Microbiologically Confirmed Early Lyme Disease

TO THE EDITOR: Smith and colleagues (1) provided much-needed information about the characteristics of erythema migrans caused by *Borrelia burgdorferi* that will grow in Barbour–Stoenner–Kelly culture. In addition, they commented on erythema migrans–like lesions in the southeastern United States, which are associated with *Amblyomma americanum*, or lone star ticks (2), and suggested that “another agent, perhaps even from the *Borrelia* genus, may cause the infection.” For this statement, they gave three references, one of which is a study by Campbell and associates that was sponsored by the U.S. Centers for Disease Control and Prevention (3).

In fact, Campbell and associates concluded quite the opposite, that is, that these tick bite–associated annular rashes were not caused by *B. burgdorferi* or related spirochetes. The state epidemiologist who initiated the study and I, as the clinician who supplied most of the patients, declined authorship because of the exclusion of relevant data and the lack of objectivity. We published our objections in the *Journal of Clinical Infectious Diseases* (4) and *Missouri Medicine* (5), including data excluded by Campbell and associates, and reiterated our opposite conclusion: that atypical *B. burgdorferi* or related spirochetes were completely compatible with data gathered in the study.

Now that increasing evidence is implicating these “Lyme-like” lone star tick–associated rashes as borreliosis, it would be a terrible mistake to give credit to Campbell and associates. In fact, on 31 March 1996, after the Campbell study’s publication, Dr. David Dennis of the Centers for Disease Control and Prevention, a co-author, was quoted in the *Kansas City Star* as saying that these rashes “could be caused by tick spit for all we know.” For factual reasons, one should avoid giving credit for properly implicating borreliosis to those who actively and, in my opinion, inappropriately opposed the concept.

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References

Resident Burnout

TO THE EDITOR: Although the articles on housestaff burnout by Shanafelt and colleagues (1, 2) and the accompanying editorial by Clever (3) present a disturbing snapshot of the mental health of trainees, they do not take important related issues into account. First, none notes that the difficult working conditions of residents are time limited. One would certainly approach the question of altering working conditions differently if residents were permanently consigned to the demands of a training environment, rather than experiencing them, as they now do, for a few years.
Second, none of the pieces addresses the potential consequences of substantial changes in the demands of residency training. Change (generally in the form of limitations on work hours) is advocated as an unalloyed good, intended to improve the life of housestaff. Even if one accepts that work changes would, in fact, boost housestaff morale, that is certainly only part of the picture.

Training programs need to concern themselves not only with the mental and physical health of their residents but also with the quality of care they ultimately provide as practicing physicians. Will physicians trained in a more nurturing environment really be better doctors? Will they provide the same level of dedication that patients have a right to expect? Will they display the very essence of professionalism, which is to put their patients’ interests ahead of their own? If they are not expected to do this as trainees, how then will they learn to do it as practitioners? It is telling that Clever says “we know from experience and instinct” that more nurturing training leads to better doctoring down the line without citing any evidence to support the claim, thereby displaying the very antithesis of the evidence-based thinking and practice that we hope to instill in our trainees.

No one should support the perpetuation of difficult working conditions for residents as a professional right of passage. By the same token, no one should tamper with such conditions without considering the impact on the future practice of medicine.

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References

TO THE EDITOR: The report by Shanafelt and colleagues (1) and the accompanying editorial by Clever (2) demonstrate that residents’ burnout occurs with great frequency and may affect patient care. As Shanafelt and colleagues point out in their Table 4, these problems continue at levels unchanged over the past 15 years despite improvements in residency training, such as night float and smaller case loads. Trainees enter residency without practical knowledge yet are trained correctly, if he is confused about what he is doing, then he tends to develop angst, which is a setup for burnout. In other words, it is likely that the lack of appropriate training in medical school and residency lead to burnout.

Academic medicine has, over the years, been primarily interested in itself and its own interests, such as grants and publications. Clinical education has been allowed to atrophy. For example, turn to page 245 in a Cecil Textbook of Medicine, 18th edition (2). The author writes that isotonic saline contains 140 mEq of sodium and 140 mEq of chloride. The fact is that isotonic saline in use in clinical medicine is normal saline, which contains 154 mEq of sodium and 154 mEq of chloride. How could an author contributing to a major textbook of medicine not know such a basic fact? How could the editors miss this or not know it? One can make rationalizations to explain this, but the truth may be relatively straightforward. There are far too many in academic medicine who lack competence.

I could point to other examples of errors, but suffice it to say that the level of suboptimal performance reported by Shanafelt and colleagues probably reflects the overall level of competence in academic medicine. The actions of the residents are mirroring those of their professors. The article by Shanafelt and colleagues was coura-

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References
TO THE EDITOR: As Shanafelt and colleagues revealed (1), resident burnout is prevalent. Residents face long hours, sleepless nights, and heavy workloads, and they often find themselves frustrated with patient care responsibilities. The effects of this burnout, though, are not confined to a resident’s personal life or attitude. As a third-year medical student rotating through clinical clerkships, I have found that the burnout inherent in residency extends beyond the residents’ lives and into the sphere of medical education.

The chief resident of my gynecology rotation described the process succinctly. “As an intern, you spend your entire year lost,” she explained. “During your second and third years, you become ‘toxic’: cynical, hardened, skeptical. Finally, during your fourth year, you start to see the light.” Unfortunately, as residents travel through this process from “lost” to “toxic” to finally “seeing the light,” medical students are with them for the journey. And, as a result of the close association between residents and medical education, rotating students acquire a certain “toxicity”: disillusion with patient care, loss of compassion, and frustration with the demands of the current residency system.

The article by Shanafelt and colleagues addresses important concerns for the livelihood of medical residents and the resulting impact on patient care. The direct effect of resident burnout on medical students’ attitudes must be evaluated, as well. Medical students learn from resident attitudes as well as from their clinical knowledge. When the medical system creates in residents attitudes aversive to effective medical care, the impact of that system extends far beyond the confines of day-to-day hospital medical practice.

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Reference

The Effect of HFE Genotypes on Measurements of Iron Overload

TO THE EDITOR: Since the publication of our article on the effect of genotypes on measurements of iron overload (1), we have continued our study. We present data that were gathered up to 5 September 2001 on 153 C282Y/C282Y homozygotes (79 women and 74 men) and on 31 356 white or white–Hispanic persons who were not homozygotes. In the latter group, transferrin saturation values and serum ferritin values, respectively, were available for 15 471 women and 15 204 men and for 15 009 women and 14 821 men. Sensitivities and specificities relating to these cohorts are presented in the accompanying Table.

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References
Table. Updated Sensitivity and Specificity of Transferrin Saturation and Serum Ferritin Levels for Detection of C282Y/C282Y Homozygosity*

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* Values in parentheses are 95% CIs.

Chiropractic

TO THE EDITOR: The article by Meeker and Haldeman (1) on chiropractic is highly informative on several issues but equally misleading on other points, particularly research. For instance, the authors state that there is no evidence of publication bias in the chiropractic literature. We have recently shown that, in the United Kingdom, nonpublication of severe adverse effects of chiropractic seems to be close to 100% (2). The authors also claim that 43 randomized, controlled trials of spinal manipulation for back pain have been published, but they fail to mention that most of them do not relate to chiropractic spinal manipulation.

Meeker and Haldeman state that the most recent systematic review of chiropractic for back pain, which arrived at a (partly) positive conclusion, was published in 1999 but fail to mention two systematic reviews published at the same time. One concluded that “the effectiveness of manipulation in patients with chronic pain is poorly documented” (3), while the other found “conflicting evidence on the effects of spinal manipulation in acute and chronic low back pain” (4). Similarly, the authors cite one positive study of spinal manipulation for infantile colic but do not mention a larger trial, which concluded that “chiropractic spinal manipulation is no more effective than placebo” (5). Furthermore, Meeker and Haldeman inform us that “nonserious side effects of manipulation may consist of localized discomfort, headache, or fatigue that resolves within 24 to 48 hours.” However, the very study they cite in support of this statement also shows that such adverse events occur in about 50% of all patients.

This is just some of the evidence in this article that suggests biased interpretation. Readers deserve a more objective evaluation of chiropractic, which remains a highly controversial subject.

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Reference

References

TO THE EDITOR: After reading the article by Meeker and Haldeman (1), I have the following questions for them. First, where is the “basic science” of chiropractic? In a day and age of detailed neuroimaging by high-resolution computed tomography and magnetic resonance imaging, of neurophysiology studies and evoked potentials and electromyography, why has no one been able to demonstrate subluxations and their effects, on which chiropractic is based? Second, why doesn’t chiropractic limit itself to spinal manipulation and back pain, for which there appears to be some evidence of benefit, instead of extending its reach to megavitamins, colonic irrigation, and other methods for diseases such as asthma, sinus trouble, emphysema, menopause, and psoriasis? Third, if indeed there are legitimate and beneficial treatments and their effects, on which chiropractic is based? Fourth, where is the pretension to medical skill. In my opinion, which is unchanged by Meeker and Haldeman’s paper, that describes chiropractic.

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Reference

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Reference

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Letters

TO THE EDITOR: In their paper on chiropractic (1), Meeker and Haldeman overlook one basic flaw: The chiropractic theory of vertebral subluxation and spinal manipulative adjustments is false. It is just as false today as when it was formulated more than a century ago by the founder and magic healer D.D. Palmer. A vertebral subluxation has never been demonstrated to exist and has never been shown to cause disease by theoretically interfering with neural integrity. Chiropractic has not contributed one single piece of scientific medical knowledge to prove its validity in the past 100 years. Admittedly, it has gained considerable public and political credence, but this is based only on belief and not scientific evidence. Nothing has basically changed scientifically in chiropractic since the very beginning.

I have often challenged chiropractors to name one single disease that chiropractic spinal adjustment can effectively treat, cure, improve, or prevent, along with scientific evidence to prove it, and no one has thus far been unable to do so. Claims of effective treatment of otitis media, childhood asthma, tension headaches, and even low back pain with chiropractic have all been negated by recent studies. For chiropractic to be considered scientific medical care rather than alternative unscientific care, it must first abandon the false theory of subluxations as a cause of disease and spinal adjustments as a treatment. Chiropractic colleges must stop teaching this theory and must convert to accredited medical colleges, as osteopathic schools have done, or must limit the scope of chiropractic to scientifically oriented physical therapy.

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References

IN RESPONSE: These letters illustrate the strong, emotional, often vitriolic responses any discussion of chiropractic evokes in certain physicians. The charge of bias cuts in many directions, as Dr. Ernst is well aware. We stand by our specific statement that overall, there is no evidence of biased reporting of randomized trials of spinal manipulation. We agree that the basic science of manipulation and of the concept of subluxation is controversial and has not yet been fully developed. However, scientists in chiropractic institutions and major universities around the world, supported by major public and private funding agencies, are making progress. Greater understanding of the theories on which manipulation is based will become available. We agree that many of the randomized trials we described were on spinal manipulation rather than specifically on chiropractic manipulation itself, but we believe that this is not a significant point. Chiropractors use all forms of manipulation. In the United States, more than 90% of all spinal manipulation services are provided by chiropractors, and research on spinal manipulation, like that on any other treatment method, is equally of value regardless of the practitioner providing it.

We elected to include only the most recent and comprehensive English-language reviews of manipulation trials out of at least 60 published in the past 25 years. The paper by Brox and colleagues to which Dr. Ernst refers is in Norwegian and does not include all trials of manipulation for chronic back pain. van Tulder, in addition, has published inconsistent conclusions (2–4). Nevertheless, 10 of 11 nationally developed practice guidelines for back pain have recommended manipulation as a treatment option (4). The trial by Olafsdottir and colleagues on infantile colic, also mentioned by Dr. Ernst, was published too late to be included in our list and does not change our already cautious conclusions.

Minor reactions to spinal manipulation are known to all practitioners and most patients of chiropractors and tend to be no more than a nuisance in practice. Patients do not seem to be concerned about these side effects. Patient satisfaction remains higher for chiropractic care than for any treatment with which chiropractic has been compared (5), and the dropout rate due to side effects in clinical trials on manipulation and chiropractic is negligible.

Chiropractic as a profession has made many advances in education, ethics, practice, and theory that give it the trappings of a mainstream health care profession, but there are still a number of claims, as pointed out by these letters, that have not been adequately evaluated. It was for this reason that we described chiropractic as a profession at the crossroads between mainstream and alternative medicine.

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References

East Asian Medicine

TO THE EDITOR: In the history of its interaction with the Chinese paradigm of the body, biomedicine has never started from the premise that the Chinese might have gotten something right. The anatomic paradigm has proved so strong that other paradigms have generally been dismissed as superstition or quackery. As early as the 17th century, western observers rejected Chinese readings of the body because the Chinese “did not know anatomy.”

In his article on acupuncture, Kapchuk (1) notes that the concept of qi provides a rationale for explaining change. Indeed, both Chinese philosophical systems and medicine have long focused on understanding and articulating the dynamics of change. Key terms like qi express the nature of process—not structures of things, but dynamic functions, in relationship.
We have often been so swayed by the power of our governing intellectual paradigms that we have found it difficult to take seriously Chinese models that register different orders of experience. Efforts at translation over time have all sought to establish equivalencies between Chinese and western concepts, whether through humor, medical electricity, or, as Kaptchuk’s useful article notes, basic scientific evidence. Presumably, other systems are valid only when translatable into biomedical representations of the body.

As those who speak second languages know, each language captures sometimes elusive dimensions of reality. Accurate translation can require retaining original terms, like qi. A second language teaches us about our own. The challenge to biomedical researchers involves a willingness to become bilingual, taking both paradigms as expressions of something significant about the real.

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IN RESPONSE: Dr. Barnes’ comment on the need for a sensitive bilingual encounter between distinct medical systems is greatly appreciated. Indeed, knowledge of other medical systems necessarily encourages self-awareness and is an antidote to arrogance. I find myself so much in agreement with Dr. Barnes that I wonder whether her metaphor of language and “translation” may not be radical enough. The difference between traditional East Asian concepts of medicine and biomedicine extends beyond linguistic issues to the philosophical foundations of their epistemology (1). Chinese medicine relies on the veracity of the senses, the person-centered experience either as reported by the patient or perceived by an observant practitioner. Ultimately, biomedicine depends on deliberately controlled experiments and understands that what is to be most accurate must be shielded from the bias of human observation. More than saying something different about the “real,” Chinese medicine and biomedicine engage a reality as distinct and foreign as Ptolemy’s universe.

Nonetheless, any vibrant medical system needs to do more than sympathetically understand other systems. It may seek to selectively adopt—metabolize, if you will—techniques or strategies independent of theoretical understanding. For example, Chinese medicine incorporated many Greco-Persian herbs during the Tang dynasty (618 to 907 AD) (2), and some practitioners in China are currently in the process of creatively reformulating acupuncture based on modern physiology (3). Indeed, biomedicine has managed to already borrow some of China’s traditional medicine without its theoretical underpinning (for example, ephedrine from the Chinese herb Ephe- drina sinica [4] or artemisinin from the Chinese herb Artemisia apiacea as a new potential drug for malaria [5]). It may be that a genuine and full medical encounter needs to combine respect and self-reflection with opportunities for creative enrichment.

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References

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Intestinal Pseudo-Obstruction in a Diabetic Man: Role of the Mitochondrial A3243G Mutation

TO THE EDITOR: A 33-year-old Japanese man was referred to our hospital in October 2000 for evaluation of a 4-month history of unexplained abdominal pain and diarrhea, which had already prompted two hospitalizations for intestinal pseudo-obstruction. He had developed diabetes mellitus at 20 years of age and hearing loss at 27 years of age but had no history of seizures or stroke-like episodes. His mother and her brother also had diabetes mellitus. The patient was short in stature (159 cm) and weighed 41 kg.

Physical examination by a neurologist revealed no signs of neuromuscular disease, including diabetic neuropathy, but bilateral sensorineural hearing loss was confirmed. Diabetic retinopathy was not noted. Despite long-term treatment with insulin, the patient’s hemoglobin A1C level was 8.7% (normal range, 4.3% to 5.8%). Lactate and pyruvate concentrations, respectively, were 23.5 mg/dL (normal range, 4.0 to 16.0 mg/dL) and 57 μmol/L (normal range, 34 to 102 μmol/L) in blood and 38.9 mg/dL (normal range, 10.0 to 20.0 mg/dL) and 1.2 mg/dL (normal, 0.6 to 1.1 mg/dL) in cerebrospinal fluid.

Figure. Electron micrograph of a mucosal epithelial cell with normal mitochondria (left) and a smooth-muscle cell with abnormal mitochondria (right).

Each white bar indicates 500 nm. (Ultrathin sections were double-stained with 1% uranyl acetate and lead citrate; original magnification, ×15 000).
fluid. Findings on colonoscopy were normal, and the result of the xylose absorption test was 8.6 g (normal range, 5.0 to 8.0 g). Histologic examination of a biopsy specimen from the rectum showed no mucosal abnormality.

After the patient’s informed consent was obtained, molecular genetic analysis of microdissected tissue revealed the mitochondrial A3243G mutation, which was heteroplasmic (mutant and wild type) in the mucosa and almost homoplasmic (mutant) in the muscularis mucosae. Consistent with these genetic findings, electron microscopy revealed normal mitochondria in the mucosal epithelial cells and an increased number of abnormally swollen mitochondria in the smooth-muscle cells of the muscularis mucosae (Figure). The A3243G mutation was also detected in a low percentage of peripheral leukocytes and was confirmed by cloning and DNA sequencing. The patient’s abdominal symptoms were somewhat controlled by conventional and conservative therapy.

On the basis of these findings, our patient received a diagnosis of maternally inherited diabetes and deafness (1) that manifested as intestinal pseudo-obstruction because of myogenic, rather than neurogenic, dysfunction caused by the A3243G mutation. The mitochondrial A3243G mutation in transfer RNALeu(UUR) is most commonly associated with mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS). Such a moderately deleterious mutation is usually heteroplasmic in cells that commonly contain hundreds of mitochondria and thousands of mitochondrial DNA; the energy-generating capacity of these cells generally remains near normal until approximately 90% of mitochondrial DNA are mutated (2). Intestinal pseudo-obstruction has been noted in mitochondrial disorders, including MELAS and, most commonly, mitochondrial neurogastrointestinal encephalomyopathy caused by multiple deletions of mitochondrial DNA (3–5). However, the absence of the A3243G mutation in the stomach and intestine of some patients with MELAS and pseudo-obstruction suggests that the primary disease may lie outside the gastrointestinal tract (5). Our study indicates that in patients with mitochondrial disorders, it may be necessary to determine the actual mitochondrial abnormality responsible for specific tissue dysfunction.

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References

Immune Thrombocytopenia after Losartan Therapy

TO THE EDITOR: An 82-year-old woman presented with nosebleeds and with easy bruising of 2 weeks’ duration. She was not bleeding from any other site and had no history of similar symptoms. Other medical conditions included congestive heart failure and depression. Two weeks before presentation, she stopped taking quinapril and was switched to losartan because of cough. Other medications included nortriptyline and bumetanide, which she had been taking for more than 2 years. The patient had no history suggestive of infection; no history of chemotherapy, radiation, or malignancy; and no family history of any bleeding diatheses.

On physical examination, the patient was hemodynamically stable, alert, and oriented and in no acute distress. Results of heart and lung examination were unremarkable. There was no splenomegaly or hepatomegaly. On skin examination, the patient was found to have petechiae over her body and palate. Laboratory evaluation revealed a hemoglobin level of 137 g/L, a leukocyte count of 5.8 x 10^9 cells/L (40% neutrophils, 47% lymphocytes, 9% monocytes, 3% eosinophils, 1% bands), and a platelet count of 5.0 x 10^9 cells/L. Flow cytometry of the peripheral blood showed normal CD4–CD8 ratio with no evidence of any lymphoproliferative disorder. A peripheral smear showed decreased platelets without clumping or satellitism.

Because the patient was thought to have immune thrombocytopenia from losartan, this therapy was discontinued. The patient was given one dose of Rh o(D) immune globulin and began receiving oral prednisone, 40 mg/d. Her platelet count improved to 298.0 x 10^9 cells/L within 1 week. Prednisone was tapered over the next 2 months, and the patient’s platelet count has remained normal on follow-up evaluations.

Because thrombocytopenia onset occurred 2 weeks after losartan therapy was started, we believe that our patient had immune thrombocytopenia secondary to this drug. Immune thrombocytopenia secondary to losartan has never, to our knowledge, been reported in the literature. Any future cases would suggest a rare, causal association between losartan therapy and clinically significant thrombocytopenia.

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Favorable Long-Term Outcome of a Patient with Transcobalamin II Deficiency

TO THE EDITOR: Background: Transcobalamin II (TCII) deficiency is a rare autosomal recessive disease. Transcobalamin II is a major plasma transport protein for cobalamin. The TCII–cobalamin complex binds to a cell receptor and is then internalized. Therefore, TCII deficiency leads to cellular cobalamin deficiency and results in early-onset megaloblastic anemia, immunologic deficiency, and neurologic abnormalities. Of interest, serum cobalamin levels are normal in patients with TCII deficiency, indicating that most cobalamin is carried by transcobalamin I. Transcobalamin II deficiency is diagnosed if plasma levels of TCII are undetectable. The first case of this
disorder was reported 30 years ago. Since then, fewer than 50 pediatric cases have been reported, and favorable outcome in adulthood is rare.

Case Report: Our patient was born in 1973. His parents were first cousins; two of his brothers had died of infection in infancy. In the first weeks of life, the patient presented with anorexia, diarrhea, severe pancytopenia, and agammaglobulinemia. Transcobalamin II deficiency was diagnosed by the absence of TCII in serum (1). Intramuscular administration of cobalamin, 1 mg/wk, resulted in clinical and laboratory remission. Initial adherence to treatment was good, and no complications were noted during childhood. Growth and cognitive and social development were normal until adulthood.

At 20 years of age, the patient discontinued follow-up and his cobalamin injections were given at longer intervals. Eight years later, he was hospitalized with a retinal hemorrhage and otherwise normal findings on physical examination. He was pancytopenic (leukocyte count, $2.4 \times 10^9$ cells/L; granulocyte count, $0.5 \times 10^9$ cells/L; platelet count, $11 \times 10^9$ cells/L; hemoglobin level, 56 g/L), and the bone marrow was hypercellular with an increase of early erythroblasts and no excess of blast cells. Erythroblasts and proerythroblasts were megaloblastic. Intramuscular administration of 4 mg of cobalamin led to reticulocytosis, leukocytosis, and thrombocytosis within 7 days. Thereafter, oral treatment (1 mg of cobalamin daily) was tried but was followed by early relapse of pancytopenia. Long-term intramuscular cobalamin was then prescribed. The patient is now well, and his blood counts are normal.

Discussion: Few reports have described outcome of this disease in childhood (2–4). Most children have megaloblastic anemia and digestive symptoms in early infancy, which are corrected with intramuscular cobalamin. During childhood, patients may have recurrent bacterial and viral infections that are probably related to neutropenia and hypogammaglobulinemia. Impaired cognitive development and neurologic sequelae (epilepsy and gait disturbance) were observed in patients with TCII deficiency who had extended duration of illness, received inadequate cobalamin treatment, or were initially treated with folic acid instead of cobalamin (3, 4). Treatment is long-term, high-dose, intramuscular cobalamin supplementation (1 mg/wk). In some cases, oral therapy can be attempted with success (5).

This is one of the first reports of favorable long-term outcome of TCII deficiency: The neurologic condition of our patient is normal. Our findings indicate that TCII deficiency can be managed without sequelae if high-dose vitamin B$_{12}$ supplementation is administered early and regularly.

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References

Correction: The Effect of HFE Genotypes on Measurements of Iron Overload

In an article on the relationship of iron measurements to hemochromatosis genotypes (1), some errors in the calculation of the specificity of transferrin saturation measurements were presented in Table 5. Corrected data are given in the current Table.

Reference

Table. Corrected Values for Specificity of Transferrin Saturation and Serum Ferritin Level for Detection of C282Y/C282Y Homozygosity*

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* Values in parentheses are 95% CIs.
Quality Indicators for Prevention and Management of Pressure Ulcers

TO THE EDITOR: In the article by Bates-Jensen on quality indicators for prevention and management of pressure ulcers in vulnerable elders (1), the third indicator for prevention concerns nutrition. Bates-Jensen states that since poor diet, particularly low dietary protein intake, is an independent predictor of pressure ulcer development, nutritional intervention or dietary consultation should be instituted if malnutrition is suspected. The sentence concluding this section, however, acknowledges that “no direct evidence shows that adequate nutrition will prevent ulcers.”

In fact, there is a good bit of evidence to the contrary, both for dietary consultation (2) and for nutritional intervention, especially tube feeding (3–5). In many cases, it’s fairly clear that patients who are chronically ill and nearing death develop low albumin levels, weight loss, and many other abnormal “markers of nutritional status,” as well as pressure ulcers, as part of the relentless deterioration. Increasing nutrient intake in cases like this (for example, in patients with cancer) has not been shown to benefit the patient.

It is important to recognize this for at least two reasons. First, many debilitated, vulnerable people are subjected to tube feeding as a result of the logic described by Bates-Jensen, despite the fact that no substantive trial has ever shown benefit. Second, lawsuits against those who provide care to these very ill patients often build on statements like those in her article.

A conscientious, comprehensive effort should be made to provide good food to all who will eat. There is no good evidence that nutritional intervention or dietary consultation beyond this provides any additional benefit at all.

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References