COMMENTARIES

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Should We Be Teaching Residents How to Bill for Their Outpatient Services?

ABBREVIATION. RVU, relative value unit; RVUw, work relative value unit; APC, ambulatory payment class; HCFA, Health Care Financing Administration; CPT, Current Procedural Terminology; OIG, Office of the Inspector General.

The article entitled "What if Residents Could Bill for Their Outpatient Services" by Ng and Lawless[1] in this month's issue of *Pediatrics* describes the potential impact on a pediatric outpatient clinic if billing sheets completed by residents were used as the actual source of sending charges to third-party payers. Current Procedural Terminology codes generated by a blinded review of the resident's outpatient notes were compared with those produced by the resident. For acute care visits, resident codes disagreed with those of the reviewer 62% of the time with 83% of the disagreements resulting from under coding by residents. The authors estimated that this under coding would have resulted in a 1-year reduction in collections of $43 676. Residents' accuracy in coding did not improve with additional years of training. Using a sophisticated model employing relative value units (RVUs), work relative value units (RVUws), ambulatory payment class (APCs) reimbursement rates based on national Medicaid data, numbers of residents in the program, visits per resident, and resident time in continuity and acute care clinics, the authors estimated that the total yearly revenue for the outpatient clinic that could be generated by residents billing correctly would be $4 516 123. First-year residents could be expected, based on the model, to generate $67 239; second-years residents, $87 593; and third-year residents, $96 072. We are not told how accurately the faculty supervisors of these residents actually coded for the services that they performed in the context of their supervisory duties or how this compared with the hypothetical amounts derived if the residents' shadow forms had been used to generate bills. Although not surprising, these findings will be interesting and provocative for continuity clinic directors and preceptors.

Of course, training programs that receive graduate medical education dollars from the Health Care Financing Administration (HCFA) are not allowed to bill for services performed by residents. Teaching physicians and the departments they work for can bill only for those services that they *personally* perform and not for those that they merely supervise. (A "primary care exemption" loophole allows billing under very specific circumstances for services not personally performed by the teaching physician.) Many institutions are (like my own) fortunate enough to have "compliance officers" who protect academic medical centers from penalties that could accrue by making sure that only those services personally performed by the attendings are billed for and that such billings accurately reflect the service provided. Of course, since neither the compliance officers nor the FBI agents from whom they protect us have the time or inclination to actually observe what services attendings are personally performing while in teaching settings, the real issue comes down to "documentation," ie, compliance with recording the necessary number of "elements" of the history, physical, and medical decision-making to justify a particular Current Procedural Terminology (CPT) code. Indeed the medical record in most centers is no longer a document designed to communicate medical information from one member of the patient's care team to another for the benefit of the patient's health, but rather a document to prove that the attending has not cheated the third-party payer into paying for something done by a resident. Teaching attendings, who find themselves arguing with their compliance officer about whether they have personally "documented" enough elements in the note to justify a 99 213 or 214 instead of a 212 for the 1-year-old who might have had bronchiolitis or pneumonia but turns out to have a cold, may long for the days when they argued over the relative merits of the Problem-Oriented Medical Record compared with the "Source"-oriented one. What would Dr Weed[2] have to say about the "Compliance-Oriented Medical Record?"

So what *should* we be trying to teach future pediatricians while they work with us in continuity and acute care clinics? Ng and Lawless argue that "pediatricians in training need more preparation for the financial issues of practice management that they must face in the real world." But I wonder. Are we succeeding so well in making them experts in differentiating the child who is "sick" from the one who isn't; in counseling parents about their infant's sleep problems, temper tantrums, or risk of injuries; in recognizing which children or parents are depressed, victims of violence, or at risk for suicide, and in being

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competent care providers for children with special health care needs, that we can afford to give up some of that time and instead spend it teaching them how to correctly code a clinic visit? After being coached by coding experts, faculty physicians probably are in a position to teach a resident how to "game" the system. Reminding a resident that we actually did look at the skin of the child with a high fever and "teaching" her that recording the absence of a rash or petechiae counts as an "element" that must be counted in determining the correct code may be a legitimate function of a clinic attending. But given the time constraints of teaching in the clinic, what is our real mission: making sure that that she knows why examining the skin of a febrile child is an important thing to do or making sure that her coding is correct?

There was a time when an attending's signature below that of the resident meant that she had used her judgment in deciding which parts of the history, examination, or decision-making she should repeat. There was an assumption that the attending could actually be trusted to make this determination based on the clinical situation, how well she knew the patient, her appraisal of the resident's skills and knowledge, and that she took responsibility for everything that was done as well as the outcome. I don't know how good the evidence is that this trust has been widely abused in pediatric outpatient clinics, but we are now told that billing on this basis is fraudulent and can subject us to severe penalties or even criminal charges. You can bill only for what you document as having personally performed. (If you have attended compliance training and signed a form attesting that you have done so, your institution has no obligation to stand behind you or support you if your documentation is challenged by a payer.)

The additional time required for the actual personal (repetitious?) performance of the key elements of the history, physical, or decision-making as well as the documentation has to come from somewhere. I worry that it is coming from the time formerly devoted to teaching. Is this an improvement in medical education? Wouldn't it just be simpler all around if we did it all ourselves and let the residents watch? If we didn't have residents, would we need compliance officers?

Residents can't be expected to learn everything that they need to know to be competent pediatricians during their residency. Adult learning theory suggests that it is not until one perceives a need to know something that one is really able to learn it. Most residents (in contrast to first-year practitioners) seem to have little interest in learning about RVUs, APCs, correct coding, and the like. Perhaps learning that incorrect coding puts them at risk for the wrath of the compliance officer or the Office of the Inspector General (OIG) or, because they undercoded, the wrath of their practice manager or even that doing so could subject them to charges of "enticing" more children into their practices, is something that can wait until they actually are in the (God help them) real world.

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Uncertainty in the Management of Viral Lower Respiratory Tract Disease

ABBREVIATIONS. RSV, respiratory syncytial virus; ICU, intensive care unit; CAIV, cold-adapted, live-attenuated influenza vaccine.

Approximately 3% of children in the United States will be hospitalized in the first year of life because of a viral infection of the lower respiratory tract.[1,2] Viruses that account for the vast majority of hospitalizations resulting from pneumonia and bronchiolitis include respiratory syncytial virus (RSV), parainfluenza viruses (particularly type 3), influenza viruses, and adenoviruses. A recent report from the Centers for Disease Control and Prevention provides important information on the epidemiology of pediatric viral lower respiratory tract disease in the United States, estimating that 123 000 hospitalizations resulting from bronchiolitis occur each year in children in the first year of life.[1] During the 17 years covered in this report from 1980 to 1996, hospitalization rates for children <12 months with viral infection of the lower respiratory tract increased more than twofold. RSV alone accounts for 50% to 90% of bronchiolitis hospitalizations and 20% to 50% of pediatric hospitalizations for pneumonia. Approximately 500 RSV-associated deaths occur each year in the United States.[3] This mortality figure is lower than an estimate made in 1985 by the National Institute of Medicine, at least partly because of improvements in the management of hospitalized infants.[4] The annual cost of RSV hospitalization for infants in the United States is estimated to be in excess of $300 million to $400 million.[5]

Despite the fact that about 16% of hospital admissions for children in the first year of life are because of viral lower respiratory tract illness, there remains a remarkable lack of consensus on the optimal management of patients.[1,6] Viral infection of the lower airway is generally a self-limited condition. Nonethe-

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less, bronchiolitis is notorious for variation in disease expression based on a number of factors, including the presence of underlying heart or lung disease, gestational age, chronological age, and viral strain.[7] In this issue of *Pediatrics*, Willson et al[8] explore the intriguing issue of differences in resource utilization for infants hospitalized with viral lower respiratory infection at 10 geographically diverse children's medical centers and demonstrate a striking variation in practice patterns. One of the important contributions of this paper is the use of the Pediatric Component of the Comprehensive Severity Index to standardize comparison of patients with similar disease acuity at different institutions. Not surprisingly, hospital costs correlated strongly with intensity of intervention. However, the finding of an inverse correlation between disease severity on admission and institutional average costs suggests an opportunity for cost savings through reduction of inappropriate care without a compromise in quality of care.

Standardizing clinical practice by the use of guidelines-based education has been shown to reduce admissions, reduce resource utilization, and shorten length of stay for hospitalized infants with bronchiolitis without increasing readmission rates or decreasing family satisfaction.[9,10] Ongoing educational efforts can sustain changes in management over time.[11] A number of issues regarding optimal management of infants hospitalized with bronchiolitis are yet to be resolved with appropriate placebo-controlled trials. But the challenge raised in the report by Willson et al is to modify practice patterns based on what is currently known and to avoid costly interventions that do not alter the course of disease.[8]

What criteria form the basis for a decision to hospitalize an infant with bronchiolitis? Certain underlying diseases place an infant at increased risk of more severe disease. Congenital heart disease (particularly cardiac lesions associated with increased pulmonary blood flow or pulmonary artery hypertension), prematurity, season of birth, and the requirement for supplemental oxygen in an infant with chronic lung disease are well-recognized markers for more severe disease.[7] Other conditions may place a child at variable risk of severe disease and may lower the threshold for admission: neuromuscular disease, history of recurrent aspiration, congenital anomaly of the airway, familial dysautonomia, myasthenia gravis, Down syndrome, cystic fibrosis, and an immunodeficiency state. Variation in disease presentation as a function of geographic location has been described but is incompletely understood, perhaps reflecting differences in altitude or differences in air quality in certain urban cities.[12] A report from New York State described a fourfold variation in hospitalization rates for viral lower respiratory infections with the strongest predictor of admission being low socioeconomic status.[13] Other factors influencing a decision for admission may include reimbursement considerations, bed availability, and referral patterns.

Once admitted, what factors contribute to the dramatic differences in resource utilization noted in this study? Management strategies for children with viral infections of the lower respiratory tract are primarily supportive but the type and frequency of monitoring will add to hospital costs.[14–16] All infants hospitalized with lower respiratory tract disease require careful clinical assessment of respiratory status, including measurement of oxyhemoglobin saturation. Low concentrations of supplemental oxygen are generally sufficient to maintain adequate oxygen saturation. Monitoring of arterial carbon dioxide tension will be needed in a small number of infants. Adequate hydration is important and for tachypneic patients, parenteral therapy may be necessary. Judicious use of the laboratory to confirm a viral etiology by culture or by detection of RSV antigen in nasopharyngeal aspirates generally is appropriate to rule out a bacterial etiology. However, repeat testing of a patient already known to be infected or screening of an asymptomatic contact generally is not appropriate. Numerous studies have confirmed the importance of infection control policies in prevention of nosocomial viral infections.[17,18] A recent report from Children's Hospital of Philadelphia demonstrated that such policies are also cost-effective; each dollar spent on infection control saved an estimated $6 that would have been spent on nosocomial disease.[19] Patients who acquire nosocomial RSV disease are more likely to have underlying cardiopulmonary abnormalities than infants admitted with community acquired RSV disease and, therefore, more likely to have a complicated course. Failure to initiate isolation procedures in a timely manner may add to the cost of RSV disease.

A decision to admit to the intensive care unit (ICU) is generally based on the possible need for intubation because of progressive hypercarbia, increasing hypoxemia despite supplemental oxygen or apnea. Criteria for ICU admission will vary among physicians as noted in the study by Willson et al[8], which demonstrated a range in ICU utilization of 19% to 56%. Most infants admitted to the hospital with bronchiolitis or pneumonia will not have underlying disease that places them at increased risk of respiratory failure. Among otherwise healthy infants, ICU admission because of respiratory deterioration is an uncommon occurrence. In a study from Children's Hospital at Strong, only 1.8% of 542 previously healthy, full-term infants required transfer to ICU for evolving respiratory distress.[20] Some institutions lack a transitional care or step-down unit. This may add to hospital costs when a patient spends extra time in a more expensive ICU bed because of concern that an improving infant requires closer observation than can be provided on the ward.

Bronchodilator therapy is commonly used in the management of hospitalized infants with bronchiolitis although conclusive evidence of efficacy has not been demonstrated. More than 90% of the patients in the report by Willson et al[8] received this therapy. The results of most prospective, placebo-controlled trials with β-2-agonist inhalation fail to demonstrate a significant improvement in oxygen saturation, time to discharge, or reduction in wheezing.[21–23] Similarly, agents with α-adrenergic or anticholinergic activity

have not conclusively shown a beneficial effect in trials in hospitalized infants with bronchiolitis. Because of concern that reactive airway disease may be misdiagnosed as bronchiolitis, one approach is to assess results of bronchodilator therapy after the initial dose. Repeat doses of an inhaled bronchodilator are then continued only in the small number of infants with well-documented improvement in respiratory function soon after the first dose. Some studies report that inhalation bronchodilator therapy can cause paradoxical bronchospasm with worsening hypoxia.[24]

Placebo-controlled trials with corticosteroids have failed to demonstrate a beneficial effect on the course of bronchiolitis in hospitalized infants.[25,26] Nonetheless, in the study by Willson et al[8], between 8% and 61% of patients received corticosteroid therapy. There has been some interest in the possibility that simultaneous administration of an antiviral agent and an antiinflammatory agent such as a steroid might reduce the viral load and shorten the disease process, but there is insufficient clinical experience to support this approach at this time. There is a theoretical concern that steroid therapy during the acute stage of illness could result in higher viral titers and prolonged viral shedding.[27]

Ribavirin, a synthetic purine nucleoside, is the only antiviral agent which has been licensed by the Food and Drug Administration for the management of RSV bronchiolitis/pneumonia.[28,29] Early trials documented a modest antiviral effect from ribavirin as defined by a reduction in RSV titer in nasopharyngeal secretions relative to controls. However, it has proven more difficult to demonstrate clinically relevant benefit from ribavirin therapy. Well-conducted, placebo-controlled trials with ribavirin have failed to demonstrate a consistent difference between groups in terms of a requirement for mechanical ventilation, duration of stay in the pediatric ICU, or duration of hospitalization. In some placebo-controlled trials, a modest improvement in oxygen saturation has been reported in ribavirin recipients, but this is of uncertain clinical significance. Furthermore, concern has been expressed regarding the choice of placebo in these trials because both water and saline may induce bronchospasm in control patients, introducing bias in favor of ribavirin efficacy. It has been known for some time that infants hospitalized with severe lower respiratory tract disease resulting from RSV are at increased risk of recurrent episodes of wheezing, recurrent lower respiratory tract illness, and abnormal pulmonary function testing later in childhood.[30] Long-term follow-up studies of ribavirin-treated patients have been difficult to conduct in a rigorous fashion but they have not provided reproducible data to suggest ribavirin has an effect on pulmonary outcome.[31–33] Use of this antiviral agent has become difficult to justify because of the high cost of ribavirin therapy and inconsistent reports of efficacy.

Antibiotics are unlikely to have therapeutic value in a hospitalized patient with bronchiolitis. Nonetheless, many patients have blood cultures obtained and receive parenteral antibiotic therapy, particularly infants with an abnormal chest radiograph. Approximately 25% of infants will have radiographic evidence of atelectasis or consolidation consistent with a possible bacterial infection.[34] However, bacteremia or bacterial pneumonia in hospitalized infants with bronchiolitis is unusual. Otitis media occurs in infants with RSV bronchiolitis, but most patients can be treated orally if antibiotic therapy is necessary.[35]

In view of the burden placed on patients and on the health care system by viral infections of the lower respiratory tract, what future options may become available for control of viral disease? Despite >35 years of effort, it has proven difficult to develop a safe and effective RSV vaccine for young infants that produces protective immunity but does not enhance natural infection.[36] Subunit RSV vaccines consisting of surface glycoproteins F and G, which stimulate neutralizing antibody, seem to be safe and immunogenic in seropositive children as young as 12 months, although efficacy has not been determined. A live-attenuated, temperature-sensitive RSV vaccine for intranasal immunization that elicits both a local mucosal antibody response and systemic immunity is under development.[37] Temperature sensitive strains preferentially replicate at the lower temperature of the nasal cavity and less efficiently at core body temperature. Intranasal administration of the vaccine strain results in a subclinical infection that induces immunity by simulating a natural infection of the upper airway. To date, problems encountered with such attenuated vaccines include lack of genetic stability (back-mutation to virulence), overattenuation (does not induce immune response), and underattenuation (causes symptoms in vaccinee). Currently, the only option for prevention of RSV infection in high-risk infants is passive immunoprophylaxis with either a polyclonal hyperimmune globulin, Respigam, or preferentially, with a humanized murine monoclonal anti-F glycoprotein antibody preparation, palivizumab.[38] Both preparations have been shown to be safe and efficacious in large, well-designed clinical trials.[39] This intervention is restricted to a relatively select number of high-risk infants because of cost. Most infants hospitalized with RSV infection will not fall into a high-risk group and therefore will not satisfy the recommended guidelines for passive immunoprophylaxis. Only a relatively small number of total RSV hospitalizations will be prevented by targeting high-risk infants, although these infants are most likely to experience a complex hospital course.[40] To dramatically decrease the overall burden of disease and cost associated with RSV, a vaccine will be required.

Inactivated influenza vaccines for use in infants ≥6 months of age are currently recommended for high-risk infants and children.[41] A promising approach to influenza control is a cold-adapted, live-attenuated influenza vaccine (CAIV) administered via the intranasal route. Phase III studies suggest that CAIV containing 2 attenuated A and 1 B strains is >90% effective in children in preventing cases of influenza resulting from strains included in the vaccine and only slightly less effective against strains that demonstrate antigenic drift.[42,43] Hospitalization

rates in children <5 years resulting from influenza infection appear to be equivalent to those in adults over 50 years of age for whom the influenza vaccine is currently recommended.[41] The ease with which CAIV can be administered is likely to result in reconsideration of a recommendation for universal vaccination of children against influenza, once the vaccine is licensed.

After RSV, parainfluenza virus type 3 is the most common cause of hospitalization attributable to bronchiolitis and pneumonia in young children. Vaccines under consideration include subunit vaccines containing surface glycoproteins, as well as live-attenuated vaccines. An attenuated, intranasal parainfluenza 3 vaccine has been shown to be safe, immunogenic, and genetically stable in seropositive as well as seronegative infants as young as a few months of age.[44] A live, oral adenovirus vaccine consisting of serotypes 4 and 7 for use in military recruits was available for a number of years but has not been studied in civilians and is no longer being produced.[45]

Although most infants with bronchiolitis can be safely managed as outpatients, hospitalization rates for this illness seem to be increasing. Because the optimal course of management of hospitalized infants is not clear and because the course of illness is variable from patient to patient, differences in the use of various interventions and procedures is to be expected. Important advances in the management of patients with bronchiolitis have dramatically reduced mortality rates to <1% among most groups of hospitalized infants. The task now is to carefully define those interventions that are efficacious, those interventions that are unlikely to be effective, and those interventions that need evaluation in controlled clinical trials.

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Influenza Virus Continues to Pose New Challenges

During a typical influenza season, 20% to 30% of children are infected with influenza virus, with even higher rates during epidemic years. Influenza infection is frequently regarded as a self-limited illness in children. However, 2 recent articles highlight the morbidity associated with pediatric influenza infections in terms of respiratory hospitalizations, outpatient visits, and antibiotic prescriptions.[1–2] In this issue of *Pediatrics*, Chiu et al[3] demonstrate that influenza virus is associated with another significant morbidity in children—febrile seizures.

Using a comprehensive surveillance system at Queen Mary Hospital, Hong Kong, Chiu et al[3] compared the incidence of febrile seizures among children 6 months to 5 years of age hospitalized with influenza A infections to similar children hospitalized with parainfluenza or adenovirus infections. They reported that among children hospitalized with influenza A infections, 19.5% had febrile seizures. Among children admitted with parainfluenza virus or adenovirus infections, the incidence of febrile seizures was 12.2% and 9%, respectively. Children infected with influenza A virus not only had significantly higher rates of febrile seizures, but they also had higher rates of repeated seizures during the same illness than those infected with parainfluenza virus or adenovirus (odds ratio: 6.7 [95% confidence interval: 2–22.5]).[3]

Although these results are intriguing, additional studies on the association between influenza A and febrile seizures are warranted. Because the authors limited the study to febrile children infected with influenza A, parainfluenza, or adenovirus, they did not comprehensively examine all children with febrile seizures. Also, we do not know how these results compare with other viral etiologies, including influenza B, human herpesvirus-6, or human herpesvirus-7.[4] Because the study definitions of influenza A, parainfluenza, and adenovirus infections (culture or rapid antigen) were not clearly stated, it is difficult to assess the potential role of misclassification bias.

Although earlier studies had established a link between influenza infections and seizures, this study has provided the most definitive evidence to date. This retrospective cohort study was possible primarily because the emergency department has had a low threshold for admitting children with acute febrile illnesses and because comprehensive respiratory viral cultures and rapid viral diagnostic studies were routinely obtained on hospitalized children.[3] In this hospital, viral testing has been demonstrated to reduce costs by shortening hospital stays and by reducing antibiotic use.[5] We hope that the investigators will continue to use this large data set to further the understanding of pediatric viral infections. In the meantime, the rest of us will try to promote a similar approach in our medical centers.

What does this report tell us about the pathogenesis of influenza infections? Does the influenza virus have tropism for the central nervous system or does the increased rate of febrile seizures simply reflect the known tendency of influenza to induce high fever? The nearly twofold higher incidence of febrile seizures in children infected with influenza A, as compared with those infected with parainfluenza virus or adenovirus, persisted even after multivariate analysis adjusted for the peak temperature and duration of the fever.[3] So, the answer does not seem to lie with the magnitude of the fever.

Several reports of influenza-associated encephalopathy from Japan have suggested that the influenza virus may be targeting the brain. In 1995 Mizuguchi[6] reported "a new disease entity in Japan that manifested itself as acute encephalopathy after viral infection with influenza A, influenza B, or other viruses". The disorder predominantly affected children between 6 to 18 months of age living in Japan and Taiwan. Since the original description of influenza-associated encephalopathy, many additional cases have been reported.[7] Most of the described children developed encephalopathy within 2 days of the onset of influenza symptoms, and the first neurologic sign was generalized convulsions. The calculated incidence rate of influenza-associated encephalopathy in Japan has been between 7 and 12.8 cases per 100 000 children.[8,9] Many of the children with

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influenza-associated encephalopathy either died within a few days of disease onset or had long-term sequelae. The authors emphasized that this disease entity was not Reye's syndrome because the patients with influenza-associated encephalopathy had no history of aspirin intake, had rapid loss of consciousness, and coma ensued within 24 hours. In addition, reports of influenza-associated encephalopathy have indicated that bilateral thalamic lesions are often evident on neuroimaging.[10]

Although episodes of acute encephalopathy were reported in the influenza pandemics of 1918 and 1957, these early reports occurred primarily in adults and none manifested neurologic signs within the first 2 days of illness.[11,12] To our knowledge, similar reports of influenza-associated encephalopathy have not appeared in the Western literature. Whether it is unique to Japanese or Taiwanese children or whether genetic, environmental, or other unknown factors are responsible remains a mystery. As noted by Chiu et al,[13] the recent report of a novel amino acid substitution at the receptor-binding site of the hemagglutinin gene of influenza A that correlates with viral tropism is intriguing. Surveillance for influenza-associated encephalopathy is ongoing in the United States through a large multistate study funded by the Centers for Disease Control and Prevention. Whether this surveillance system will detect cases of influenza-associated encephalopathy remains to be determined.

A practical question remains. Given all of the pediatric influenza-related morbidity, including the reported associations with febrile seizures and encephalopathies, should young children routinely be immunized with the influenza vaccine? This question is hotly debated in the pediatric infectious disease community. In 1998 the Advisory Committee on Immunization Practices formed a working group to explore whether they should recommend annual influenza vaccination for young children without high-risk medical conditions. Recent studies indicate that influenza-attributable hospitalization rates in pediatrics are highest among young children and are comparable with rates seen in other high-risk groups, such as the elderly.[1,2] These findings persisted even after the authors accounted for the cocirculation of respiratory syncytial viruses. Many believe the time has come to recommend routine influenza immunization for all children <5 years of age. Consistent with this position, a decision analysis has predicted that routine influenza immunization of preschool children would be cost-effective.[14]

Some warn that the logistics of a wide-scale pediatric influenza immunization program would be too problematic and could not be implemented. For example, only a small percentage of the high-risk children recommended for yearly influenza vaccination actually receive vaccine.[15] However, a number of approaches have increased vaccine coverage levels for other childhood diseases, and for influenza vaccination in high-risk adults. Patient reminder systems, multicomponent educational interventions, standing orders, provider reminder and recall cues, and after-hours clinics for the delivery of vaccines are a few approaches that might be implemented.[16–18] Monitoring immunization rates at the local and national level, and providing feedback to providers is equally important.[16] In a study of children admitted to our pediatric hospital with febrile or respiratory symptoms during the influenza season, parents of high-risk children commonly cited the lack of knowledge or the lack of a physician recommendation for influenza vaccine as the main reason for not vaccinating their children.[19] "An ounce of prevention" still remains preferable to "a pound of cure."

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Sedation, Risk, and Safety: Do We Really Have Data at Last?

ABBREVIATIONS. AAP, American Academy of Pediatrics; EEG, electroencephalogram; JCAHO, Joint Commission on Accreditation of Healthcare Organizations.

The July 2001 issue of *Pediatrics* contained a commentary by Freeman entitled "The Risk of Sedation for Electroencephalograms: Data at Last."[1] This commentary, written to accompany a paper by Olson et al,[2] made several statements based on erroneous statistical inference. We are grateful to the editors of *Pediatrics* for granting us this forum in which to respond to that commentary. The great importance of safety for children who need sedation is clearly reflected in past publications of this journal, most notably the American Academy of Pediatrics (AAP) "Guidelines for Monitoring and Management of Pediatric Patients During and After Sedation for Diagnostic and Therapeutic Procedures."[3]

We, as did Dr Freeman, commend Olson and colleagues for demonstrating that sedation for electroencephalograms (EEGs) in children is usually not necessary when effective behavioral techniques are employed. Nevertheless, there are occasions when behavioral techniques are inadequate, as demonstrated by the 513 patients (18% of the total in this series) who received sedation with chloral hydrate and other drugs. Three of these patients developed oxygen desaturation to between 82% and 88%. This corresponds to a Pao_2 of about 45 to 55, a significant degree of hypoxemia. Although transient hypoxemia is unlikely to lead to long-term consequences, unrecognized or inadequately treated hypoxemia, especially when related to airway obstruction, may progress to more severe complications. It is to the investigators' credit that their compliance with the AAP guidelines for monitoring during sedation resulted in prompt recognition and treatment of the airway problems without sequelae. This ability to rescue from complications is strongly emphasized in the 2001 standards of the Joint Commission on Accreditation of Healthcare Organizations (JCAHO).[4]

Dr Freeman asserts that because these hypoxic episodes occurred in children with risks for airway problems, one cannot ascribe the desaturation episodes to the chloral hydrate. There is no evidence whatsoever to support this contention, nor to support the implication that because these children might have desaturation episodes during natural sleep that chloral hydrate poses no additional risk. Pharmacologically induced loss of consciousness is not the same as natural sleep. We contend that these events further emphasize the need for adherence to the monitoring guidelines, because not all children at risk are readily identified prospectively. Furthermore, a major requirement of the AAP guidelines is to obtain a careful history and physical examination with particular attention to the airway and issues of ventilatory control. Without requirements to follow such guidelines for presedation screening, the recognition of children at increased risk for sedation complications may not occur until airway obstruction ensues *after* the administration of sedation.

A classic article published in the *Journal of the American Medical Association* nearly 20 years ago was entitled "If Nothing Goes Wrong, is Everything All Right?"[5] Its authors discuss the inability to infer safety from investigations in which there were no complications. In Olson's study, in which 513 patients received sedation, it is not surprising that serious adverse outcomes were not detected—the incidence of these events is likely to be in the magnitude of <1 per 10 000. The Olson study clearly lacks the statistical power to draw any conclusions regarding safety. It seems that the authors, and Dr Freeman, fell into the trap of a type II statistical error. Dr Freeman dramatically compounds the erroneous conclusion that sedation is "safe" when he further questions whether "a qualified and credentialed individual is needed at whatever level of sedation or anesthesia is achieved, either intentionally or unintentionally." This statement, which belies all credulity, was not even a hypothesis advanced in the study in question. One wonders what the outcome might have been in the patients who developed airway obstruction, had properly trained and skilled nursing and medical personnel not been present to detect and intervene in those events. The study by Coté et al, which Freeman dismisses because there is no denominator, did not intend to describe the incidence of sedation complications, but rather analyzed the characteristics of certain practices common to those events.[6] In that study, practices similar to those that Dr Freeman seems to advocate were identified as having a high risk of complications. The suggestion that an EEG technician, with no medical training or certification, can be relied on to effectively recognize and treat airway obstruction, hypoventilation, and hypoxemia, or that a pulse oximeter is an effective substitute for a qualified and vigilant clinician is, we believe, a recipe for disaster.

It is important to comment on the authorship, intent, and implications of the "Guidelines for Monitoring and Management of Pediatric Patients During and After Sedation" promulgated by the Committee on Drugs of the AAP.[3] These guidelines were developed by a multidisciplinary group of pediatric spe-

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cialists, and included only 1 anesthesiologist out of the 18 authors listed on the document. These guidelines, and the 2001 JCAHO standards,[4] were not designed to increase cost or create roadblocks for clinical care. They were written to address the problem of iatrogenic injury and medical error by using a systems approach, in which high-risk techniques are identified and avoided, and replaced with alternative strategies designed to minimize risk while maximizing efficacy. In other industries, such as aviation and nuclear power, such an approach has effectively decreased error by orders of magnitude. A recent Institute of Medicine report has clearly made these goals a priority for the medical profession.[7] Last year's report, "To Err Is Human: Building a Safer Health Care System," indicated that medical errors are symptoms of a dysfunctional system—the majority of medical errors do not result from individual recklessness, but from basic flaws in the way a health system is organized.[8] Dr Freeman would have us continue to use dysfunctional systems that will eventually fail, resulting inevitably in death or other catastrophic outcomes. To begin this process, we must examine the acceptable rate of error in any system and "design in" safety mechanisms in our routine practices. Certainly, the issues of cost, reimbursement, and resource allocation are factors in establishing these systems, but patient safety must always remain the foremost objective.

The proposition advanced by Dr Freeman that anesthesiologists have a conflict of interest in developing or disseminating guidelines for the safe conduct of sedation is both insulting and unfounded. The Institute of Medicine report specifically acknowledged the specialty of anesthesiology as being a pioneer in the field of patient safety. The authors note that "anesthesia is an area in which very impressive improvements in safety have been made . . . anesthesiology has successfully reduced anesthesia mortality rates from 2 deaths per 10 000 anesthetics administered to 1 death per 200 000."[8] Pediatric anesthesiologists are pediatric specialists (indeed, many of us are also pediatricians) whose concern is the safety and welfare of pediatric patients. As anesthesiologists, we are recognized experts in the provision of sedation and the monitoring and care of infants and children who have received drugs that alter consciousness. We have no interest in providing anesthesia services to patients receiving EEGs, nor are we aware of any anesthesia service that does so routinely. Our objective is not any professional, financial, or personal gain from the use of safe techniques for caring for sedated infants and children; it is the desire to see that the inherent risks in those procedures have been minimized as much as possible. The adversarial role into which Dr Freeman appears to thrust us is one we vehemently reject—we are your colleagues, working toward the same goals of child health and advocacy.

We recognize that a commentary is by its nature a statement of opinion, but nevertheless believe that the commentary by Freeman reflects an outdated approach to sedation in children that is no longer "state of the art" or even acceptable. Continuing to do things in the way they have been done in the past does not necessarily serve the interests of children, who deserve better. Great progress has been made in the past 15 years in increasing the safety of sedation care for children, much of it spearheaded by the AAP, and often with the assistance of anesthesiologists who specialize in the care of children. This is not the time to reverse that trend.

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STEM CELLS HINT AT PROMISE FOR INBORN BRAIN DISEASES

"In further testimony to the medical promise of stem cells, biologists have found that human neural stem cells can become incorporated in a fetal monkey's brain and share in its development, suggesting a novel way of correcting inborn brain diseases.

The human stem cells were injected into the brains of monkeys in the womb, and helped not only to construct the monkeys' brains but also to form the reservoir of stem cells from which new brain cells are generated throughout adult life.

The experiment, being published electronically by *Science* magazine today (July 27, 2001), was performed by Dr Evan Y. Snyder, an expert on brain stem cells at Harvard Medical School; Dr Curt R. Freed of the University of Colorado, a researcher who uses fetal brain cells to treat Parkinson's disease; and their colleagues. . . . Dr Freed said [that] much groundwork had to be laid before any clinical studies could begin. For one thing, researchers must establish that enough stem cells can be delivered to make a therapeutic difference . . ."

Wade N. *New York Times.* July 27, 2001

Noted by JFL, MD

Uncertainty in the Management of Viral Lower Respiratory Tract Disease

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