equal access to basic and essential health and legal care."

If we are to embark on a national debate about making health care a constitutionally guaranteed right, there would be welcome symmetry in doing the same for the legal profession. To be sure, lack of access (for whatever reason) to legal counsel for such basic services as review of contracts and drawing up of wills can be as ruinous to the life of our citizenry as lack of access to medical care. It would be as ambiguous to decide what constitutes "basic and essential" legal care as it will be to decide what constitutes "basic and essential" medical care. It would also be refreshing, and very beneficial in the end, to see the legal profession have to take a long hard look at its own tattered "delivery system," with its obvious inequities and inadequacies. To see medical and legal professionals doing this side by side would be inspiring.

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Reference

1. Davidoff F, Reinecke RD. The 28th Amendment [Editorial]. *Ann Intern Med.* 1999;130:692-4.

To the Editor: I must take issue with Davidoff and Reinecke's editorial (1). I do so tentatively, however, wondering whether I have made the mistake of taking seriously a proposal intended to be tongue-in-cheek.

The reference to slavery and the 13th Amendment is inapposite. With regard to slavery, one could argue that what "put it right once and for all" was not so much the 13th Amendment as the Civil War, which made that amendment possible. What made passage of the 13th Amendment legally necessary was the fact

that the framers of the Constitution had explicitly accommodated the "peculiar institution" (2) in the text of the Constitution. However messy the authors believe the U.S. health care system to be, the mess is not explicitly provided for in the text of the Constitution.

Nor is the proposed right of "equal access to basic and essential health care" analogous to the Sixth Amendment right to the assistance of counsel. In the latter case, the right of the accused to counsel (and the state's duty to provide the same) arises because the accused finds himself or herself involved in a process, initiated by the state, the goal of which is to deprive the accused of his or her life, liberty, or property. In the case of health care, it is not state action that threatens patients but rather their disease. The two scenarios would be analogous only if we had a fault-based system of health care, wherein care was denied to those responsible for their own illnesses.

We are told that the proposed amendment would serve as a catalyst for the forging of a political consensus on the meaning of "basic and essential health care" and how to provide it, but let me suggest that that is precisely what such an amendment would not do. Instead, having constitutionalized the health care issue, we would see an endless stream of cases flowing through the federal courts, challenging both state initiative and the lack of state initiative in this area.

The proposed amendment, with its tenuous guarantee, is not so much simple as simplistic. I have been around camels only once, but it was long enough to know that they stink. I suspect that the authors are wrong about how their camel would be received, and I doubt that such a "rough beast" would be welcome in the tent of persons with a discerning nose.

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2. Stamp KA. *The Peculiar Institution: Slavery in the Ante-Bellum South.* New York: Vintage Books; 1956.

To the Editor: I feel compelled to comment on the editorial on the 28th Amendment (1). I believe that a constitutional amendment guaranteeing health care is a bad idea. Bureaucrats and politicians will control health care even more than they do now, to the detriment of patient care and physicians' sanity. A 28th Amendment as described by the authors would burden individual practitioners with more and more intolerable demands and restrictions.

I learned very early in life that for every right there is a responsibility. If the U.S. government truly wishes to have any say in how health care is delivered in this country, they must take responsibility for the outcome. I personally would favor being a salaried employee of the government under the following conditions: 1) Immunity from medical malpractice liability (as a government employee, I could not be sued for medical mishaps; rather, the federal government would be responsible); 2) a salary commensurate with my education, training, and experience; 3) 4 weeks' vacation, 2 weeks' educational leave, 7 personal days, and 14 sick days per year; 4) a retirement plan in which 15% of my gross income was placed in a mutual fund or other investment vehicle of my choosing; 5) full retirement at age 65; 6) a disability insurance plan with coverage similar to the one I have now. You get the picture. If I have to be an employee, I expect not only the restrictions of an employee but also the benefits. If I am to remain an employer and an individual practitioner of medi-

cine, I wish to be free of the crazy micromanagement inherent in managed care and the Medicare program.

This 28th Amendment would be pure folly, causing an unprecedented flight from the profession of dedicated physicians who simply cannot take any more abuse. The government and, more specifically, Medicare, have already inundated the medical profession with bizarre regulations and dictates. The latest government-sponsored insanity is the deputizing of senior citizens to become “Medicare cops,” turning in their physicians for a bounty of up to \$1000. One of Murphy’s laws reads: “Things could always be worse.” Davidoff and Reinecke’s editorial is just an example of how bad things could be with yet another government rule.

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Reference

1. Davidoff F, Reinecke RD. The 28th Amendment [Editorial]. *Ann Intern Med.* 1999;130:692-4.

To the Editor: Davidoff and Reinecke’s editorial (1) discussed access to health care coverage and health care as a right. This discussion is not about access; it is about coverage. Everyone has access to any emergency department or doctor’s office. Paying for the medical services is the actual issue. Providing universal coverage of health care is not the right approach because it requires forcefully taking and redistributing resources from others. This violates our constitutional rights. Taking from productively earned resources and redistributing to persons who are not working hurts everyone. Society in effect is rewarding those who do not have health insurance and punishing those who do. Charity is an option to provide health care and works by choice, not coercion; it arises from those who can most easily give their resources away.

In addition, changes in health care systems should be initiated locally. Every community has different needs and will best work through its issues if allowed to do so itself. National decrees often do not fit well with community goals and objectives. Bureaucracy and lack of accountability would mark health insurance coverage administered through the national government. If choosing to provide help for those without health coverage, an individual should focus his or her efforts on the training and education necessary for job placement, earned income, and purchase of health insurance coverage. Most important, he or she should work with the persons in communities who have jobs, helping them plan for their futures and preventing them from getting to the point where they are jobless without insurance. This is where our energies should be directed.

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Reference

1. Davidoff F, Reinecke RD. The 28th Amendment [Editorial]. *Ann Intern Med.* 1999;131:692-4.

In response: Dr. Neubauer’s suggestion that all citizens be granted equal access to legal services does seem reasonable. Access to legal services issue is a whole other debate, however, since legal services are not exactly analogous to health care.

Our editorial was not “tongue in cheek,” as Dr. Paola suggests; these are serious, indeed life and death, issues. At the same time, he did pick up on a hint of satirical tone, in the tradition of Jonathan Swift’s “A Modest Proposal.” As he suggests, the 13th Amendment did neutralize the recognition of slavery in the original Constitution. In making the point, however, he underscores the Constitution’s concern with social and economic issues, not just intrusions of the state. As to his concern that a constitutional amendment would overburden the courts with challenges to this initiative, we already have a seemingly endless stream of legal cases arguing the right to specific treatments for specific diseases, in addition to those dealing with perceived misadventure. At least some of the legal “new” challenges under a Constitutional

amendment would be on the broad grounds of general access, some encompassing class actions.

Dr. Romano has somehow concluded that the amendment we propose would automatically convert all physicians into salaried federal employees. What he has read into our proposal is exactly what the amendment is intended not to do. It would establish only the “what”—the fact of access—but leave the “how”—financing, administration, delivery of services—entirely up to the states, the cities, the marketplace, and the professions.

Dr. Anderson equates “access” with financial coverage. We agree that the financing of health care is crucial, but it is certainly not the only element of access that matters. Excellent insurance coverage means nothing if facilities, providers, and knowledge aren’t available. As to forcing the redistribution of resources from those who have “earned” it to those who haven’t, Dr. Anderson has got his figures wrong: The great majority of the medically uninsured (and underinsured) are employed. We are talking equity here, not charity.

More than 10 states now have laws that guarantee access to complementary and alternative medical practices. Thus, on the one hand, we face an additional irony: a willingness to legislate access to special forms of health care in response to the demands of special interest groups, while we have lacked the will to assure access, at least so far, to even the most basic medical services as a matter of equity. On the other hand, legislative action on universal access is actually beginning to happen: At the state level, for example, an amendment to the Illinois state constitution has now been proposed that would establish health care as a right. Referred to as the “Bernadin Amendment” (in honor of the late Joseph Cardinal Bernadin), the amendment reads:

“Health care is an essential safeguard of human life and dignity, and there is an obligation for the State of Illinois to ensure that every resident is able to realize this fundamental right. On or before May 31, 2002, the General Assembly by law shall enact a plan for universal health care coverage that permits everyone in Illinois to obtain decent health care on a regular basis.”

The chief sponsor of the amendment is Representative Mike Boland, at 605 17th Avenue #2, East Moline IL, 61244; fax, 309-752-7186.

At the national level, Representative Chaka Fattah (D, Pennsylvania) is planning to introduce the Health Care Access Assurance Act of 1999 in the upcoming session of Congress. The act would “require each State to provide a minimum level of access to health care to all citizens of such State as a condition for participation in federal health care funding program.” Mr. Fattah’s office can be reached at 1205 Longworth House Office Building, Washington, DC 20616; phone, 202-225-4001.

Straws in the wind? We can hope so.

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Editor

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The Hospitalist Movement

To the Editor: I am deeply disturbed by the articles in the supplement on hospitalists (1). This concept was developed by the insurance industry for economic reasons, and it appears to be gaining support from leadership of the American College of Physicians–American Society of Internal Medicine. The hospitalist concept significantly diminishes and devalues us as a specialty and in the eyes of the patient community. During a patient’s hospitalization, we demonstrate our greatest worth to our patients, and they remember us most for helping guide, care, and advocate for them. This is especially important at a time when many patients believe that the interests of the physician community are divided between the patient and insurance companies.

Daily interaction with residents, colleagues, and hospital staff is an essential aspect of who we are as specialists in internal medicine. We are viewed as resources because of our expertise in

hospital care. We offer a comprehensive perspective on our hospitalized patient. If we allow the hospitalist concept to take root and help validate it, we will be playing a pivotal role in redefining ourselves simply as “primary care providers” (physician and non-physician) delivering only outpatient care. In the eyes of the public and the insurance industry, little will differentiate us from the other members of this category.

We were deemed primary care providers by the insurance industry. We earned our board certification in internal medicine. It is incumbent upon our leadership to vigorously defend and advocate who we are as internists and the importance and special role we play in the overall care of our patients. Shame on the leadership of the American College of Physicians–American Society of Internal Medicine if it fails to recognize this and helps diminish, devalue, and perhaps destroy the specialty that we have chosen as our careers.

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Reference

1. **Wachter RM, Goldmann DR, eds.** The Hospitalist Movement in the United States. *Ann Intern Med.* 1999;130(4 Pt 2):337-88.

To the Editor: I enjoyed reading the recent supplement (1) and Dr. Goldmann’s accompanying editorial regarding the hospitalist movement (2). As a self-confessed cynic of this brave new world, my concerns center on the fact that this movement seems based on an effort, as Dr. Goldmann states, to develop “greater efficiency, decreased length of stay, significant cost savings, and improved quality of care.” Is it not remarkable that “improved quality of care” appears almost as an afterthought to financial pressures?

Another of my other concerns is that hospitalists are, in fact, physicians who are mostly “newly trained and just beginning to forge careers” (2). Is it not possible that, sometime in the not-too-distant future, hospitals and health care systems will determine that all of the cost savings possible from hospitalists have been realized and that the movement is no longer necessary? If so, what will become of these physicians? Will the efficiency of their ambulatory skills have deteriorated to the extent that they find themselves no longer marketable?

Physicians and our professional organizations have a responsibility to remain skeptical of any effort that is driven primarily by costs rather than by quality. I join Dr. Goldmann’s call for continued evaluation of this movement. I hope that hospitalists will always be able to reduce costs, provide the best quality of care, and enjoy a great degree of professional satisfaction. However, my years of experience in the health care delivery system lead me to suspect otherwise.

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1. **Wachter RM, Goldmann DR, eds.** The Hospitalist Movement in the United States. *Ann Intern Med.* 1999;130(4 Pt 2):337-87.
2. **Goldmann DR.** The hospitalist movement in the United States: what does it mean for internists? [Editorial] *Ann Intern Med.* 1999;130(4 Pt 1):326-7.

To the Editor: Dr. Wachter (1) extols the benefits of “specialized care” for many situations, including the care of complex diseases, such as AIDS. As proof, he cites the 1996 study by Kitahata and colleagues (2). However, the study he cites compared generalists who had varying levels of experience; it did not compare specialists with generalists. In fact, the threshold of experience that the “specialists” had in this study was low: only 5 patients with AIDS.

As this example illustrates, it is reasonable to argue that increased experience is likely to improve outcomes, but that is not necessarily the same thing as “specialization.” The questions are, Does this effect plateau at a certain level? If so, at what level? Dr. Wachter arbitrarily sets this level at 25% of time (probably to include academic hospitalists such as himself) and argues that

this is qualitatively different from the 12% of the time that an “average” physician spends. If 25% is better than 12%, why stop at this level? If specialization is the answer, why not 75% or, for that matter, 100%?

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2. **Kitahata MM, Koepsell TD, Deyo RA, Maxwell CL, Dodge WT, Wagner EH.** Physician’s experience with the acquired immunodeficiency syndrome as a factor in patients’ survival. *N Engl J Med.* 1996;334:701-6.

To the Editor: Central to the debate about the hospital movement are medical inpatients who do not require a specialized unit and often have no affiliated primary care physician. Vying for the opportunity to care for these patients are the hospitalists, most of whom are internists who have chosen the hospital as their practice site (1). Of interest, Schroeder and Shapiro (2) view hospitalists as “fleeing” the “people side” of medicine for the technology of tertiary care. It is my experience as a hospitalist on a teaching service at University of Pennsylvania Medical Center that I provide as much primary care as tertiary care.

For too long, overburdened residents were responsible for the medical care of hospitalized patients in academic centers. Hospitalists at my institution attempt to assume that responsibility. They now direct care that encompasses communication, education, implementation of best practices, coordination of specialty care, assistance with transitions to home or intermediate care, and end-of-life care for patients and families who cannot face death in their homes. These roles are not at odds with that of internists in the ambulatory setting but rather are remarkably aligned and complementary.

Among the variety of roles suggested by Dr. Goldman (3), hospitalists in academic medical centers have many goals: primary care of indigent persons, general medical care for all hospitalized patients, education of residents and students with respect to evidence-based use of hospital technologies and services, and clinical research. The tension between inpatient and outpatient internists seems to be artificial, created more by financial considerations than divergent concepts of patient care. If the debate is reduced to market share and turf wars, then it is hospitalized patients who will lose.

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1. **Lindenauer PK, Pantilat SZ, Katz PP, Wachter RM.** Hospitalists and the practice of inpatient medicine: results of a survey of the National Association of Inpatient Physicians. *Ann Intern Med.* 1999;130(4 Pt 2):343-9.
2. **Schroeder SA, Shapiro R.** The hospitalist: new boon for internal medicine or retreat from primary care? *Ann Intern Med.* 1999;130(4 Pt 2):382-7.
3. **Goldman L.** The impact of hospitalists on medical education and the academic health system. *Ann Intern Med.* 1999;130(4 Pt 2):364-7.

To the Editor: The hospitalist movement is in its infancy, with few programs experienced enough to determine the necessary components of hospitalist training. At Long Island Jewish Medical Center, three years of evolution have provided our hospitalist group with an opportunity to observe many facets of the process of inpatient care. Like Goldman’s (1), we also think that a career path in hospital medicine is viable. The many roles a hospitalist performs extend beyond the wards, and it is clear that hospitalist development is a lengthy process and that individual career development must be nurtured through continued education and training. Our “pathways” program allows hospitalist faculty to excel in research, education, or quality improvement. Although research and education are not unique to hospitalists, the daily activities of hospitalists make both inpatient outcomes study and medical education opportunities for innovation. For example, we

are designing an inpatient teaching curriculum for the medical housestaff.

The ideal model of a hospital-based physician with expertise in critical pathways, data analysis, and re-engineering skills is still developing, and the optimal training program for such a physician is still unclear. We advocate a modified 3-year categorical internal medicine program as an appropriate base on which to build a career (2). Concepts of quality and resource utilization, for example, are foreign to most recent medicine graduates. The value-added service of nonclinical activities will largely determine whether hospitalists will become a permanent part of U.S. medicine.

We have also learned that burnout is a reality. Accordingly, we have adjusted our "on-service" time from 12 to 8 to 6 months. If growth and development of academic faculty are desired, appropriate time must be allocated.

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1. **Goldman L.** The impact of hospitalists on medical education and the academic health system. *Ann Intern Med.* 1999;130(4 Pt 2):364-7.
2. **Wartman SA, Silver AL, Walerstein SJ, Flansbaum B, Conner D.** The hospitalist movement: an education and research agenda. *New Medicine.* 1999;3:11-6.

In response: Dr. Starr raises legitimate concerns regarding the impact of hospitalists on internal medicine, notwithstanding our finding that 89% of hospitalists are internists (1). He may be relieved to learn that the earliest hospitalist groups were developed by physicians; insurance companies are only recent converts. Moreover, most hospitalist programs are voluntary; the independent choice of physicians like Dr. Starr is fueling the movement. He will be heartened by the vigorous stance that the American College of Physicians–American Society of Internal Medicine and the National Association of Inpatient Physicians have taken against the mandatory use of hospitalists by insurance companies (2). Finally, perhaps he will recognize that average internists now spend more than 85% percent of their time caring for ambulatory patients, even when there are no hospitalists. Internists can no longer depend on their hospital work for education or for demonstrating their competitive edge over other providers.

I applaud Dr. Hoffman's skepticism. The data on the value of hospitalists are positive (3–5), but more research is needed. Although I too worry about burnout, our survey of nearly 400 hospitalists showed that 94% were satisfied and few anticipated returning to primarily ambulatory practices (1). A generation ago, internists who focused on emergency or critical care medicine were probably counseled not to lose their ambulatory skills so as to keep their options open in case their new "specialties" evaporated. Few such physicians were forced to return to outpatient practice, and I suspect the same will be true for hospitalists.

Dr. Rastegar is right to question the 25% threshold for inpatient care in my hospitalist definition. In fact, about two thirds of the hospitalists we surveyed were fully hospital-based (1). However, a definition of 100% or even 50% inpatient time would exclude most academic hospitalists. At my institution, hospitalists serve in the inpatient role for 3 to 6 months per year; this seems to generate the experience, expertise, and investment in the inpatient service needed to meet the spirit of the definition. We also benefit from the participation of a group of nonhospitalists on our medical service, although they continue to be somewhat less efficient than hospitalists (3; Auerbach A. Unpublished data). Our hospitalists generally maintain a small outpatient presence, which may help prevent burnout and improve their sensitivity to ambulatory issues. Research is needed to determine whether this outpatient time is sustainable and to define the "optimal" amount of inpatient practice time for hospitalists.

The experiences of Wald and Flansbaum and colleagues reflect my own and those I've observed at dozens of academic and community hospitals. In these programs, once the turf battles are

put aside, reasonable job descriptions constructed, communication links forged, and sustainable support obtained, patients and trainees benefit from the presence of hospitalists who can hone their acute care skills, remain available to hospitalized patients throughout the day, and make continuous improvement in the inpatient environment part of their daily agenda. Moreover, academic hospitalists are forging exciting educational pathways and undertaking research to answer important questions about the hospital and its interfaces. The value of these efforts, combined with data on the costs and quality of hospitalist versus nonhospitalist care, will determine the viability of this new field.

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2. **Maguire P.** Use of mandatory hospitalists blasted. College, others protest plans that force doctors to give up inpatient care. *ACP Observer.* 1999;19:1.
3. **Wachter RM, Katz P, Showstack J, Bindman AB, Goldman L.** Reorganizing an academic medical service: impact on cost, quality, patient satisfaction, and education. *JAMA.* 1998;279:1560-5.
4. **Stein MD, Hanson S, Tammamo D, Hanna L, Most AS.** Economic effects of community vs. hospital-based pneumonia care. *J Gen Intern Med.* 1998;13:774-7.
5. **Diamond HS, Goldberg E, Janosky JE.** The effect of full-time faculty hospitalists on the efficiency of care at a community teaching hospital. *Ann Intern Med.* 1998;129:197-203.

In response: As noted by Flansbaum and colleagues, we still have much to learn about hospital medicine as an academic career, especially with regards to teaching and burnout. The University of California, San Francisco, academic hospitalist model emphasizes the concurrent roles of both teaching-attending and physician-of-record for essentially all patients on a ward team (1). Given this key role in education as well as clinical care, burnout would have an academic impact extending well beyond the job satisfaction of the individual hospitalist. Because of these high stakes for our entire program, we believe that 6 months of acute care inpatient work is the absolute maximum for an academic hospitalist with concurrent inpatient teaching-attending responsibilities.

In this academic model, the limitation of inpatient "systole" to 4 to 6 months puts substantial pressure on the individual and the system to find meaningful, remunerative activities for the remaining months of "diastole." For some of our hospitalists, diastole includes a significant role as an ambulatory clinician and clinical teacher. For many others, however, diastole focuses on leadership of key teaching programs, inpatient consultation, a hospital administrative role, or funded research. Much like the cardiac cycle, hospitalist diastole is an active process that uses substantial energy, although less intensively than during systole.

Chronic tachycardia produces a cardiomyopathy that is due to catecholamine overstimulation and is exacerbated by inadequate diastole. Cardiomyopathy is often complicated by diastolic dysfunction as well as systolic dysfunction. In my opinion, the durability of hospitalist careers, and hence our ability to attract and retain outstanding physicians to such careers, will be partly related to solving the challenge of systolic load but will be even more dependent on diastole's duration, sustainability, value, and remuneration.

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Reference

1. **Wachter RM, Goldman L.** Implications of the hospitalist movement for academic departments of medicine: lessons from the UCSF experience. *Am J Med.* 1999;106:127-33.

Physiologic Left Ventricular Cavity Dilatation in Elite Athletes

To the Editor: Pelliccia and colleagues (1) described a subset of highly trained athletes with a striking enlargement of the left ventricular cavity. They found that 14% of study participants had left ventricular cavity dimensions compatible with primary dilated cardiomyopathy in the absence of systolic dysfunction, and they suggested that this resulted from an extreme physiologic adaptation to intensive athletic conditioning. Although all athletes in this series denied the use of illicit drugs, data on testing for anabolic steroids might have been informative. These drugs are commonly used to enhance athletic performance and muscle development, and athletes may have been discouraged from volunteering such information to the medical division of the Italian National Olympic Committee.

Ventricular remodeling, hypertension, myocardial ischemia, and sudden cardiac death have each been temporally and causally associated with anabolic steroid use in humans (2–5). These effects are known to persist long after steroid use has been discontinued. The prevalence of left ventricular enlargement without systolic dysfunction in athletes using anabolic steroids is not known. Thus, we cannot exclude the possibility that the morphologic remodeling described by Pelliccia and colleagues may have been partially determined by the use of anabolic steroids.

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1. Pelliccia A, Culasso F, Di Paolo FM, Maron BJ. Physiologic left ventricular cavity dilatation in elite athletes. *Ann Intern Med.* 1999; 130:23-31.
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4. Ferrera PC, Putnam DL, Verdile VP. Anabolic steroid use as the possible precipitant of dilated cardiomyopathy. *Cardiology.* 1997;88: 218-20.
5. Ferenchick GS. Association of steroid abuse with cardiomyopathy in athletes. *Am J Med.* 1991;91:562.

In response: Dr. Ribeiro asks whether the marked left ventricular remodeling we described in elite athletes may have been partially determined by anabolic steroid use. This intriguing question also suggests that the substantial left ventricular cavity dilatation present in some highly trained athletes may be not an entirely physiologic phenomenon and possibly an expression of a pathologic condition. Athletes examined as a part of our medical program at the Institute of Sports Science in Rome have denied the use of all illicit drugs, including anabolic steroid hormones; however, actual testing for these drugs is not a routine part of our program. Although we cannot exclude the possibility that some athletes may have been taking steroid hormones, it seems unlikely that a substantial proportion of cyclists, cross-country skiers, and rowers/canoists (the athletes with the most marked left ventricular cavity enlargement) have been using anabolic steroids over the long term to enhance performance. Excellence in these disciplines primarily requires high aerobic power and anaerobic threshold, which are not likely to be enhanced by long-term use of anabolic steroid hormones.

Furthermore, the precise effect of these hormones on left ventricular structure remains unresolved. In fact, although a mild increase in left ventricular wall thickness and mass has been described in power-trained athletes taking steroid hormones, no significant effect on left ventricular cavity has been reported (1–4). Therefore, it seems unlikely that the marked left ventricular cavity enlargement we described represents the consequence of anabolic steroid abuse.

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2. Urhausen A, Holpes R, Kindermann W. One- and two-dimensional echocardiography in bodybuilders using anabolic steroids. *Eur J Appl Physiol.* 1989;58:633-40.
3. De Piccoli B, Giada F, Benettin A, Sartori F, Piccolo E. Anabolic steroid use in body builders: an echocardiographic study of left ventricle morphology and function. *Int J Sports Med.* 1991;12:408-12.
4. Thompson PD, Sadaniantz A, Cullinane EM, Bodziony KS, Catlin DH, Torek-Both G, et al. Left ventricular function is not impaired in weight-lifters who use anabolic steroids. *J Am Coll Cardiol.* 1992;19: 278-82.

Hand Hygiene in Hospitals

To the Editor: May I add to the list of reasons why health care workers do not wash their hands as frequently as they should, as referred to in Pittet and colleagues' paper (1) and in Dr. Boyce's editorial (2)? The list should include the following: too few hand-washing basins and poor access to them; antiquated faucets that are hard to turn on and off, do not easily mix hot and cold water, and do not allow hands to be comfortably washed under a gentle warm stream; a hot water supply whose temperature has, through heightened and possibly exaggerated fear of legionella contamination, been raised too high; soap and towels that are not regularly replenished by designated staff; and a lack of microbiology education, the result of which is staff members who do not understand when and why to wash their hands.

These deficiencies are more the responsibility of management than of individual staff members, and it is often institutional skimping on facilities and staffing that provides the excuses for neglect of handwashing. Dr. Boyce refers to Semmelweis in his editorial; perhaps what is now needed is a formal comparison of outcomes in groups of surgical wards where disinfection by handwashing between each procedure is enforced and other groups of wards where there is no intervention. If the results are as expected, management will learn the price that poor hand hygiene exacts in morbidity and extended postoperative stays. Management staff may then be persuaded to create the conditions in which correct handwashing procedure will be observed.

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2. Boyce JM. It is time for action: improving hand hygiene in hospitals [Editorial]. *Ann Intern Med.* 1999;130:153-4.

Misdiagnosis of HIV Infection

To the Editor: There is a continuing effort to detect HIV in the acute phase of the infection. Because the antibody response takes time to develop, the direct detection of a viral component is expected to be more sensitive than antibody detection. Rich and colleagues (1) described three patients with false-positive results on viral load assays. This report merits consideration of certain issues pertinent to all diagnostic tests.

When a test has more than two possible outcomes, its accuracy can be reported as pairs of sensitivity and specificity corresponding to each degree of abnormality. This approach, the basis for receiver-operating characteristic analysis, maximizes the use of diagnostic information (2). When the diagnostic threshold is set at a lower degree of abnormality, the sensitivity of the test tends

to increase but its specificity tends to decrease. The opposite occurs when a higher diagnostic threshold is selected. In the case of HIV viral load assays, if more viral units must be detected to report the test result as abnormal, the specificity will increase. As noted by Rich and colleagues, "the lowest reported plasma viral load during seroconversion is more than 17 times higher than the highest viral load detected in our three patients." Thus, for the diagnosis of acute infection, the threshold should probably be set much higher.

In addition, case reports are not a substitute for prospective studies that assess a diagnostic test in a blinded fashion against a validated gold standard, and the pretest probability of the disease plays a fundamental role in the interpretation of a test result. Patients with a very low pretest probability of disease and an abnormal test result will not have a high post-test probability, no matter how accurate the test might be (3).

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To the Editor: Rich and colleagues (1) described three patients given a false diagnosis of HIV-1 infection on the basis of falsely positive HIV-1 plasma viral load test results. The authors urged caution in using HIV-1 plasma viral load testing to diagnose HIV infection. We agree, and urge caution for the converse: Viral load testing should not be used to exclude a diagnosis of HIV-1 infection. We reviewed the charts of the 324 seropositive patients enrolled in our HIV clinic and found 13 patients (4%) receiving no antiretroviral therapy who had plasma viral loads less than 400 HIV RNA copies/mL; 6 of these patients had viral loads less than 50 HIV RNA copies/mL according to an ultrasensitive assay (Table). Viral load testing was done with the reverse transcriptase polymerase chain reaction (PCR) assay (Amplicor, Roche, Basel, Switzerland). Of note, each patient also had a relatively preserved immune status on the basis of absolute CD4 counts, percentage of CD4 cells, and ratios of helper to suppressor cells.

It is conceivable that if these 13 patients had been screened for HIV-1 infection using plasma viral load testing or CD4 subsets, the diagnosis of HIV-1 may have been missed. Because enzyme-linked immunosorbent assays and Western blot testing typically involve a 10- to 14-day turnaround, physicians caring for critically

ill patients may turn to "surrogate" tests (such as PCR or measurement of CD4 counts) to make or exclude a diagnosis of HIV-1 infection. We emphasize that plasma viral load testing should be used clinically to initiate antiretroviral therapy and assess its effectiveness; it should not be used to make or exclude a diagnosis of HIV infection. Of concern, PCR has been proposed as a method for screening blood products (2). Whether any of our subset of patients is infected with *nef*-deleted HIV-1 is not known (3).

Disclaimer: The views expressed in this letter are those of the authors and do not reflect the official policy or position of the Department of the Navy, Department of Defense, or the U.S. Government.

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In response: Dr. Palmas suggests that different levels of diagnostic sensitivity (1) could help minimize false-positive results. Prospective studies can evaluate this issue and quantify the frequency of misdiagnosis of HIV infection by HIV-1 plasma viral load testing. In the meantime, physicians should use caution in diagnosing HIV infection solely on the basis of a detectable HIV-1 plasma viral load. However, we continue to encourage efforts to rapidly and accurately diagnose acute HIV infection. This is a responsibility for all care providers, not just specialists in the field.

Goodman and colleagues describe the converse situation, in which an undetectable HIV-1 plasma viral load is found in persons who are HIV infected but not receiving antiviral therapy. The authors report that 13 of the HIV-infected patients in their cohort had plasma viral loads less than 400 copies/mL and that 6 had an undetectable plasma viral load on ultrasensitive assay. All of these patients had preserved CD4 cell counts and percentage of CD4 cells and normal ratio of helper to suppressor cells. These persons may become long-term nonprogressors, although they have not had a sufficient duration of follow-up (2). Using HIV-1 plasma viral load testing to diagnose HIV-1 infection can

Table. Plasma Viral Loads, CD4 Cell Counts, and Helper:Suppressor Cell Ratios in 13 HIV-Infected Patients*

Patient	Date of Diagnosis	Plasma Viral Load		CD4 Cell Count		Helper:Suppressor Cell Ratio
		Standard PCR Assay	Ultrasensitive PCR Assay	Absolute	Proportion	
		copies/mL		cells/mm ³	%	
1	1992	NT	<40	1104	43	1.30
2	1993	NT	238	714	30	0.63
3	1991	<400	535	645	46	1.77
4	1992	<400	NT	658	38	0.90
5	1998	<400	<25	756	45	1.15
6	1997	<400	28	806	32	1.00
7	1993	NT	<50	993	43	2.39
8	1998	NT	41	650	41	1.05
9	1997	NT	<25	465	43	1.48
10	Unknown	<400	NT	698	32	0.82
11	1994	<400	NT	904	34	1.26
12	1998	NT	184	743	38	1.31
13	1992	<400	NT	630	37	1.03

* NT = not tested; PCR = polymerase chain reaction.

also provide false-negative results. Laboratory data should be interpreted on the basis of the clinical scenario and are not a substitute for vigilant clinical evaluation that combines serologic and plasma viral load testing with clinical follow-up.

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Myocardial Infarction in HIV-Infected Men Receiving Protease Inhibitors

To the Editor: We describe four men with HIV infection who sustained myocardial infarction (two of which were fatal) after 24 to 29 months of protease inhibitor therapy. These observations confirm and extend those of Henry and colleagues (1), and they occurred in a clinic setting in which amelioration of clinical AIDS has been frequently replaced by variable features of the insulin resistance syndrome (lipodystrophy, hyperlipidemia, pancreatitis, diabetes, and, more recently, significant macrovascular disease). The four men (ages 35 to 44 years) were smokers. Two of them (Table) developed new, protease inhibitor-related hypertriglyceridemia and hypercholesterolemia. One of the four patients was hyperlipidemic before protease inhibitor therapy, and another had no lipid abnormality.

Our observations of new-onset diabetes and hyperlipidemia among many of our patients with AIDS receiving protease inhibitor therapy (specifically, hypertriglyceridemia severe enough to be associated with pancreatitis) are in concert with those of others (2-4).

Not all of the features of this protease inhibitor-related metabolic syndrome (hyperlipidemia, lipodystrophy, diabetes, and myocardial infarction) need to appear together. We could not identify which protease inhibitor, if any, may be more likely than others to be responsible. Carr and colleagues (5) have proposed that protease inhibitors as a class, through homology to regions within proteins that regulate lipid metabolism, bind to those regulatory elements. This leads to impairment of hepatic chylomicron uptake and, in turn, to features of the insulin resistance syndrome and diabetes in those susceptible to it. Thus, AIDS, a fatal illness that is routinely and effectively managed with protease inhibitors, now seems to be presenting with potentially serious new risks associated with that therapy.

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Table. Lipid Findings in HIV-Infected Protease Inhibitor Recipients Who Had Myocardial Infarction

Patient	Age	Triglyceride Level		Cholesterol Level	
		Before PI Therapy	During PI Therapy	Before PI Therapy	During PI Therapy
y		mmol/L (mg/dL)			
1	40	2.71 (240)	4.16 (368)	4.19 (162)	5.36 (207)
2	44	1.99 (176)	1.93 (171)	4.24 (164)	4.34 (168)
3	36	1.35 (120)	4.18 (370)	4.55 (176)	6.21 (240)
4	35	2.69 (238)	2.98 (264)	4.94 (190)	4.87 (188)

* PI = protease inhibitor.

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Ocular Venous Occlusion and Hyperhomocysteinemia

To the Editor: De Bruijne and colleagues (1) recently reported that 63.6% of 26 patients with venous ocular thrombosis had either fasting hyperhomocysteinemia or elevated post-methionine loading homocysteine concentrations. This association has been previously published (2), and, although a control group is lacking, this observation suggests a relation between hyperhomocysteinemia and venous ocular occlusion. Whether homocysteine-lowering treatment may prevent this complication remains to be determined. We recently observed a case that may help to address this question.

A 30-year-old man had been treated by peritoneal dialysis since January 1995 for end-stage renal disease caused by focal and segmental hyalinosis. At the start of peritoneal dialysis the fasting total homocysteine concentration was 38 $\mu\text{mol/L}$ (normal, $<15 \mu\text{mol/L}$). Starting in February 1996, dialysis adequacy was poor but the patient declined to undergo hemodialysis. In March 1996, his fasting total homocysteine concentration was 123 $\mu\text{mol/L}$. In April 1996, the patient reported acute loss of vision in his left eye. Branch retinal venous thrombosis was diagnosed. Results of coagulation tests were normal, and the patient had neither diabetes mellitus nor hyperlipidemia. From April 1996 to December 1996, regular ophthalmologic controls showed three episodes of branch retinal venous occlusion in both eyes. Transplantation with a cadaveric kidney done in January 1997 was successful. The patient's serum creatinine level was 95 $\mu\text{mol/L}$ 10 days after transplantation and remained normal. The fasting total homocysteine concentration was 24, 21, and 23 $\mu\text{mol/L}$, respectively, 6, 12, and 18 months after transplantation. No further ocular complication occurred after transplantation and the dramatic decrease in the total homocysteine concentration.

Patients with end-stage renal disease have an increased prevalence of hyperhomocysteinemia (3). In severe hyperhomocysteinemia due to homozygous cystathionine β -synthase deficiency, homocysteine-lowering vitamin supplementation reduces the incidence of atherothrombotic events. Management of this severe form of hyperhomocysteinemia is the paradigm for the treatment of mild to moderate hyperhomocysteinemia. Successful renal transplantation decreases total homocysteine concentrations (4). In our case, this homocysteine-lowering effect was associated with a dramatic improvement in vision. This observation suggests that treatment of hyperhomocysteinemia may prevent venous ocular occlusion.

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Cyclophosphamide-Associated Uroepithelial Toxicity

To the Editor: The urologic complications of cyclophosphamide therapy often limit its use (1). We describe what we believe to be the first case in the literature of isolated upper urinary tract toxicity. This toxicity occurred after a cumulative oral cyclophosphamide 1.4 g, which is lower than any reported to date.

A 72-year-old woman with a 7-year history of Wegener granulomatosis presented with gross hematuria and dysuria of 1 week's duration. She received cyclophosphamide (1.4 g) over 2 weeks as treatment for relapse of the granulomatosis.

On admission, the patient appeared acutely ill but was hemodynamically stable. Urinalysis showed gross hematuria with no casts. Renal ultrasonography showed mild hydroureteronephrosis. Cystoscopy revealed normal bladder uroepithelium and bleeding from the right ureteral orifice. Retrograde ureteropyelography showed marked irregularity of the mucosa of the upper ureter, renal pelvis, and calyces (**Figure**). Renal and pelvic arteriography showed no evidence of pruning. On percutaneous nephroscopy, the uroepithelium was gray and necrotic, with dystrophic calcifications in the extracted fragments. The collecting system was thoroughly irrigated, and copious debris was removed. Subsequently, the gross hematuria resolved and renal function improved.

Acrolein, a liver metabolite of cyclophosphamide, is the cause of cyclophosphamide-induced renal toxicity (2, 3). Urologic complications occur during prolonged contact of acrolein with the uroepithelium. The obstruction at the ureteropelvic junction due to necrosis and sloughing of the renal pelvis uroepithelium played a role in the pathogenesis of isolated upper urinary tract toxicity. Plotz and colleagues (4) defined the cystoscopic findings in cyclophosphamide toxicity as telangiectasia, focal diffuse ulcerations, hemorrhage, and necrosis of the uroepithelium, as evidenced in our patient. The findings on nephroscopy, the therapeutic response to discontinuation of cyclophosphamide therapy, and the normal angiogram argue against rapidly progressive glomerulonephritis. This case highlights the need to evaluate the entire uroepithelial tract for toxicity in the setting of normal cystoscopic findings in patients taking cyclophosphamide.

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CD123^{hi} Dendritic Cell Lymphoma: An Unusual Case of Non-Hodgkin Lymphoma

To the Editor: A new hemopoietic-derived lineage of dendritic cells was recently reported; this lineage is characterized by a particularly high expression of the interleukin-3 receptor α -chain (CD123^{hi}) (1). Unlike Langerhans cells, normal CD123^{hi} dendritic cells migrate through peripheral blood to lymphoid tissues in the absence of inflammatory stimuli; they are found in fetal and adult lymph nodes, tonsils, and thymus. No tumoral counterpart of this dendritic cell subset has yet been identified.

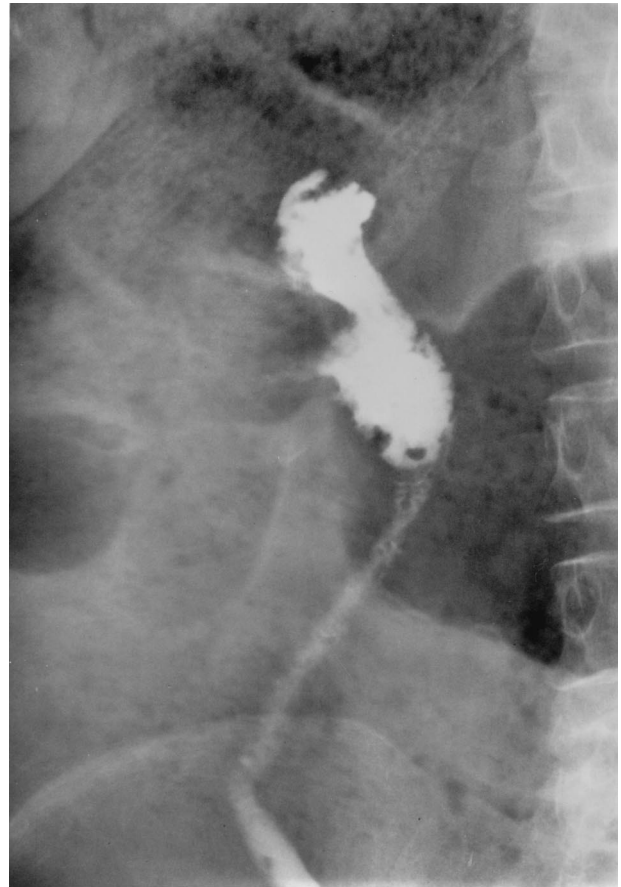


Figure. Top and bottom. Retrograde ureteropyelograms show marked irregularity of mucosa of upper ureter, renal pelvis, and calyces.

We describe a patient with a histopathologic diagnosis of non-Hodgkin lymphoma in whom immunophenotypic and molecular studies ruled out the B, T, or natural killer cellular origin of the neoplastic cells, which exhibited a phenotype identical to that of CD123^{hi} dendritic cells. A 62-year-old man presenting with cutaneous nodules over the right scapula and both supraclavicular and axillary lymphadenopathies was given a diagnosis of high-grade diffuse large B-cell non-Hodgkin lymphoma (REAL [Revised European Lymphoma] classification) (2). Chemotherapy with cyclophosphamide, hydroxydaunomycin/doxorubicin, Oncovin, and prednisone (CHOP) led to transient response by the third cycle, but severe generalized bone pain developed just after the sixth course.

Laboratory evaluation showed leukocytosis (leukocyte count, 9.2×10^9 cells/L, with 10% mononuclear agranular blasts with prominent nucleoli. These cells expressed CD45^{dim}/CD4^{dim}/HLA-DR^{hi}/CD123^{hi}, and lacked both lymphoid (CD3/TCR and CD79a/cIg) and myelo-monocytic (MPO/CD14) markers. The blasts were also CD1a⁺/CD10⁺/CD13⁻/CD20⁺/CD29⁺/CD32^{dim}/CD33⁻/CD34⁻/CD35^{dim}/CD36^{dim}/CD38^{hi}/CD49d⁺/CD54^{dim}/CD55^{dim}/CD59^{hi}/CD62L^{dim}/CD71⁺/CD117⁻/CD122^{dim}/HLA-DP^{hi}/HLA-DQ^{hi}/HLA-ABC^{hi}. T-cell receptor and IgH genes were found in germline configuration, thus excluding the lymphoid origin of this cell population. No cytogenetic abnormalities were detected. Treatment was continued with IMVP₁₆ (3), but

disease progression followed shortly thereafter, with increasing proportion of circulating blasts. The patient died of severe pulmonary infection.

We believe that this neoplasia may represent the neoplastic counterpart of normal CD123^{hi} dendritic cells.

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