

are almost certainly skewed to the more serious injuries, because minor trauma is often treated at home without medical intervention. We also stress that additional studies need to be performed on the efficacy of the individual soft surfaces.

In our study, we referenced studies by Farnsworth⁴ and Chalmers,⁵ both of which describe the height of the equipment as a relative risk factor. They do not make recommendations regarding the surfaces below the equipment, however. In their study, Mott et al⁶ acknowledge: "Most injuries were equipment-related. Surface types cannot be considered in isolation from equipment." We make no claims as to how the surfaces below the equipment are maintained. We did not inspect the individual playgrounds to ensure compliance with ASTM standards. We simply reported on the surfaces and the related injuries. We feel that we obtained a wide cross-section of the playgrounds in the greater Boston area. Whether or not these playgrounds met the ASTM standards is unknown and was not the primary purpose of the study. Finally, we acknowledge that there is a difference between "supervision" and "being there." In our telephone survey, we asked specifically if the child was being supervised by a person over the age of 18. A parent's definition of supervision is the key. If a parent feels that watching his/her child play is synonymous as manually supporting him/her as he/she climbs, then that is how supervision needs to be defined. We made no conclusions as to the level of supervision. We allowed the parents the freedom to define their level of supervision as they deemed appropriate. The level of supervision does have a significant role in allowing the child to use the equipment, which is the reason we included this issue in our study.

We wish to thank Drs Thompson and Hudson for their observations of our study, and we hope we have clarified any issues that they had. However, based on our findings in conjunction with our experience in caring for children with monkeybar/jungle gym injuries, we find no reason to alter our conclusions.

MARK L. WALTZMAN, MD
MICHAEL SHANNON, MD, MPH
ANNE P. BOWEN, MS, RN
MARY CHRISTINE BAILEY, MD
Children's Hospital
Boston, MA 02115

REFERENCES

- Sosin DM, Keller P, Sacks J, et al. Surface-specific fall injury rates on Utah school playgrounds. *Am J Public Health.* 1993;83:733-735
- Mott A, Rolfe K, James R, et al. Safety of surfaces and equipment for children in playgrounds. *Lancet.* 1997;349:1874-1876

- Mott A, Evans R, Rolfe K, et al. Patterns of injuries to children on public playgrounds. *Arch Dis Child.* 1994;71:328-330
- Farnsworth CL, Silva MS, Mubarak SJ. Etiology of supracondylar humerus fractures. *J Pediatr Orthop.* 1998;18:38-42
- Chalmers DJ, Langley JD. Epidemiology of playground equipment injuries resulting in hospitalization. *J Paediatr Child Health.* 1990;26:329-334
- Mott A, Rolfe K, James R, et al. Safety of surfaces and equipment for children in playgrounds. *Lancet.* 1997;349:1874-1876

Is the G985A Allelic Variant of Medium-Chain Acyl-CoA Dehydrogenase a Risk Factor for Sudden Infant Death Syndrome? A Pooled Analysis

To the Editor.—

Studies examining the relationship between medium-chain acyl-CoA dehydrogenase deficiency (MCADD) and sudden infant death syndrome (SIDS) have shown conflicting results. With >90% of individuals diagnosed with MCADD possessing at least one copy of the G985A allelic variant, it seems likely that if an association between MCADD and SIDS existed, an association would also be seen between the G985A and SIDS. We therefore assessed the results from 11 studies published through 1998 that evaluated the relationship between G985A and SIDS.

Table 1 summarizes the results of the pooled analysis. Since rates for SIDS and G985A are different between the United States and elsewhere (Europe and Australia), analyses were performed stratified by the 2 regions. Using published data on the rates of SIDS and G985A in different populations and the proportion of infants with SIDS who had 1 or 2 G985A alleles, we used Bayes' theorem to estimate the probability of an infant with 1 or 2 G985A alleles dying from SIDS. The probability of SIDS among persons homozygous for the G985A allele was estimated as 1% in the U.S. (range: 0%–68%) and 3% for Europe and Australia (range: 0%–77%). This estimate is 10 times higher than the risk for SIDS in the US population and 32 times higher than the risk for SIDS in the European and Australian populations. Using Miettinen's formula, we estimated that the proportion of SIDS in both populations that can be attributed to homozygosity for G985A was <.1%. The probability of SIDS among infants heterozygous for the G985A allele was also estimated as <.1% for the United States and Europe and Australia, and produced risks .6 and .3 times the rate of SIDS in the United States, and Europe and Australia, respectively.

Several issues need to be considered in interpreting these find-

TABLE 1. Pooled Analysis of Risk for SIDS According to G985A Homozygosity and Heterozygosity

Geographic Region	SIDS cases								Non-SIDS Controls			
	Number of SIDS	G985A/G985A				G985A/-				Number of Controls	G985A/G985A	G985A/-
		Number	P(SIDS)†	RR‡	AF§	Number	P(SIDS)†	RR‡	AF§			
United States												
Maryland ¹	309	1	6.8%	68	.3%	1	.03%	.3	na	—	—	—
California ^{1,2}	1224	0	0%	—	—	3	.02%	.2	na	—	—	—
Maryland (a) ^{*3,4}	262	0	0%	—	—	3	.1%	1	na	—	—	—
New York ^{1,3,4}	67	0	0%	—	—	3	.4%	4	na	70	0	1
North Carolina (a) ^{2,4}	119	0	0%	—	—	4	.3%	3	na	2611	0	31
Total	1981	1	1%	10	.04%	14	.06%	.6	na	2681	0	34
Europe and Australia												
France ³	100	0	0%	—	—	1	.1%	1	na	—	—	—
France ³	57	1	77%	855	1.7%	0	0%	—	—	—	—	—
Denmark ^{1,3}	120	0	0%	—	—	1	.1%	0.8	na	—	—	—
Scotland ³	233	0	0%	—	—	3	.1%	1	na	552	0	2
Germany (a) ^{2,4}	153	0	0%	—	—	0	0%	—	—	200	0	14
Australia (a) ²	708	0	0%	—	—	7	.1%	1	na	—	—	—
Total	1371	1	3%	32	.07%	5	.03%	.3	na	752	0	16

* (a): abstract.

† P(SIDS): $P(\text{SIDS}|\text{G985A}/\text{G985A}) = (P(\text{G985A}/\text{G985A}|\text{SIDS})) (P(\text{SIDS})/P(\text{G985A}/\text{G985A}))$.

$P(\text{SIDS}|\text{G985A}/-) = (P(\text{G985A}/-|\text{SIDS})) (P(\text{SIDS})/P(\text{G985A}/-))$.

‡ RR (relative risk) = $(P(\text{SIDS}|\text{G985A}/\text{G985A})) / (P(\text{SIDS}|\text{not G985A}/\text{G985A}))$.

§ AF (population attributable fraction) = $(P(\text{G985A}/\text{G985A}|\text{SIDS}))((\text{RR}-1)/\text{RR})$.

ings. First, 7 studies are case series and 4 studies are published as abstracts. However, comparisons of SIDS cases to reported control groups produced similar results, and analyses excluding abstracts do not change the conclusions. Second, we excluded many studies in the literature for incorrect definitions of SIDS, the sudden and unexplained death of an infant younger than 1 year of age; however, studies included in this analysis may suffer from potential selection bias of their selected SIDS cohort. Third, none of the studies possess adequate power to detect G985A homozygosity, and their small sample sizes lead to unstable allele frequency estimates, resulting in wide ranges of penetrance and relative risk. Last, the lack of stratification by racial and ethnic groups is of concern because heterogeneity for both G985A and SIDS exists.

Despite these limitations, the data summarized are the best available for assessing the relationship between MCADD and SIDS. The data suggest that infants homozygous for G985A may have an increased risk for SIDS, whereas infants heterozygous for G985A do not. Furthermore, the G985A MCAD allelic variant accounts for a minimal percentage of SIDS cases in the United States and Europe and Australia. There is clearly a need for large population-based studies to appropriately elucidate this relationship (see reference 5).

SOPHIA S. WANG, PhD*†

PAUL M. FERNHOFF, MD‡

MUIN J. KHOURY, MD, PhD*

*Centers for Disease Control and Prevention
National Center for Environmental Health
Office of Genetics and Disease Prevention
Atlanta, GA 30341-3724

‡Emory University
Department of Pediatrics
Division of Medical Genetics
Atlanta, GA 30322

REFERENCES

1. Boles R, Buck E, Blitzer M, et al. Retrospective biochemical screening of fatty acid oxidation disorders in postmortem livers of 418 cases of sudden death in the first year of life. *J Pediatr* 1998;132:924-933
2. Arens R, Gozal D, Jain K, et al. Prevalence of medium-chain acyl-coenzyme A dehydrogenase deficiency in the sudden infant death syndrome. *J Pediatr*. 1993;122:715-718
3. Ged C, El Sebai H, de Verneuil H, Parrot-Rouleau F. Is genotyping useful for the screening of medium chain acyl-CoA dehydrogenase deficiency in France? *J Inherit Metab Dis*. 1995;18:253-256
4. Miller M, Brooks J, Forbes N, Inset R. Frequency of medium-chain acyl-coA dehydrogenase deficiency G985 mutation in sudden infant death syndrome. *Pediatr Res*. 1992;31:305-307
5. Wang S, Fernhoff P, Hannon H, Khoury M. Medium chain acyl CoA dehydrogenase (MCAD) deficiency human genome epidemiology (HuGE) review. *Genet Med*. 1999;1:332-339

Evidence-Based Medicine

To the Editor.—

Regarding Dr Bauchner's comments in an article published in your journal,¹ I was struck by the author's concern that evidence-based medicine (EBM) "may be distracting from the important goal of finding ways to change physician behavior." Dr Bauchner discussed in a nice overview the rigors of the statistical methods incorporated in the definition of EBM. The components of the EBM definition neglected, however, concerns "making decisions . . . about the care of individual patients."

It has been argued that 70% of the management decisions we apply as pediatricians have not been approved for use in the pediatric age group. Therefore, practicing pediatricians have little evidence to go on. The AAP serves as a demonstrated leader in organizing consensus recommendations from the data that does exist which impacts on the pediatrician's clinical expertise in the form of clinical practice guidelines. This AAP contribution truly supports practicing medicine that is evidence-based, which I argue is not the same entity as practicing EBM. This conceptual difference was not clearly distinguished in Dr Bauchner's article. The realm of clinical expertise is not isolated to relevant informa-

tion gleaned from scientific articles nor clinical practice guidelines, but also includes the years of training and practice experience of the pediatrician.

The true strength of EBM is in taking the realm of a physician's clinical expertise after the clinical database is obtained, stepping back, and asking "What is wrong with *this* patient?" With this overview the pediatrician can then apply the EBM decision-making model to the individual patient,² which helps to prevent the easy temptation to blindly proceed down a clinical practice guideline. A critical review of relevant literature is helpful in this process if available, but statistical analysis is not a requirement in practicing EBM. The definition of EBM cannot stand alone, and needs to be incorporated into the decision-making model. To the pediatrician, the statistical part of EBM is only 30% of the task; the other 70% of the process should be devoted to making decisions.

Effective training in EBM will demonstrate its strength in making clinical decisions, which I argue should accentuate rather than distract the important goal of finding ways to change physician behavior. In the excellent lead your journal has taken at introducing EBM into pediatrics, I encourage writers to your journal to contribute material that additionally demonstrates the application of the decision-making component of EBM.

GARY M. ONADY, MD, PhD

Medicine-Pediatrics Program

Wright State University School of Medicine

Frederick A. White Health Center

Dayton, OH 45401-0927

REFERENCES

1. Bauchner H. Evidence-based medicine. *Pediatrics*. 1999;103:1029-1030
2. Richardson SW, Wilson MC, Nishikawa J, Hayward RSA. The well built clinical question: a key to evidence-based decisions. *ACP J Club*. 1995; 123:A12-13

In Reply.—

I found the comments by Dr. Onady quite erudite, and I appreciate the opportunity to further discuss evidence-based medicine (EBM).

My commentary was meant as an introduction to EBM for readers of *Pediatrics*. There had been no previous editorial about EBM in *Pediatrics*, and hence my comments represent only a brief synopsis of the subject. I have been impressed that as I encounter practitioners around the country, only some are aware of EBM.

I, too, believe that evidence must be combined with clinical experience before making decisions with patients about their care. My comments were not meant to minimize the importance of clinical experience. Interestingly, a series of articles in *Lancet* recently raised concerns about the ethics of EBM with regard to clinical experience and individual patient preference.¹ It is an enlightening series. My colleagues and I recently submitted a paper about EBM for the practitioner that further discusses the intersecting circles that lead to a clinical decision. They include clinical experience, evidence from the literature, societal values, and individual patient (and family) preference.

I applaud the Academy's attempt to make the practice of pediatrics more evidence-based. Certainly the consensus statements help fill gaps when data are unavailable. Unfortunately, it is unclear how much evidence is reviewed when these statements are promulgated. It would be helpful if the amount of evidence supporting these statements was rated, similarly to what has been done in the *Guide to Clinical Preventive Services*. I was pleased to see that in the most recent Academy practice parameter (guideline) on urinary tract infections, the quality of evidence was included.²

Dr Onady comments briefly on the issue of statistics. Recently we developed a curriculum about EBM for residents in our primary care track. It is an 8-part series of small-group discussions, that includes information about reviewing certain types of articles (diagnostic tests, therapeutic advances, practice guidelines, etc), specific discussion about EBM and changing physician behavior, and concludes with the residents preparing a critically appraised topic. Our faculty have discussed at length how much statistics are necessary to present to residents as part of this curriculum. Currently, we have decided that the resident should understand sensitivity, specificity, positive and negative predictive values, *P* val-