CORRESPONDENCE

OUTCOMES AND COSTS OF CARE FOR ACUTE LOW BACK PAIN

To the Editor: The excellent study of the outcomes and costs of care for low back pain by Carey et al. (Oct. 5 issue)[1] found that chiropractors charged considerably more than primary care physicians but had higher scores for patient satisfaction. Neither finding is surprising. Although some chiropractors advise patients to stop coming after their symptoms are gone, others urge them to continue coming indefinitely for "preventive maintenance" or to make the spinal adjustments "hold."[2] Some chiropractic "practice-builders" encourage this and actually give seminars and write about virtually every type of resistance a person might have to "lifetime care."[3] Practice-builders also teach how to increase patients' satisfaction. Whereas some of the techniques recommended are ethically sound (and would be useful for physicians as well), others (such as routinely sending birthday cards) are emotionally seductive.[2]

Many chiropractors appear thorough because they ask large numbers of irrelevant questions (such as whether patients had any falls during childhood) and perform large numbers of orthopedic and neurologic tests that are either redundant or not pertinent. It would be interesting to know whether patients' degree of satisfaction was related to being told that problems were due to "subluxations" (a chiropractic term for misaligned vertebrae) that the chiropractor (with heroic effort) purported to correct.

I have observed the chiropractic marketplace closely for over 25 years. Many chiropractors prescribe vitamin products or homeopathic remedies to many of their patients. About 75 percent prescribe vitamin supplements,[4] and 36 percent dispense homeopathic treatments.[5] It would be interesting to know whether Carey et al. detected any such use in their study. It would also be interesting to know whether there were any differences in practice style between the chiropractors who agreed to participate in the study and those who did not. I suspect that the participants offer more rational care.

STEPHEN BARRETT, M.D.
P.O. Box 1747
Allentown, PA 18105

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To the Editor: Physicians who treat acute low back pain already know that it is usually self-limited and almost always resolves in a few days to a few weeks, leaving no residual effects, regardless of the treatment rendered. Radiography usually fails to reveal specific disease. Magnetic resonance imaging is rarely indicated in the acute phase and often reveals "abnormalities" that do not correlate with clinical symptoms or findings.

The article by Carey et al. has already been interpreted by the media as finding that the treatments provided by orthopedic surgeons, chiropractors, and primary care physicians are of equal value. In fact, however, the paper is flawed in that the practitioners involved "were aware of the overall purpose of the study."

Furthermore, patients were included in the study after they had selected practitioners, a method that obviously yielded results reflecting the patients' biases and expectations about the

optimal type of treatment. The authors provide no assurance that the study patients were given the same treatment that they might have been given in a nonstudy environment. . . .

Many patients treated for low back pain are victims of work-related accidents or motor vehicle accidents. They are different from those whose back pain develops at home or in other situations in which a third party is not involved, and they have different outcomes.

In the real world, assessment is further complicated by the fact that plaintiffs' attorneys often refer their clients to practitioners who provide favorable diagnoses (with a poor prognosis) and render prolonged and expensive treatment. In each of the fields described, there are wide variations among both practitioners and patients.

ROBERT A. GOLDSTONE, M.D.
140 Chestnut St.
Ridgewood, NJ 07450

To the Editor: None of the various types of providers studied by Carey et al. produced appreciably better outcomes than the others in patients with acute low back pain. The unavoidable conclusion is that the treatments offered by all were ineffective. On the other hand, it is unacceptable to abandon the 31 percent of patients who had not completely recovered after six months, or the 5 percent who remained functionally disabled after that time.

It should not be news nowadays that aggressive treatment is not indicated during the first couple of weeks of low back pain, that early mobilization is helpful, and that radiography offers little benefit initially to patients with uncomplicated back pain. But I constantly see that these facts are still news to many providers.

ERIC T. SHEBAR, M.D.
MVA Center for Rehabilitation
Springfield, MA 01104

To the Editor: Any veteran family physician has learned by experience that except for the temporary relief of pain, there is no medication, physiotherapy, or "manipulation" that alters the course of the usual musculoskeletal pain syndromes. Experienced physicians have also learned that if they share this knowledge with their patients, they will frequently lose them to chiropractors.

JOHN E. HAMPTON, M.D.
10 Kinnaman Ave.
Washington, NJ 07882-9297

Dr. Carey replies:

To the Editor: Dr. Goldstone is concerned that the patients seeing each type of provider may have differed somewhat because they selected their own practitioners. We measured multiple factors at base line, and the differences among the patients seen by different types of practitioners were small.[1] To control for possible selection bias, we excluded patients who were referred (an issue in the case of orthopedic surgeons) or had chronic back pain (an issue for doctors of chiropractic). When we controlled for factors such as the presence of sciatica and base-line functional status, the differences among groups of patients narrowed, indicating that further control for the severity of low back pain would be unlikely to reveal substantial differences among groups. Although it is possible that practitioners altered their practice patterns because they knew they were in a study, we found little evidence, for example, of reduced use of radiography by orthopedic surgeons and chiropractors — a controversial issue in those specialties. Our study is not unique. A study by Shekelle and colleagues, which we cited, found similar patterns of visits and expenditures in the RAND Health Insurance Experiment, in which the type of care given for back pain was not a primary outcome and changes in physicians' behavior would therefore not be an issue.[2]

Dr. Shebar urges us not to "abandon" patients with chronic back pain. We of course agree. Large studies have noted that the majority of the social and health care costs for low back pain are incurred by the small minority of patients (5 percent) who have chronic pain.[3,4] Improved methods of returning these persons to full functioning are needed. The 31 percent of patients who have minor residual symptoms do not, in our view, require intensive rehabilitation. These patients are back at work, can perform all the usual activities of daily living, and have positive responses on just 1 or 2 of the 23 items on the Sickness Impact Profile, as adapted by Roland and Morris.[5,6] These patients may be at increased risk of recurrence, and we are still following our cohort to evaluate this risk. They may also be candidates for improved strategies of self-care. Our data do indicate that intensive evaluation and treatment of all patients with acute back pain is unlikely to be a cost-effective method of improving outcomes in such patients.

The comments of Dr. Hampton and Dr. Barrett highlight the differences between allopathic and chiropractic physicians in their communication with patients. Over 75 percent of patients who see primary care physicians for back pain stay with their index provider. We found some use of vitamins among patients who see chiropractors, but we also found that these patients use substantial amounts of nonsteroidal antiinflammatory drugs. Although allopathic physicians may criticize the explanatory model used in chiropractic, their patients' level of satisfaction may be lower than that of chiropractic patients because these physicians have no such model of their own. Explanation and reassurance are core components of the physician's role.

TIM CAREY, M.D., M.P.H.
University of North Carolina
Chapel Hill, NC 27599-7590

1. Carey TS, Garrett J, Jackman A, et al. The outcomes and costs of care for acute low back pain among patients seen by primary care practitioners, chiropractors, and orthopedic surgeons. N Engl J Med 1995;333:913-7.
2. Shekelle PG, Markovich M, Louie R. Comparing the costs between provider types of episodes of back pain care. Spine 1995;20:221-7.
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HEREDITARY HEMORRHAGIC TELANGIECTASIA

To the Editor: With respect to the review of hereditary hemorrhagic telangiectasia by Guttmacher et al. (Oct. 5 issue),[1] we would like to highlight some additional points in the recognition and management of this condition. A hallmark of hereditary hemorrhagic telangiectasia is profound variation in severity between members of the same family. In one family with a mutation in the endoglin gene,[2] manifestations ranged from mild disease that was barely apparent clinically to severe visceral involvement. We have also seen families in which

the disease is minor in multiple generations before being manifested as pulmonary arteriovenous malformations in one member in childhood. The phenotypic variation is not well understood; the underlying mutation cannot be solely responsible. One factor appears to be phenotypic modification by the female hormones, which may help in the treatment of hemorrhage,[3] but may adversely affect disease progression, particularly in the lungs. There is a female predominance of pulmonary arteriovenous malformations, and deterioration in the vascular bed may occur with pregnancy.[4] Furthermore, pulmonary arteriovenous malformations are not restricted to families with mutations in the endoglin gene[2] (and unpublished observations).

These findings have implications for clinical screening of families with hereditary hemorrhagic telangiectasia, because no affected member can be assumed a priori not to have a pulmonary arteriovenous malformation. Clinical suspicion of pulmonary arteriovenous malformations stems from a history not just of stroke or cerebral abscess, but more often of multiple transient ischemic attacks, which may have been forgotten or dismissed by the patient. All those at risk in a family should be screened for pulmonary arteriovenous malformations once in childhood, once after puberty, before pregnancy, and then at 10-year intervals. Screening can be done inexpensively and safely with a method such as pulse oximetry (performed for 10 minutes each with the subject in the standing and supine positions) in conjunction with posteroanterior and lateral chest x-ray films.[5] Suspicious results should lead to more extensive investigations with albumin microspheres labeled with technetium-99m, lung and kidney scanning, or contrast echocardiography.[5] A policy of five-year follow-up of all persons with pulmonary arteriovenous malformations by means of helical computed tomography (CT), as suggested by Guttmacher et al.,[1] would be very expensive and would expose young people to too much radiation. There are simpler, safer, and cheaper alternatives.

CLAIRE L. SHOVLIN, M.A., M.R.C.P.
Boston, MA 02115 Harvard Medical School

London W12 0NN, J.M.B. HUGHES, D.M., F.R.C.P.
United Kingdom Hammersmith Hospital

1. Guttmacher AE, Marchuk DA, White RI Jr. Hereditary hemorrhagic telangiectasia. N Engl J Med 1995;333:918-24.
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To the Editor: We are currently screening all members of families with hereditary hemorrhagic telangiectasia in the Danish county of Funen (500,000 inhabitants) for the presence of pulmonary arteriovenous malformations. The prevalence of hereditary hemorrhagic telangiectasia in Funen is 1.38 per 10,000.[1] All participants are clinically examined for the presence of telangiectatic lesions, and pulse oximetry with subjects in the supine and standing positions is performed. If hereditary hemorrhagic telangiectasia is diagnosed or if the oxygen saturation in either position is below 96 percent, contrast echocardiography is performed. Our preliminary data suggest that pulmonary arteriovenous malformation can be present in patients with normal oxygen saturation. Among 11 patients with untreated pulmonary arteriovenous malformations verified by contrast echocardiography, 6 had oxygen-saturation values $\geq$96 percent in the supine as well as the standing position.

Screening for the presence of pulmonary arteriovenous malformation is recommended in late childhood and again at the end of adolescence. The pulmonary arteriovenous malformations are usually diagnosed in young age groups and have not been found as the first symptom of hereditary hemorrhagic telangiectasia in older patients. In our study we have found one 64-year-old man who was classified as not having hereditary hemorrhagic telangiectasia on clinical examination. Because there was a history of a transient ischemic attack and low values were found on pulse oximetry (oxygen saturation, 94 percent in the supine position and 95 percent in the standing position), contrast echocardiography was performed and showed a pulmonary arteriovenous malformation. The patient's father had hereditary hemorrhagic telangiectasia in a very mild form, without signs of pulmonary arteriovenous malformation on contrast echocardiography, whereas two uncles and two aunts had pulmonary arteriovenous malformations with severe symptoms.[2] This particular case is of interest because the patient had no history of either epistaxis or gastrointestinal bleeding. On clinical examination he only had a few telangiectatic lesions on the trunk and none at the typical sites, such as the nose, lips, mouth, conjunctivae, or fingers.

Our findings suggest that contrast echocardiography is a safe and sensitive method for the diagnosis of pulmonary arteriovenous malformations. The method should be used when there are broad indications, especially in persons with a family history of hereditary hemorrhagic telangiectasia with a high prevalence of pulmonary arteriovenous malformations.

ANETTE D. KJELDSEN, M.D.
POUL VASE, M.D., PH.D.
5700 Svendborg, Denmark Svendborg Hospital

HENRIK OXHØJ, M.D., PH.D.
5000 Odense C, Denmark Odense University Hospital

1. Vase P. Telangiectasia haemorrhagica hereditaria Mb. Osler: en epidemiologisk, genetisk og klinisk undersøgelse. (Thesis. Odense, Denmark: Odense University, 1988.)
2. Vase P, Holm M, Arendrup H. Pulmonary arteriovenous fistulas in hereditary hemorrhagic telangiectasia. Acta Med Scand 1985;218:105-9.

The authors reply:

To the Editor: Drs. Shovlin and Hughes highlight the profound variations within families in manifestations of hereditary hemorrhagic telangiectasia, as well as the serious complications sometimes seen with pregnancy. Determining whether the causes of such variations and complications are hormonal, genetic, or environmental (or some combination of the three) will be critical to a full understanding of this condition.

Although pulmonary arteriovenous malformations can occur in anyone with hereditary hemorrhagic telangiectasia, they are significantly more common in those in whom the disease is due to a mutation in the endoglin gene than in those whose disease is due to mutations in other genes.[1] Thus, we believe that variations among families are also crucial and that clinicians need to be more aggressive in screening for these malformations in families in which hereditary hemorrhagic telangiectasia is linked to the endoglin gene.

The question of how best to screen for pulmonary arteriovenous malformations is complex. Kjeldsen et al. point out that pulse oximetry can be a helpful tool when the results are positive, but it misses a substantial number of these lesions. This has been our experience as well. More sensitive screening methods include contrast echocardiography,[2] shunt studies involving albumin microspheres labeled with technetium-99m,[3] and high-resolution helical CT without contrast medium.[4] Of these, both shunt studies and CT have the disadvantage of involving exposure to radiation (at similar levels in each). However, CT is the most widely available method and the best at determining which lesions are clinically important.[4]

Follow-up with helical CT is expensive and poses a risk from radiation exposure. However, many patients with a history of a pulmonary arteriovenous malformation due to hereditary hemorrhagic telangiectasia have small, untreated malformations that are growing.[5] Because these lesions may grow sufficiently to lead to such complications as brain abscess or stroke, in our view they warrant follow-up with techniques that are more sensitive than positional oximetry and that can detect the lesions before they become large enough to cause neurologic sequelae.

The current debate about screening for pulmonary arteriovenous malformations underscores the point that it is impossible to define optimal practice for a number of aspects of hereditary hemorrhagic telangiectasia without rigorous prospective trials. Although such trials are often important in common disorders, they are particularly necessary for conditions that both are rare and exhibit genetic heterogeneity. Until rigorous prospective studies are conducted of hereditary hemorrhagic telangiectasia, reasonable and experienced people will continue to disagree about a number of areas of diagnosis and management for this family of conditions.

ALAN E. GUTTMACHER, M.D.
Burlington, VT 05401 University of Vermont

DOUGLAS A. MARCHUK, PH.D.
Durham, NC 27710 Duke University

ROBERT I. WHITE, JR., M.D.
New Haven, CT 06520-8042 Yale University

1. Berg JN, Guttmacher AE, Marchuk DA, Porteous MBM. Clinical heterogeneity in hereditary haemorrhagic telangiectasia: are pulmonary arteriovenous malformations more common in families linked to endoglin? J Med Genet (in press).
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GENE THERAPY

To the Editor: The report by Knowles et al. (Sept. 28 issue)[1] and the accompanying editorial[2] on gene therapy for cystic fibrosis gave a very fair account of some of the difficulties involved. We are concerned, however, that the two articles taken together may send an unbalanced message. A study of liposome-based gene therapy reported recently,[3] with a trial design very similar to that of Knowles et al., gave encouraging results — a correction of the chloride-permeability defect of approximately 20 percent without any adverse effects. Although this is unlikely to be adequate for therapy, the understanding of liposome-mediated gene transfer is advancing rapidly. We would therefore question whether the future lies only in either immunosuppression to limit viral side effects or the development of improved viral vectors as suggested, rather than also in the development of more efficient nonviral vectors.

We now know that both adenoviral-mediated and liposome-mediated transfer of the cystic fibrosis transmembrane conductance regulator (*CFTR*) gene into human airway epithelium in vivo is an inefficient process. Although these reports are valuable and correct some of the overenthusiasm surrounding gene therapy, it would be a great pity if they pushed the pendulum too far the other way by ignoring the possibilities for nonviral delivery systems for gene therapy.

ERIC ALTON, M.D.
DUNCAN M. GEDDES, M.D.
London SW3 6LR, Imperial College of Science,
United Kingdom Technology and Medicine

1. Knowles MR, Hohneker KW, Zhou Z, et al. A controlled study of adenoviral-vector–mediated gene transfer in the nasal epithelium of patients with cystic fibrosis. N Engl J Med 1995;333:823-31.
2. Leiden JM. Gene therapy — promise, pitfalls, and prognosis. N Engl J Med 1995;333:871-3.
3. Caplen NJ, Alton EWFW, Middleton PG, et al. Liposome-mediated *CFTR* gene transfer to the nasal epithelium of patients with cystic fibrosis. Nat Med 1995;1:39-46.

To the Editor: Knowles et al. provided an excellent and long-overdue randomized, double-blind appraisal of the prospects for gene therapy that uses a first-generation adenoviral vector capable of transducing the *CFTR* gene. Their observation that local inflammatory responses probably blocked the correction of chloride transport by the adenoviral vector surprised few researchers in the field. In fact, inflammation secondary to first-generation adenoviral vectors is thought to be a major limitation of these vectors.[1,2]

However, the media reaction to this *Journal* article was surprising. Many newspapers and news magazines published stories claiming, for example, that "scientists may have pushed too far too fast in a race for [gene therapy] breakthroughs,"[3] and that "five years of [gene therapy] attempts have cured no one."[4] The hyperbole can be traced back to the media, not to participating scientists.

The vast majority of approved clinical trials of gene therapy are focused on the safety, not the efficacy, of this new technology. The continued focus on safety conflicts with the rush to develop this technology and partially explains the fact that gene therapy has yet to cure patients. Furthermore, the complex and highly regulated approval process for gene-therapy protocols, involving multiple reviews by local institutions as well as reviews by the Food and Drug Administration and the Recombinant DNA Advisory Committee of the National Institutes of Health, prohibits an excessive or premature rush to the marketplace for gene therapy. Instead, current gene-therapy research, such as the observations of Knowles et al., sets the stage for future research — a process integral to the scientific method. Many developments are already in progress for gene therapy of cystic fibrosis with the use of new second-generation adenoviral vectors, adeno-associated viral vectors, and liposomal gene transfer.[2] The promises of gene therapy will probably be fulfilled, but patients

and the public must understand that the process is hard work and may take a long time.

JOHN A. WAGNER, M.D., PH.D.
Stanford University
Stanford, CA 94305-5332 Medical Center

1. Crystal RG, McElvaney NG, Rosenfeld MA, et al. Administration of an adenovirus containing the human CFTR cDNA to the respiratory tract of individuals with cystic fibrosis. Nat Genet 1994;8:42-51.
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The authors reply:

To the Editor: We remain enthusiastic about gene therapy, despite the slower-than-hoped-for rate of progress and the inefficiency of current adenoviral-mediated and liposomal-mediated *CFTR* gene transfer.[1,2] One of the intents of our paper was to alert researchers in the field to problems with the efficiency of the adenovirus and consequently to stimulate new approaches to increase the efficiency of this vector by the use of other serotypes or new dosing strategies. Liposomes are also less efficient than we would like, as evidenced by the fact that the "encouraging results" of the study by Caplen et al.[2] that Drs. Alton and Geddes mention were modest and transient (lasting a few days) and required repetitive dosing (every 10 to 15 minutes) for up to 7 hours. We agree with Drs. Alton and Geddes that we should continue a broad-based approach to the development of vectors, including liposomes, as emphasized in the last paragraph of our report. As Dr. Wagner indicates, progress will result from iterative basic-science and clinical-research studies. Our study revealed low-level gene transfer and illustrates how quantitative this field has become. Such is the expected course of events in a new area of applied biology, as addressed in the editorial by Dr. Leiden,[3] and we remain optimistic that continued efforts will yield benefits for the application of gene therapy to the treatment of human disease.

MICHAEL KNOWLES, M.D.
RICHARD BOUCHER, M.D.
Chapel Hill, NC 27599-7020 University of North Carolina

1. Crystal RG, McElvaney NG, Rosenfeld MA, et al. Administration of an adenovirus containing the human CFTR cDNA to the respiratory tract of individuals with cystic fibrosis. Nat Genet 1994;8:42-51.
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3. Leiden JM. Gene therapy — promise, pitfalls, and prognosis. N Engl J Med 1995;333:871-3.

HYDROXYUREA IN SICKLE CELL DISEASE

To the Editor: We wish to comment further on the possible risks of hydroxyurea therapy in sickle cell disease, a subject of considerable attention since the results of the Multicenter Study of Hydroxyurea in Sickle Cell Anemia were reported by Charache et al. (May 18 issue).[1]

We agree that the caution advised by Ho and Murgo[2] and Silver[3] in their letters (Oct. 12 issue) is necessary. Unfortunately, the single sentence from our editorial[4] quoted by Ho and Murgo distorts the thrust of our paragraph on the long-term safety of hydroxyurea. Continued monitoring of patients with sickle cell disease and others receiving hydroxyurea for secondary cancers and other unanticipated side effects of this drug is essential.

Equally important are the implications of data from the multicenter study on hemoglobin F levels in patients treated with hydroxyurea, presented on December 4, 1995, at the meeting of the American Society of Hematology in Seattle. Steinberg et al.[5] noted that between 25 percent and 50 percent of the treated patients had no change in hemoglobin F levels during two years of therapy, confirming earlier observations in a study in which compliance was ensured.[6] Data from the multicenter study showing a strong correlation between the clinical response and the hemoglobin F level were also briefly reported.

These results, in our opinion, suggest that hemoglobin F levels should be monitored closely in patients being treated with hydroxyurea. If a marked increase in the level of hemoglobin F (an absolute increase of at least several percentage points) fails to occur after three to six months, consideration should be given to discontinuing the drug. It is possible that mechanisms other than the inhibition of hemoglobin-S polymerization by hemoglobin F could make treatment with hydroxyurea beneficial. Until there is better evidence for these postulated effects, however, they should not influence routine decisions about therapy.

In addition, the fact that a considerable fraction of patients do not have a response to hydroxyurea reinforces our plea for "continued study of other dosage regimens, other drugs, and combinations of agents"[4] to effect greater responses in hemoglobin F levels and possibly greater clinical benefits. A recent report to the director of the National Institutes of Health is quite cautious about the clinical prospects for gene therapy in the foreseeable future.[7] It is also essential that the results of current therapies, based on modulating endogenous gene expression, not be overinterpreted, which might result in a de-emphasis on the necessary basic and clinical research.

ALAN N. SCHECHTER, M.D.
GRIFFIN P. RODGERS, M.D.
Bethesda, MD 20892 National Institutes of Health

1. Charache S, Terrin ML, Moore RD, et al. Effect of hydroxyurea on the frequency of painful crises in sickle cell anemia. N Engl J Med 1995;332:1317-22.
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6. Rodgers GP, Dover GJ, Noguchi CT, Schechter AN, Nienhuis AW. Hematologic responses of patients with sickle cell disease to treatment with hydroxyurea. N Engl J Med 1990;322:1037-45.
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To the Editor: Charache et al. report a 44 percent reduction in the median annual rate of painful crises in adults with sickle cell disease treated with oral hydroxyurea. However, the long-term risks of such treatment remain unclear. Although melanonychia (hyperpigmentation of the nails) has been reported in patients receiving hydroxyurea for chronic myelogenous leukemia,[1,2] this finding has not been reported in patients with sickle cell disease. Hydroxyurea-induced horizontal

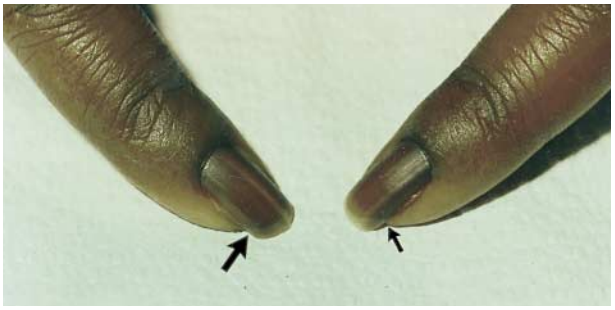

Figure 1. Fingernails of a 31-Year-Old Black Woman with Hemoglobinopathy SS and Hydroxyurea-Induced Melanonychia Characterized by Longitudinal (Large Arrow) and Diffuse Horizontal (Small Arrow) Bands.

hyperpigmentation of the nails has been described,[2-4] but there are only a few cases with the hydroxyurea-induced longitudinal form of hyperpigmentation.[1,3]

We treated six adults with sickle cell disease (four women and two men; age range, 18 to 45 years) with hydroxyurea (1 to 1.5 g daily). Five of the six patients had hemoglobin SS disease; one patient had hemoglobin S–β^0-thalassemia. Before therapy, each patient had reported at least three severe painful events per year requiring parenteral analgesics.

After six months of treatment with hydroxyurea, five of the six patients reported a decrease in the number of painful events. Melanonychia developed in two of the four women after six weeks of therapy. The 20 nailbeds of each affected woman showed heterogeneous patterns of nonpainful longitudinal bands to the tips of the nails, as well as diffuse horizontal bands of hyperpigmentation (Fig. 1). The longitudinal bands ranged from 0.5 to 4 mm in width, whereas the horizontal bands appeared diffuse and were difficult to measure. The nails did not appear to be thickened or atrophic, and the adjacent skin was normal.

One of the two affected women had a small area of hyperpigmentation on the left lateral aspect of the tongue, as well as on her palms and soles, suggesting a systemic effect. The face, gums, and buccal mucosa have not been involved to date. In the other woman, only the nails were involved. The two affected patients considered discontinuing hydroxyurea because of this undesirable cosmetic effect. However, both patients decided to continue taking the drug because of the reduction in painful events.

We do not know of predisposing factors for melanonychia and whether this cosmetic feature will have a negative effect on compliance with treatment. It may be reversible, as has been demonstrated in one patient with chronic myelogenous leukemia.[4]

PAT ADAMS-GRAVES, M.D.
CATHY HELTSLEY, M.D.
STEVEN DEITCHER, M.D.
Memphis, TN 38163 University of Tennessee

1. Baran R, Kechijian P. Longitudinal melanonychia (melanonychia striata): diagnosis and management. J Am Acad Dermatol 1989;21:1165-75.
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TUBERCULOSIS IN A NEIGHBORHOOD BAR

To the Editor: In the report of Kline et al. describing an outbreak of tuberculosis among bar patrons (July 27 issue),[1] 4 of the 14 active tuberculosis cases occurred among patients seronegative for the human immunodeficiency virus who had positive tuberculin tests but normal chest radiographs. This is not a new phenomenon[2]; among contacts of patients with active tuberculosis on Navy ships, 3 to 25 percent of those whose tuberculin skin tests converted from negative to positive had positive sputum cultures despite having normal chest films and sputum smears.[3] Kent et al. obtained daily cultures for one month from 12 patients with recently converted tuberculin skin tests and isolated *Mycobacterium tuberculosis* from 10 of them (83 percent).[4] Given the more sensitive techniques now available for detecting *M. tuberculosis*, the actual proportion may be even higher.[5] The optimal treatment for culture-positive patients with normal chest radiographs and their degree of infectiousness are not known.

All four patients in the study by Kline et al. who had positive cultures with normal chest radiographs had cough, and two had weight loss. This suggests that tuberculin conversion may not always be asymptomatic, that these symptoms may be common among people with a history of alcohol and cigarette use, or that active tuberculosis disease was in the process of developing in these patients. Perhaps tuberculosis infection, like primary HIV infection, may be characterized by a distinct symptom complex.[6]

The authors conducted a prompt and thorough investigation in a difficult context, effectively limiting the further spread of tuberculosis. The thoroughness of their evaluations may have identified several patients with positive cultures in whom active tuberculosis would not have developed.

THOMAS R. FRIEDEN, M.D., M.P.H.
New York City
New York, NY 10013 Department of Health

TIMOTHY R. STERLING, M.D.
Keesler AFB, MS 39534 Keesler Medical Center

PATRICIA M. SIMONE, M.D.
Centers for Disease
Atlanta, GA 30333 Control and Prevention

1. Kline SE, Hedemark LL, Davies SF. Outbreak of tuberculosis among regular patrons of a neighborhood bar. N Engl J Med 1995;333:222-7.
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HEALING BY DESIGN

To the Editor: In Horsburgh's "Healing by Design" (Sept. 14 issue),[1] he does not mention the pernicious way in which medical economic pressures are influencing modern hospital design. Hospitals in competition with one another for paying

patients spend extravagantly for lobbies and entrances more appropriate for a bank or Las Vegas hotel, while cutting back on professional and ancillary staffing to try to improve their bottom lines. Compounding the problem is the desire of philanthropists to erect impressive testimonials to their generosity.

Simple but relatively skilled and labor-intensive care remains the key to managing many common and compelling medical problems.[2] Building fantasy-vacation hospitals in an age when American hospitals are ruthlessly cutting professional and ancillary staff strikes me as inappropriate husbandry of our health care resources.

ARTHUR D. FORMAN, M.D.
University of Texas
Houston, TX 77030 M.D. Anderson Cancer Center

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2. Thiyagarajan C, Silver JR. Aetiology of pressure sores in patients with spinal cord injury. BMJ 1984;289:1487-90.

To the Editor: As Dr. Horsburgh points out, the recent interest in hospital design has focused appropriately on patients and their families as the "consumers" of hospital services. To achieve the goal of optimizing health care delivery, however, hospital design must also optimize coordination among health care providers. For example, horizontal spaces in hospitals encourage face-to-face interaction between health care providers, sometimes facilitating consultation or important discussions regarding the treatment of patients.[1] Health care providers at the old Peter Bent Brigham Hospital in Boston (where "pavilion style" ward buildings were connected by a single conduit affectionately called "The Pike") or at the Cleveland Clinic Foundation (where the "Skyway," a horizontal, elevated walkway, connects the hospital to the outpatient building) appreciate the advantages of horizontal spaces that encourage face-to-face meetings and interactions. Also, having alcoves along hospital corridors permits private conversations, thereby averting the hazards of "elevator talk."[2]

JAMES K. STOLLER, M.D.
Cleveland, OH 44195 Cleveland Clinic Foundation

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Dr. Horsburgh replies:

To the Editor: I share Dr. Forman's dismay that some hospitals have spent large sums for sumptuous spaces that have little effect on patient care. Good design can provide positive symbolic meaning without opulence. Such misguided expenditures emphasize the importance of involving health care providers in the design process.

Dr. Stoller identifies additional positive attributes of horizontal hospitals that have contributed to the increase in construction of such facilities in the past decade.

C. ROBERT HORSBURGH, JR., M.D.
Emory University
Atlanta, GA 30303 School of Medicine