identify *Plasmodium falciparum* as the underlying cause of sepsis, an anesthesiologist diagnose Guillain-Barré syndrome when called to intubate for suspected botulism, and internists appropriately advocate surgery when surgeons were dragging their feet. I suspect that attention to detail and a drive to find the right answer carry more weight than the brand of specialty certification.

Drs. Wilson and Manecke describe in stark terms the corrosive effects of increasingly restrictive interpretations of Residency Review Committee for Internal Medicine communications. My own experience with the Accreditation Council for Graduate Medical Education suggests these fears are more real than imagined. Baystate's program requested a waiver so that anesthesia-trained physicians could be considered key clinical faculty based on program requirement II.C.1.a.2: ". . . have current certification in the subspecialty by the American Board of Internal Medicine or possess qualifications judged by the Review Committee to be acceptable." We were told, quite bluntly, that there are no acceptable alternative qualifications and that American Board of Internal Medicine subspecialty certification is a "must" (Accreditation Council of Graduate Medical Education, personal communication, April 2, 2009), even for individuals initially certified in medicine who have Critical Care certification *via* another board.

The United States is facing a shortage of intensivists (3–5) and we must not needlessly exclude otherwise qualified individuals from training the next generation. There is no time to fiddle while Rome burns.

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REFERENCES

1. Rath T, Conchie B: Strengths-Based Leadership. Washington, Gallup Press, 2009
2. Gardner H: Frames of Mind: The Theory of Multiple Intelligences. Revised Edition. New York, Basic Books, 2004
3. Angus DC, Kelley MA, Schmitz RJ, et al: Current and projected workforce requirements for care of the critically ill and patients with pulmonary disease: Can we meet the requirements of an aging population? *JAMA* 2000; 284:2762–2770
4. Angus DC, Shorr AF, White A, et al, on behalf of COMPACCS: Critical care delivery in the United States: Distribution of services and compliance with Leapfrog recommendations. *Crit Care Med* 2006; 34:1016–1024
5. Cohn SM, Price MA, Villarreal CL: Trauma and surgical critical care workforce in the United States: A severe surgeon shortage appears imminent. *J Am Coll Surg* 2009; 209:446–452

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Pregnant women are at increased risk for severe A influenza because they have low serum 25-hydroxyvitamin D levels

To the Editor:

A recent article in *Critical Care Medicine* (1) pointed out that pregnant women and immunosuppressed patients have increased risk of severe A (H1N1) influenza. One of the reasons they have increased risk is their low serum 25-hydroxyvitamin D levels. Vitamin D deficiency is common in pregnant women (5%–50%) and in breastfed infants (10%–56%), despite the widespread use of prenatal vitamins, because these are inadequate to maintain normal serum 25-hydroxyvitamin D levels (≥32 ng/mL) (2). Risk of influenza is linked to low serum 25-hydroxyvitamin D levels (3). A recent randomized, controlled trial involving Japanese school children found that for those taking 1200 IU/d of vitamin D3 and no additional vitamin D3, the relative risk of influenza A was 0.36 (95% confidence interval, 0.17–0.79) (4). In addition, vitamin D protects against ensuing pneumonia. In an ecological study of case fatality rates in the United States during the 1918 influenza pandemic, the index of summer solar ultraviolet-B dose explained 46% of the variance for 12 communities surveyed by The United States Public Health Service (5). Two explanations were provided for this finding: reduced proinflammatory cytokine production by 1,25-dihydroxyvitamin D and induction of cathelicidin by 1,25-dihydroxyvitamin D. Cathelicidin has antimicrobial and antiendotoxin effects.

Thus, especially during flu season but also any time of the year, pregnant women should be encouraged to increase their serum 25-hydroxyvitamin D levels to 40 to 80 ng/mL through supplementation with several thousand international units per day of vitamin D3 or solar ultraviolet-B when the sun is high enough that one's shadow is shorter than one's height. This amount is safe for >99% of the population, and the higher serum 25-hydroxyvitamin D levels will reduce the risk of many diseases in addition to influenza and pneumonia, including other bacterial and viral infectious diseases, cancer, cardiovascular disease, autoimmune diseases, as well as adverse pregnancy outcomes.

Dr. Grant has received funding from the UV Foundation (McLean, VA), the Sunlight Research Forum (Veldhoven), Bio-Tech-Pharmacal (Fayetteville, AR), and the Vitamin D Council (San Luis Obispo, CA), and has received funding from the Vitamin D Society (Canada). Dr. Cannell is founder and principal of the nonprofit educational group, the Vitamin D Council, and also receives royalties from Purity Products Inc.

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REFERENCES

1. Lapinsky SE: H1N1 novel influenza A in pregnant and immunocompromised patients. *Crit Care Med* 2010; 38(4 Suppl):e52–e57
2. Holmes VA, Barnes MS, Alexander HD, et al: Vitamin D deficiency and insufficiency in pregnant women: A longitudinal study. *Br J Nutr* 2009; 102:876–881
3. Cannell JJ, Zasloff M, Garland CF, et al: On the epidemiology of influenza. *Virol J* 2008; 5:29
4. Urashima M, Segawa T, Okazaki M, et al: Randomized trial of vitamin D supplementation to prevent seasonal influenza A in schoolchildren. *Am J Clin Nutr* 2010; 91:1255–1260
5. Grant WB, Giovannucci D: The possible roles of solar ultraviolet-B radiation and vitamin D in reducing case-fatality rates from the 1918–1919 influenza pandemic in the United States. *Dermatol Endocrinol* 2009; 1:215–219

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The author replies:

Drs. Grant and Cannell raise the interesting possibility that the increased susceptibility of pregnant women to influenza may be the result of vitamin D deficiency. There is increasing evidence that vitamin D has a number of roles beyond its traditional role in calcium metabolism, including improving immune function. Vitamin D deficiency has been suggested to be a contributor to poor outcome in intensive care unit patients in general (1). Furthermore, the bioavailability of vitamin D may be reduced in obese patients as a result of increased

uptake by adipose tissue (2), potentially accounting for another important risk factor for severe pneumonitis resulting from influenza A (pH1N1).

Although this hypothesis is thought-provoking, the data are still somewhat limited, and further research is required. A Cochrane Review in 2000 found that there was not enough evidence to evaluate the effects of vitamin D supplementation during pregnancy (3). Nevertheless, vitamin D administration appears to be safe in the pregnant patient and some nutritional recommendations may be insufficient to produce adequate serum levels in this at-risk group (4).

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REFERENCES

1. Lee P, Nair P, Eisman JA, et al: Vitamin D deficiency in the intensive care unit: An invisible accomplice to morbidity and mortality? *Intensive Care Med* 2009; 35:2028–2032
2. Wortsman J, Matsuoka LY, Chen TC, et al: Decreased bioavailability of vitamin D in obesity. *Am J Clin Nutr* 2000; 72:690–693
3. Mahomed K, Gülmezoglu AM: Vitamin D supplementation in pregnancy. *Cochrane Database Syst Rev* 2000; 2:CD000228
4. Hollis BW, Wagner CL: Assessment of dietary vitamin D requirements during pregnancy and lactation. *Am J Clin Nutr* 2004; 79:717–726

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Parenteral feeding and intensive insulin therapy

To the Editor:

The recent report of the Leuven intensive insulin therapy strategy from the surgical and medical cohorts documented an increase in mean glucose amplitude variation defined as the mean of the daily difference between minimum and maximum blood glucose in the intensively treated groups, which along with a mean morning blood glucose >6.1 mmol/L and the occurrence of hypoglycemia were associated with mortality (1). The mean daily minimum and maximum blood glucose was lower in the surgical cohort compared with the medical cohort, which may explain the better mortality results with intensive insulin therapy in the surgical intensive care unit patients (2). A factor worth exploring is the substantial use of total parenteral nutrition in the surgical cohort (3) with the majority receiving exclusive total parenteral nutrition and/or combined feeding, whereas there appeared to be exclusive enteral feeding used in the medical cohort (4). Enteral feeding is more likely to be interrupted and absorption rates of nutrients variable, making hypoglycemia more likely and perhaps mean daily minimum and maximum blood glucose greater. In fact, in the surgical cohort, 62% of the hypoglycemic events were associated with interruption of enteral feeding (3), whereas in the medical cohort with enteral feeding only, hypoglycemia was much more common than in the surgical study (2). It would be valuable for the authors to explore this possibility using their amassed data, because it might support a recommendation that the provision of parenteral feeding either totally or as a substantial component of the total glucose can be an effective adjunct to intensive insulin therapy to reduce the incidence of hypoglycemia and perhaps the mean daily minimum and maximum blood glucose and thereby further improve mortality. As well, it might in part explain the variable results from the subsequent studies of intensive insulin therapy, because it appears that only the initial surgical study extensively used total parenteral nutrition among other tests of this strategy (5).

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REFERENCES

1. Meyfroidt G, Keenan DM, Wang X, et al: Dynamic characteristics of blood glucose time series during the course of critical illness: Effects of intensive insulin therapy and relative association with mortality. *Crit Care Med* 2010; 38:1021–1029
2. Krinsley JS: Glycemic variability in critical illness and the end of Chapter 1. *Crit Care Med* 2010; 38:1206–1208
3. Van den Berghe G, Wouters PJ, Bouillon R, et al: Outcome benefit of intensive insulin therapy in the critically ill: Insulin dose versus glycemic control. *Crit Care Med* 2003; 31: 359–366
4. Van den Berghe G, Wilmer A, Hermans G, et al: Intensive insulin therapy in the medical ICU. *N Engl J Med* 2006; 354:449–461
5. Bistrian BR: Tight glucose control in critically ill adults. *JAMA* 2008; 300:2726

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The authors reply:

We thank Dr. Bistrian for his interest in our article (1). He raises the hypothesis that the route of feeding may affect blood glucose fluctuation and risk of hypoglycemia and may explain the differences in these blood glucose time series characteristics between previously studied surgical (2) and medical patient cohorts (3). Predominantly parenteral, and thus more continuous, caloric delivery could prevent large blood glucose fluctuations. Inversely, predominantly enteral delivery, with more variable absorption of nutrients and more frequent interruptions, could be the cause of higher blood glucose fluctuations. On suggestion by Dr Bistrian, we re-examined the merged database of the two adult Leuven randomized, controlled studies on intensive insulin therapy to address this interesting question.

First, we compared the feeding strategy in the two studies. Indeed, in the surgical cohort in 2001, only 19% of the patients received enteral nutrition vs. 65% of the patients in the medical cohort ($p < .0001$) in 2006. When merging the two data sets, 1066 of the 2748 patients (38%) received enteral nutrition. Enterally fed patients were equally distributed over the intensive and the conventional treatment groups (40% in the conventional group, 38% in the intensive group; $p = .2$).

Second, we examined the impact of the route of feeding on blood glucose fluctuations. The mean difference between maximum and minimum blood glucose value per day (mean daily ΔBG) was significantly higher in patients who received enteral nutrition (4.1 [2.7–5.6] mmol/L) than in patients who were fed exclusively parenterally (3.5 [2.4–5.2] mmol/L) ($p < .0001$).

Third, we assessed whether the route of feeding affected the risk of hypoglycemia. We addressed this question by including the route of feeding into a multivariable logistic regression model together with baseline risks, surgical intensive care unit vs. medical intensive care unit, randomization, and correcting for length of stay in the intensive care unit. Receiving enteral nutrition was indeed an independent risk factor for hypo-