LETTERS

To the Editor:

The article "Osmolality of Commonly Used Medications and Formulas in the Neonatal Intensive Care Unit" reports the osmolality of drugs, vitamin supplements, and feeding mixtures used in neonatal intensive care units (NICUs).[1] These osmolalities are discussed in terms of risk for necrotizing enterocolitis (NEC) and other gastrointestinal concerns; however, one piece of information was missing. Laboratory measures of osmolality of drugs and vitamin supplements can have little or no relation to the osmotic gradient set up across a biological membrane.[2] Most of the osmolality in a drug or vitamin supplement can come from the carrier or stabilizer molecules that may or may not diffuse readily across membranes.[2]

Osmolality is only a valid measure of risk if the molecules set up an osmotic gradient across a membrane *in vivo*. The osmometer measurement reflects the number of particles in the solution, which can be made up of both substances that passively diffuse across membranes as well as those that do not passively diffuse. The piece of missing information in the article is the type of molecules in the drug and vitamin supplement solutions. If the osmolality is high from passively diffusing substances, then there is no risk; if it is high from molecules such as a simple sugar, there is a risk.

The authors documented the very high osmolalities of drugs and vitamin supplements used in the NICU. Yet, in spite of these very high osmolalities, these mixtures are often well tolerated. There is no literature linking a high osmolality of drugs or vitamin supplements and NEC. The literature reviewed in the article linking osmolality to NEC were examples of a sucrose-based calcium lactate formula[3] and a high-osmolality elemental formula.[4]

Osmolalities of concern are those that are elevated from substances that do not passively diffuse across membranes, ie, especially sugars, minerals, and amino acids. These nutrients can make a large contribution to osmolality. Glucose polymers and intact protein give a lower osmolar load because these macromolecules have fewer individual particles. Laboratory measures of osmolality from nutrients that do no passively diffuse give a good measure of potential risk. Additions of these modulars require careful review and guidelines for the NICU. Any drug that has a suspension of a sugar syrup or another osmotically active substance is also a concern.

Therefore, the composition of drugs and vitamin supplements needs to be known, and the effects of these constituents must be understood to assess risk in terms of the osmolality reading.

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To the Editor:

The recent Teaching Case publication by Pahwa et al[1] presented a challenging and difficult obese patient who required prolonged nutrition support secondary to multiple medical and surgical complications. We have concerns with several points made in this Teaching Case. The first is the use of albumin (ALB) and prealbumin (PAB) to monitor the response to nutrition. As the authors point out, ALB and PAB are not indicative of nutrition response in several clinical conditions. In this Teaching Case, the patient's serum ALB dropped from admission in the postoperative state and continued to decrease after subsequent surgeries. PAB levels also remained depressed throughout this time period. The investigators attributed this response to inadequate nutrition support. To the contrary, in this patient experiencing pancreatitis and sepsis, two highly stressful conditions, depressed PAB levels were due to metabolic stress, with the liver reprioritizing visceral protein synthesis.[2] The authors even admit that the patient's PAB dropped although the patient's "general condition improved," demonstrating that many factors affect PAB in complicated patients. Also fluid shifts in the postoperative state are notorious for depressing ALB levels. Therefore, these biochemical parameters are not appropriate markers for response to nutrition support. In our clinical experience, serum ALB and PAB do not increase until the metabolic stress subsides.

The use of PAB to monitor nutrition response resulted in the patient's receiving approximately 47 kcal/kg adjusted dry weight and 2.5 g protein/kg adjusted dry weight during the 9th and 10th week of the hospital stay. At this time, the literature does not

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support exceeding 35 to 40 kcal/kg and 1.5 to 2 g protein/kg in the stressed patient because of the metabolic derangements that can occur. It is our experience that patients, especially obese patients, who receive above these recommendations experience hyperglycemia, hyperuricemia, and hypertriglyceridemia. The authors failed to provide these biochemical data in this Teaching Case. It would be interesting to know these values in a patient experiencing poor wound healing. Hyperglycemia and hyperuricemia are known to compromise immune status and impair would healing and should be avoided.

In conclusion, we believe biochemical markers are just one of many parameters to monitor in response to nutrition support, a point we highly stress in our teaching institutions. The use of ALB and PAB to monitor the response to nutrition must be used with extreme caution in the critically ill patient because many nonnutritional factors affect their levels, and overfeeding can occur with resulting detrimental metabolic derangements.

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To the Editor:

The authors thank Han-Markey, Lown, and Braunschweig for their comments regarding our Teaching Case. As indicated in the case report, wound granulation and prealbumin were used as indicators of the patient's response to nutrition support. Albumin was not used because it is not an indicator of nutritional status or response in the critically ill.

Biochemical indices were monitored to assess any metabolic complications as is routinely done at our institution for all patients. Prealbumin was not evaluated during the patient's bout with pancreatitis and sepsis or immediately after his surgeries. They were monitored after the 5th week of hospitalization when stress had subsided. A correction is needed to clarify that the patient had candida infection in the eighth week, not the more stressful sepsis as stated in the article. We regret the error and the ensuing confusion. As their letter states, this case was very difficult because of the large surgical wounds and skin breakdown that are hard to heal in such an obese individual. During the final septic period prior to his demise, prealbumin was not monitored but during the period of candida infection, the trend was followed along with wound granulation and usual biochemical indices.

The article presents intake based on adjusted body weight. Ireton-Jones and Turner[1] suggest that the use of actual body weight is more appropriate to calculate needs for the obese patient. If one calculates this patient's maximum intake during weeks 9-10 using actual dry weight, the support provided was 37.5 kcal/kg and 1.97 g protein/kg/d. This is within the ranges recommended by these reviewers and others. Even at this level of support, the patient was losing somatic muscle and fat mass with poor wound healing. Measured energy expenditure in the obese, nonhospitalized individual is significantly higher than nonobese, healthy adults.[2] This is probably a result of the fact that there is an increase in both body cell mass and body fat in obesity.[3,4]

The team monitored the patient for problems associated with overfeeding. Uric acid levels remained within normal range until weeks 17-18 when nutritional intake was 29 kcal/kg actual weight (102 kg). After this period the uric acid returned to normal. Postoperative pancreatitis was resolved but the patient was monitored using triglyceride, amylase, and lipase values. Triglyceride levels remained within an adequate range considering his infectious process and liver abcess and serum amylase. Lipase levels indicated that there was no recurrent pancreatitis. Prothrombin time and PTT were within normal range indicating liver function was adequate for synthesis of clotting factors and visceral proteins. Blood sugar levels were maintained in the 150-210 range indicating that this critically ill patient was managed conservatively. Thus, biochemical parameters indicated that overfeeding was not a concern.

We encourage more case reviews of the obese patient and nutrition support. We agree that there is no "gold standard" to follow such patients' nutritional improvement and wish to add to the body of knowledge that can improve the nutritional care of these patients. Each obese patient must therefore be individually assessed and monitored to provide the appropriate nutritional management.

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