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Letters to the Editor

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O₂ Saturation and Slings

To the Editor.—

I read with interest the recent article on cardiorespiratory stability of infants carried in slings by Stening et al.¹ This is an important topic for which objective data have not been available. Unfortunately, the article suffers from a number of significant flaws. The most glaring is the definition of desaturation as a decline in oxygen concentration to <88% for at least 10 seconds. There are 10 published studies on the normal oxygen saturation of infants. In a recent review of this issue by Poets,² it was determined that the baseline saturation of infants is 93% to 100% in term and preterm neonates and 97% to 100% in term and preterm infants; therefore, the current study has not been able to identify the number of abnormal desaturation events that occur in a sling.

It is also troubling that the authors make the assumption that, in the absence of outright respiratory arrest, significant apnea, or bradycardia, chronic and/or recurrent subclinical oxygen desaturation should not be considered a "clinically relevant" event. There are published reports of both cognitive³ and behavioral difficulties⁴ in infants who are exposed to chronic or recurrent hypoxia. The authors therefore should be more guarded in their conclusions, because the most we can say is that there were no acute events observed.

Another problem in the study is that the authors monitored their patients for a time period of only 20 minutes. This is unfortunate, because previous studies of desaturation associated with car seats^{5,6} have documented very significant drops in saturation that may not occur until after an hour of study. In addition, studies in preterm infants have demonstrated that baseline hypoxemia may be associated with apparent life-threatening events.⁷ Considering all of the above, I think it is premature to reassure parents that using a sling is not without clinically relevant risk.

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In Reply.—

We agree with Dr Bass that the 20-minute observation intervals in our study are relatively short. We intentionally chose this design to exclude potential bias, eg from effects of diurnal rhythm or developmental stage.¹ Furthermore, we tried to minimize strain on mother and child. To investigate whether the vital parameters changed with longer observation, we subdivided the observation interval into 3 phases of ~7 minutes and compared the measured mean values of oxygen saturation and heart frequency. There was no difference in these parameters between the 3 phases. Of course, this does not exclude the possibility of progressive desaturation after a longer carrying time in the sling. Nevertheless, a comparison with the studies of car seats is only possible to a limited extent. Unlike the studies cited^{2,3} in which children were observed in a static condition (in a car seat placed in a nursery), our study was intended to examine vital parameters during the more life-like dynamic condition of a normal stroll. Most probably, the children were constantly stimulated by motion, sounds, or their parents' voices.

With regard to the issue of "normal" oxygen saturation values, we point out again that, in the crossover design of our study, children served as their own controls. Saturation values of babies carried in a sling were compared with those of the same babies in a pram. A comparison with the normal values described in Poets' review⁴ is difficult for several reasons. Poets states that his oxygen saturation values are based exclusively on recordings during periods of "regular breathing (corresponding to quiet sleep) and excluding apneic pauses." We did not exclude any measurements on the basis of the prevailing respiratory pattern during the recording period. Poets cites two studies^{5,6} that describe oxygen saturation in children of similar postconceptional or postnatal age to the premature and term infants in our study. The preterm infants ($n = 160$) had a median baseline oxygen saturation (SpO₂) of 99.5%; however, the lowest baseline SpO₂ was 88.7%. In term infants ($n = 60$) the median was 98.0%, and the lowest baseline value 86.6%. These so-called normal values in healthy children are lower than in the children of our study. Unfortunately, Poets only cites median values, whereas our study is based on means. Of course, these two different values cannot be compared directly because SpO₂ levels are not normally distributed but skewed toward 100%. Furthermore, Poets' review does not state for what proportion of the time under observation children had SpO₂ values of <93% or 97%. Poets does, on the other hand, specify that 355 desaturation episodes were observed in the premature infants and 165 in the term babies (these were defined as SpO₂ < 80%). This would equate to 6 to 7 desaturation episodes per hour in the sample described in our study. However, as stated in the article, none of our children desaturated below 80%.

Similarly, our study cannot really be compared to the studies cited describing outcome in infants with presumed postnatal hypoxemia.^{7,8} In our study, we only included healthy preterm and term infants with stable cardiorespiratory measurements who did

not need additional oxygen on the day of observation. One would expect much lower mean Spo₂ values in infants with cyanotic heart failure.⁷ Also, Lou's⁸ study on attention-deficit hyperactivity disorder describes infants with recurrent episodes of hypoxemia. Using the definition of hypoxemia given by Poets^{4,9} (i.e. oxygen saturation <80% for at least 4 seconds), none of our infants suffered from hypoxemia while being carried in the sling. Tin et al¹⁰ pointed out in their study that the saturation level in preterm infants is of only little influence on neurological outcome and on the development of retinopathy. Their study showed that the development of cerebral palsy in 296 preterm infants <28 gestational weeks did not correlate with the saturation level (target saturation level between minimum 70%–90% and maximum 88%–98%). Moreover, the percentage of threshold retinopathy in 1-year survivors increased with the saturation level.

As mentioned in our article, we only observed desaturation episodes (<88%) in preterm babies. We advised caution with the use of slings for carrying preterm infants before they reach term postconceptional age, although no desaturation below 80% was seen in our study, and the clinical relevance of desaturation to Spo₂ levels between 88% and 80% is unclear. We remain satisfied with our conclusion that term babies are not at risk of desaturation while being carried in the sling.

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Gelatin Allergy

To the Editor.—

We feel relieved after reading the paper by Pool et al and the VAERS Team¹ on the prevalence of gelatin allergy in the United States. They conducted a retrospective analysis after measles-mumps-rubella (MMR) vaccination. Among 26 cases of anaphylaxis, only 6 (27%) were positive for anti-gelatin IgE antibodies. The rate of anaphylactic reactions reported to the VAERS is 1.8 per 1 million doses, and no substantial increase in number of reported allergic events after MMR was observed since the introduction of

gelatin-containing diphtheria-tetanus-acellular pertussis vaccine (DTaP) in 1997. We reported that the cases of anaphylaxis or urticaria showed high positive rates of anti-gelatin IgE antibodies, and we speculated the causal relationship of the sensitization by gelatin-containing DTaP.² Discontinuation of gelatin-containing DTaP reduced the incidence of anaphylaxis after 1999,³ and we have no report of anaphylaxis after vaccination with live virus vaccines containing hydrolyzed porcine gelatin in the last few years. Thus, we were solicitous for the incidence of anaphylaxis in the United States, but they reported that the incidence of gelatin allergy was lower than that observed in Japan.

But we suppose the different prevalence of anti-gelatin IgE depends on sensitivity for the detection of IgE antibodies against gelatin and especially on the nature of antigen for the assay. The same was the reason why the sensitization against gelatin increased in Japan. Some vaccine manufactures used poorly hydrolyzed bovine gelatin in DTaP, and some used hydrolyzed porcine gelatin. A large number of patients with anaphylaxis had a history of having DTaP containing poorly hydrolyzed bovine gelatin. Poorly hydrolyzed bovine gelatin was immunogenic when administered with alum adjuvant. They did not mention the nature of gelatin in DTaP in the United States in their paper, and we suppose that it was probably highly hydrolyzed porcine gelatin (2–3 kDa). Although it is considered as less immunogenic, gelatin-free DTaP is desirable to avoid the possibility of unnecessary sensitization against gelatin.

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In Reply.—

Drs Nakayama and Kumagai note the difference in the prevalence of anti-gelatin IgE antibodies found in sera from patients suffering anaphylactic reactions to measles-mumps-rubella (MMR) vaccines in their study in Japan (93%)¹ and our study in the United States (27%).² They suggest that this difference may be due to differences in the sensitivity and specificity of tests to detect anti-gelatin IgE, which in turn may depend on the nature of gelatin used in the assay. The solid-phase allergen for the radioimmunoassay we used was made from a random lot of flavored sugared commercial gelatin (Jell-O) and thus not exactly the same gelatin that is present in MMR vaccines. It is not clear if the "bovine gelatin" used in their assay was the same as that used in vaccines manufactured in Japan. However, we believe that differences between the gelatin in the immunoassay and the gelatin in the vaccine are unlikely to be the primary explanation for the difference in prevalence of anti-gelatin IgE found in the Japanese and US studies. In the first case report describing gelatin allergy as a cause of anaphylaxis to MMR, inhibition immunoassays were performed.³ The patient's anti-gelatin IgE antibodies directed against gelatin (the same type of gelatin used in the assay in our present study) were inhibited not only by both bovine and porcine laboratory gelatins but also by the MMR vaccine itself containing

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