Cat Scratch Disease Presenting With Peripheral Facial Nerve Paralysis

ABSTRACT. Acquired peripheral facial nerve paralysis is a relatively common disorder that affects both children and adults. The most frequent nontrauma-related etiologies in otherwise neurologically intact patients are idiopathic (Bell’s palsy) and infectious, which includes otitis media, herpes zoster, Lyme disease, herpes simplex virus, Epstein–Barr virus, and *Mycoplasma pneumoniae.*1–5

Cat scratch disease (CSD) is typically a subacute, regional lymphadenitis caused by *Bartonella henselae* that is seen in children and young adults. CSD most often has a benign, self-limited course. However, 11% of CSD patients may present atypically, most commonly with Perinard’s oculoglandular syndrome or acute encephalopathy.6–11

We present a child with the first reported case of acute facial nerve paralysis in serologically proven CSD with typical lymphadenitis.

ABBREVIATION. CSD, cat scratch disease.

CASE REPORT

A previously healthy 3-year-old boy was admitted to our hospital in October 1996 with a 2-day history of progressive left facial weakness. The child had been intermittently febrile at home (maximum 101.7°F rectal) for 4 days and a left axillary lymphadenitis was diagnosed by his physician 3 days before admission and treated with amoxicillin. There was a history of scratches to the left arm by a 4-month-old kitten 3 weeks before the child’s admission and to the right arm during the previous week. There had been no tick or other animal exposures. Interestingly, the patient’s cousin, who had been exposed to the same family of cats, was diagnosed with CSD (including osteolytic lesions) 1 year earlier. On examination, the patient was alert and verbal, although cranky. He was afebrile, with a respiratory rate of 20 breaths per minute and a pulse of 88 beats per minute. He weighed 15 kg (50% percentile) and exhibited normal development (cranial nerves, strength, deep tendon reflexes, and sensation) was normal. There was mild tympanosclerosis on the right side. The history of previous kitten scratches to the arm and the ipsilateral axillary lymphadenitis was consistent with typical CSD for this patient. However, the onset of facial nerve paralysis 1 day after the lymphadenitis presented was interesting and not expected as a typical manifestation of CSD. In the absence of trauma and with an otherwise normal neurologic and tympanic membrane examination, we considered other causes of acquired facial nerve paralysis in children. These included Lyme disease (an endemic Lyme disease area), Epstein–Barr virus, herpes simplex virus, *M pneumoniae,* and herpes zoster.1–5

We deemed it likely that the facial nerve palsy was related to the total clinical picture that included the fever and lymphadenitis. Ceftriaxone was begun pending the Lyme disease test results.

The recognized clinical manifestations of CSD continue to increase and include lymphadenitis, the oculoglandular syndrome, encephalopathy, endocarditis, bacillary angiomatosis and peliosis, skeletal lesions, transient maculopapular rash, erythema nodosum, hemolytic anemia, thrombocytopenia, atypical pneumonia, glomerulonephritis, and disseminated hepatic and splenic lesions.6–9 The most common neurologic manifestation of CSD is acute encephalopathy, which occurs in 2% to 3% of patients and is more common in adults than in children. Seizures, cerebellar ataxia, hemiparesis,

lymphocytes), hemoglobin of 11.3 g/dL, and a platelet count of 391,000/mm³. Serum alanine aminotransferase level was 30 U/L. Lumbar puncture revealed white blood cell count of 1/mm³, red blood cells 56/mm³, glucose 57 mg/dL, and protein 13 mg/dL.

The patient was treated initially with intravenous ceftriaxone (100 mg/kg per day) and oral trimethoprim–sulfamethoxazole (10 mg/kg per day). The next day, prednisone (2 mg/kg per day) was added.

Forty-eight hours after admission, the patient remained afebrile and was playful, with some improvement in the lymphadenitis and no change in the facial paralysis. Lyme disease and Epstein–Barr virus studies were negative, as were blood and cerebrospinal bacterial cultures. The patient was discharged home on trimethoprim–sulfamethoxazole and prednisone with a clinical diagnosis of CSD. Cat scratch (*B henselae*) serum serology later returned elevated at 1:256 (1:64 is considered positive). Cerebrospinal fluid polymerase chain reaction did not detect *B henselae* or *B quintana.* Lymph node biopsy or excision was not felt to be clinically indicated, thus, polymerase chain reaction studies and pathology on the lymph node were not performed.

Ten days after discharge, the skin lesions had resolved, and the lymphadenitis was decreased to 1 cm with no tenderness or erythema. The facial nerve palsy was significantly improved. Soon thereafter, it completely resolved.

COMMENT

The history of previous kitten scratches to the arm and the ipsilateral axillary lymphadenitis was quite consistent with typical CSD for this patient. However, the onset of facial nerve paralysis 1 day after the lymphadenitis presented was interesting and not expected as a typical manifestation of CSD. In the absence of trauma and with an otherwise normal neurologic and tympanic membrane examination, we considered other causes of acquired facial nerve paralysis in children. These included Lyme disease (an endemic Lyme disease area), Epstein–Barr virus, herpes simplex virus, *M pneumoniae,* and herpes zoster.1–5

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myelitis, hearing loss, sixth nerve palsy, and aphasia all have been associated with CSD encephalopathy. Up to 20% to 30% of patients with CSD encephalopathy may have cerebrospinal fluid pleocytosis. \textsuperscript{12,13} Recently, \textit{B. quintana} has been associated with central nervous system pathology. \textsuperscript{14}

Abnormal neurologic findings in the absence of encephalopathy in CSD are uncommon but may include neuroretinitis and peripheral neuritis of the extremities (pain, edema, and paresthesias). \textsuperscript{13} Facial nerve paralysis in proven CSD has not been clearly established in the literature. There is one case report of a 9-year-old boy with parotitis associated with a partial facial nerve paralysis of the marginal mandibular branch (off the cervicofacial branch) of the facial nerve that courses through the parotid gland. The diagnosis was based on a lymph node biopsy that was “consistent with a condition such as CSD,” coupled with the retrospective recall of cat contact without known scratches. \textsuperscript{15} A CSD case series focusing on CSD encephalopathy mentions facial nerve paralysis in two children, 18 months and 8 years of age, in association with CSD ocularoglandular syndrome, which includes conjunctivitis, conjunctival granuloma, and preauricular lymphadenitis. It is specifically mentioned that these cases were not studied as exhaustively as those with CSD encephalopathy and that the diagnosis was made clinically with no specific details given. \textsuperscript{13} The physical findings of the ocularoglandular syndrome or parotitis were not seen in our patient. We did not perform additional studies to eliminate neuroretinitis, but decreased visual acuity was not suspected by history or physical examination.

Given the history and clinical course of illness in this child, coupled with the strongly positive serology to \textit{B. henselae}, we are confident that all findings, including the facial nerve palsy, were attributable to CSD. The clinical criteria for diagnosing CSD may vary, and when atypical features are present that have broad differential diagnoses, it is important to have serologic confirmation when making this diagnosis. Although evidence for antibiotic efficacy in CSD is largely anecdotal, our decision to treat with trimethoprim–sulfamethoxazole was based on published reports and on our own experience with this antimicrobial agent. \textsuperscript{13,16}

We also prescribed a short course of oral steroids to possibly decrease the inflammation of the seventh cranial nerve.

Transient facial nerve paralysis is certainly not a common finding in CSD. With the concomitant skin findings, this case could be considered evidence of disseminated CSD. An alternative explanation for the facial nerve involvement is that in addition to kitten scratches to the arm resulting in the lymphadenitis, this child may have been scratched about the face and neck (thus the skin lesions on initial examination) or had had direct inoculation into his oral/nasal mucosa or conjunctiva with contiguous spread resulting in the facial nerve palsy.

Facial nerve paralysis has not been reported previously in typical CSD. Based on this case, we believe that CSD at least should be considered in a child with significant kitten exposure who presents with acquired facial nerve palsy even without lymphadenitis, parotitis, or ocularoglandular syndrome. The list of differential diagnoses of acquired facial nerve paralysis is quite lengthy, but it does not currently include CSD. We present this case to alert clinicians to this neurologic sequela for CSD. The wide spectrum of presentations in CSD should continue to keep pediatric caregivers ever-vigilant in considering the diagnosis of CSD and inquiring about exposure to cats.

\begin{figure}[ht]
\centering
\includegraphics[width=0.5\textwidth]{image}
\caption{Left facial nerve paralysis noted at admission to hospital.}
\end{figure}

\begin{thebibliography}{15}
\bibitem{2} Andersson J, Sterner G. A 16-month-old boy with infectious mononucleosis, parotitis, and a Bell’s palsy. \textit{Acta Paediatr Scand.} 1985;74:629–632
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**e1 ABSTRACT.** Control of Hyperbilirubinemia in Glucose-6-Phosphate Dehydrogenase-deficient Newborns Using an Inhibitor of Bilirubin Production, Sn-Mesoporphyrin. Timos Valaes, MD; George S. Drummond, PhD; and Attallah Kappas, MD. Background. Hyperbilirubinemia in newborns with glucose-6-phosphate dehydrogenase (G6PD) deficiency is a serious clinical problem because of the severity and unpredictability of its course. An innovative approach to this problem is suggested by previous experience with Sn-mesoporphyrin (SnMP), a potent inhibitor of bilirubin production, in moderating neonatal hyperbilirubinemia caused by ABO incompatibility, immaturity, and unspecified mechanisms.

**Objective.** To compare the effectiveness of the preventive and therapeutic uses of SnMP in ameliorating the course of bilirubinemia of G6PD-deficient neonates.

**Methods.** Neonates born at the Maternia Teratery Hospital, Athens, Greece, and found to be G6PD-deficient by cord blood testing were stratified by sex and gestational age (210–265 days and >265 days) and randomized in pairs to receive SnMP (6 μmol/kg birth weight, intramuscularly) either on the first day of life (preventive use) or if and when the plasma bilirubin concentration (PBC) level reached an age-specific threshold level for intervention (therapeutic use). In the case of failure of SnMP to control the rise of PBC levels, the protocol defined precisely the threshold PBC levels for switchover to phototherapy (PT) and, if necessary, exchange transfusion. PBC was measured daily until a declining value was obtained and the case was closed.

**Results.** A total of 86 G6PD-deficient neonates were randomized: 42 in the preventive arm and 44 in the therapeutic arm. Of the latter, 20 (45%) reached PBC levels requiring therapeutic intervention and thus received SnMP. Regardless of the trial arm, none of the 86 neonates required PT, whereas in a previous study in the same population, 33% of G6PD-deficient neonates required PT. In the intrapair sequential analysis, the favored arm was decided on the criterion of the age at closure of the case being shorter by at least 1 day. After plotting 30 untied pairs in the sequential analysis graph, the preventive use of SnMP proved to be the favored arm, and the trial was stopped. At this point, there were 2 unpaired neonates, 12 tied pairs, 22 pairs in which the preventive use of SnMP was favored and 8 pairs in which the therapeutic use of SnMP was favored. In the group analysis, infants in the preventive group, compared with those in the therapeutic group, had a lower maximum PBC level (8.2 ± 3.1 and 10.9 ± 2.8 mg/dL, respectively), which was reached at an earlier age (63.5 ± 34.8 and 82.2 ± 24.7 hours, respectively) as well as a lower closing PBC level (7.2 ± 2.9 and 9.6 ± 2.5 mg/dL, respectively) and an earlier age at closing (89.1 ± 35.6 and 110.8 ± 23.6 hours, respectively). Moreover, a PBC level of >6 mg/dL respectively), as well as a lower closing PBC level (7.2 ± 2.9 and 9.6 ± 2.5 mg/dL, respectively) and an earlier age at closing (89.1 ± 35.6 and 110.8 ± 23.6 hours, respectively). Moreover, a PBC level of >6 mg/dL

**Conclusions.** In G6PD-deficient neonates, a single dose of SnMP administered preventively or therapeutically entirely supplanted the need for PT to control hyperbilirubinemia. The preventive use of SnMP offers practical advantages in populations with a high enough prevalence of G6PD deficiency to justify cord blood screening. Pediatrics 1998;101(5). URL: http://www.pediatrics.org/cgi/content/full/101/5/e1; G6PD-deficiency, hyperbilirubinemia, heme oxygenase, Sn-mesoporphyrin, neonatal jaundice.

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**e2 ABSTRACT.** Effects of Exposure to Alcohol in Mother’s Milk on Infant Sleep. Julie A. Mennella, PhD, and Carolyn J. Gerrish, PhD. **Objective.** To test the hypothesis that exposure to alcohol in breast milk affects infants’ sleep and activity levels in the short term.

**Methods.** Thirteen lactating women and their infants were tested on 2 days, separated by an interval of 1 week. On each testing day, the mother expressed 100 mL of milk, while a small, computerized movement detector called an actigraph was placed on the infant’s left leg to monitor sleep and activity patterning. After the actigraph had been in place for ~15 minutes, the infants ingested their mother’s breast milk flavored with alcohol (32 mg) on one testing day and breast milk alone on the other. The infants’ behaviors were monitored for the next 3.5 hours.

**Results.** The infants spent significantly less time sleeping during the 3.5 hours after consuming the alcohol-flavored milk (78.2 minutes compared with 56.5 minutes after feeding alcohol in breast milk). This reduction was apparently attributable to a shortening in the longest sleeping bout (34.5 compared with 56.7 minutes for sleeping after breast milk alone) and the amount of time spent in active sleep (25.8 minutes compared with 44.2 minutes after breast milk alone); the decrease in active sleep was observed in all but 2 of the 13 infants tested.

**Conclusions.** Although the mechanisms underlying the reduction in sleep remain to be elucidated, this study shows that short-term exposure to small amounts of alcohol in breast milk produces distinctive changes in the infant’s sleep–wake patterning. Pediatrics 1998;101(5). URL: http://www.pediatrics.org/cgi/content/full/101/5/e2; alcohol, lactation, sleep, activity, development, infant behavior.

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**e3 ABSTRACT.** Adverse Effects of High-dose Vitamin A Supplements in Children Hospitalized With Pneumonia. Charles B. Stephensen, PhD; Luis Miguel Franchi, MD; Herminio Hernandez, MD; Miguel Campos, MD, PhD; Robert H. Gilman, MD, DTMH; and Jose O. Alvarez, PhD. **Objective.** To test the hypothesis that high-dose vitamin A supplements will enhance recovery of children hospitalized for the treatment of community-acquired pneumonia.

**Design.** We conducted a randomized, double-blind, placebo-controlled clinical trial of high-dose vitamin A supplements among children 3 months to 10 years of age (N = 95) admitted to hospital with community-acquired pneumonia in Lima, Peru. Children ≤1 year of age received 100 000 IU of water-miscible vitamin A on admission to the hospital and an additional 50 000 IU the next day. Children >1 year of age received 200 000 IU on admission and 100 000 IU the next day.

**Results.** Children receiving vitamin A (n = 48) had lower blood oxygen saturation (the mean difference on day 3 in hospital was 1.1%), higher prevalence rates of retractions (37% in the vitamin A group vs 15% in the placebo group on day 3), auscultatory evidence of consolidation (28% in the vitamin A group vs 17% in the placebo group on day 3), and were more likely to require supplemental oxygen (21% in the vitamin A group vs 8% in the placebo group on day 3) than children in the placebo group (n = 47). Adjustment for baseline severity of disease and nutritional status did not alter the association of vitamin A with increased clinical severity, although the difference in blood oxygen saturation was no longer statistically significant. No differences were seen in duration of hospitaliza-
tion or in chest x-ray changes 14 days after admission. No deaths occurred, and toxicity of vitamin A was not seen.

Conclusions. This study indicates that high-dose vitamin A supplements cause modest adverse effects in children recovering from pneumonia and should not be used therapeutically in such patients unless there is clinical evidence of vitamin A deficiency or concurrent measles infection. Pediatrics 1998;101(5). URL: http://www.pediatrics.org/cgi/content/full/101/5/e3; vitamin A, pneumonia, children, Peru, respiratory, lung, retinol.

e4 ABSTRACT. Attitudes of the Physician Membership of the Society for Adolescent Medicine Toward Medical Abortions for Adolescents. Nancy H. Miller, MD; David J. Miller, PhD; and Laura M. Pinkston Koenigs, MD. Objective. To document the practices and attitudes of the US physician members of the Society for Adolescent Medicine (SAM) regarding adolescent abortion and contraception, as well as physician willingness to prescribe medical abortion if approved by the Food and Drug Administration (FDA).

Design. Cross-sectional questionnaire survey.

Participants. The entire physician membership of SAM (N = 1001) was surveyed. A total of 713 physicians responded, with 668 usable surveys yielding an adjusted response rate of 70%.

Results. Of the respondents, 81% were trained as pediatricians; 58% had additional adolescent medicine training. Ninety-six percent prescribed contraception for adolescents should be available under some circumstances (96%) reported that abortion for pregnant adolescents should be given within 6 months of the missed opportunity (MO); 3) whether these vaccinations were age-appropriate according to the guidelines of the Advisory Committee on Immunization Practices; and 4) variables associated with MOs.

Design. Retrospective chart review with a nested retrospective cohort of children with MOs.

Setting. Two inner city practice settings in Milwaukee: a community health center and an academic continuity care practice.

Patients/Selection Procedure. A consecutive sample of 710 visits of inner city children ≤3 years of age with MOs, seen between January 1 and March 31, 1995. A VO was defined as any encounter when the child was vaccine-eligible according to Advisory Committee on Immunization Practices guidelines.

Results. MOs occurred in 47% (330/710) of the VO. Only 40% of the children with MOs received age-appropriate immunizations within 6 months; 30% received the vaccinations beyond the age-appropriate time. The remaining 30% either did not return or were not vaccinated on return. The variables significantly associated with MOs were 1) age: children with MOs were older than those without, with a mean age of 15.5 months vs 10.9 months; 2) minor febrile illness; 3) moderate/severe illness; 4) acute illness encounters; and 5) patient's being seen at the community health center. Only 15.5% of all MOs were justified by the presence of moderate/severe illness.

Conclusions. MOs are frequently missed in inner city children. Most of the MOs were not justified by the valid contraindication of moderate/severe illness. Sixty percent of the children with MOs did not receive age-appropriate immunizations within 6 months. These children are vulnerable to vaccine-preventable diseases such as measles and pertussis. Pediatrics 1998;101(5). URL: http://www.pediatrics.org/cgi/content/full/101/5/e5; immunization, vaccination, missed opportunities, children, pre-school.

e5 ABSTRACT. Do Missed Opportunities Stay Missed? A 6-Month Follow-up of Missed Vaccine Opportunities in Inner City Milwaukee Children. Svapna S. Sabnis, MD; Albert J. Pomeranz, MD; Patricia S. Lye, MD, MS; and Margaret M. Amateau, MD. Objectives. To determine 1) the frequency of missed vaccine opportunities (VOs) in inner city children ≤3 years of age; 2) whether the recommended vaccine(s) were given within 6 months of the missed opportunity (MO); 3) whether these vaccinations were age-appropriate according to the guidelines of the Advisory Committee on Immunization Practices; and 4) variables associated with MOs.

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e6 ABSTRACT. Anabolic Steroid Use by Male and Female Middle School Students. Avery D. Faigenbaum, EdD; Leonard D. Zaichkowsky, PhD; Douglas E. Gardner, MA; and Lyle J. Micheli, MD. Background. The prevalence of anabolic steroid use by high school and college students has been reported in the literature. However, rumors persist regarding the use of steroids by younger populations.

Objective. To assess the extent of steroid use by male and female middle school students and to explore their attitudes and perceptions about these drugs.

Methods. A confidential self-report questionnaire was administered to 466 male and 499 female students between 9 and 13 years of age (mean ± SD, 11.4 ± 0.9 years) in 5th, 6th, and 7th grades from four public middle schools in Massachusetts. The number of students reporting steroid use and differences between users’ and nonusers’ underlying attitudes and perceptions about these drugs were evaluated.
Results. The response rate was 82% (965/1175 eligible). Results indicated that 2.7% of all middle school students reported using steroids; 2.6% were males and 2.8% were females. When steroid users were compared with nonusers, 47% versus 43% thought that steroids make muscles bigger; 58% versus 31% thought that steroids make muscles stronger; 31% versus 11% thought that steroids improve athletic performance; 23% versus 13% thought that steroids make one look better; 23% versus 9% knew someone their own age who currently took steroids; 38% versus 4% were asked by someone to take steroids; 54% versus 91% thought that steroids were bad for them; and 35% versus 2% indicated that they would take steroids in the future. Additional analyses determined steroid user involvement in sports and activities.

Conclusion. The results of this study suggest that the problem of illicit steroid use extends to children and young adolescents and that a segment of this population is mindful of the potential physiologic effects of steroids. This information will be useful to pediatricians, sport authorities, and school teachers whose guidance will become increasingly more important as steroid educational interventions for male and female middle school students are developed. Pediatrics 1998;101(5). URL: http://www.pediatrics.org/cgi/content/full/101/5/66; anabolic androgenic steroid, drug abuse, risky behavior, children, adolescents.

e8 ABSTRACT. Self-reported Adherence, Management Behavior, and Barriers to Care After an Emergency Department Visit by Inner City Children With Asthma. Frederick E. Leickly, MD; Shari L. Wade, PhD; Ellen Crain, MD, PhD; Deanna Kruzon-Moran, MS; Elizabeth C. Wright, PhD; and Richard Evans III, MD, MPH. Objective. The inability to adhere to a prescribed therapeutic program for the treatment of a chronic disease may be responsible in part for continued disease activity. This problem may be more of an issue in the treatment of asthma, a common, potentially lethal chronic condition in which the lack of symptoms may be interpreted as remission. Adherence was one of the key areas of interest for the National Cooperative Inner-City Asthma Study. The focus of this study was to identify those issues reported by families that could adversely affect their adherence to an asthma care program. The identification of barriers to adherence could then form the basis of a successful intervention program. This study describes barriers to adherence, asthma management behavior, and self-reported adherence.

Methods. Patients presenting during an acute attack of asthma at an emergency department (ED) were recruited for this study. The medical record of the ED encounter was abstracted and compared with information that was obtained during a baseline interview 3 to 5 weeks later. During the baseline interview, parents were asked about health care behaviors related to adherence.

Results. There were 344 children 4 to 9 years of age living in inner city census tracts in the study. Four areas of adherence (medicine use, appointment-keeping, emergency actions, and asthma attack prevention) were investigated. The parental report of medications prescribed at the ED and the information on the abstracted ED report agreed 94.9% of the time for the β-agonists, 86.8% for steroids, and 69.4% for cromolyn. Among respondents, 85.4% of parents reported that they are able to follow the ED recommendations almost all of the time; side effects of medicines were a concern for 81.1% of caretakers who were adherent and for 89.5% of caretakers who were nonadherent. Doubts regarding the usefulness of medications occurred in 34.4% of those considered adherent and 54.2% who admitted nonadherence. Medications
were forgotten some of the time by 45.2% of the children, and 52.8% tried to get out of taking medicine. Appointments for follow-up care were kept by 69% of those given an appointment in the ED, by an estimated 60.0% of those who were told specifically to call for an appointment, and by an estimated 25.2% of those who were neither given an appointment nor told specifically to make one. Only one third of parents report that they were able to keep the child away from known asthma triggers nearly all of the time. Approximately half avoided allergens; however, only 37.5% reported avoidance of cigarette smoke. The use of preventive medicines occurred in 23.5%. Using a medicine and taking the child to a physician were reported as the first or second action during an acute attack of asthma by 72.1% of respondents.

Conclusions. Adherence to an asthma-management program involves a number of areas: medication, appointment-keeping, prevention, and applying an emergency plan of action. Barriers to adherence may exist in one or all four of these areas, leading to ineffective control of asthma. Recommendations are made for improving the patient-physician partnership to improve adherence. Pediatrics 1998;101(5). URL: http://www.pediatrics.org/cgi/content/full/101/5/e9; carnitine, valproate, valproic acid, epilepsy, liver dysfunction, hyperammonemia, lipid metabolism, Reye's syndrome, handicap, malnutrition.

e9 ABSTRACT. Valproate Therapy Does Not Deplete Carnitine Levels in Otherwise Healthy Children. Shinichi Hirose, MD; Akihisa Mitsuodome, MD; Sawat Yasumoto, MD; Atsushi Ogawa, MD; Yukiko Muta, BS; and Yasuko Tomoda, MD. Objective. To determine whether children with epilepsy undergoing valproate (VPA) antiepileptic therapy and who are otherwise healthy have a lower serum level of carnitine (CAR) and a higher plasma level of plasma ammonia than do normal children.

Methodology. A total of 45 children with epilepsy, 6.3 to 21.7 years of age, who were treated solely with VPA and were free of abnormal neurologic findings or nutritional problems were randomly selected (VPA-treated group). An age-matched control group (n = 45) was selected from subjects without epilepsy (control group). Total (T) and free (F) serum CAR, serum VPA concentration, and the plasma ammonia level were measured and analyzed.

Results. Serum VPA concentration exhibited a weak negative correlation with both T- (r = -0.34) and F-CAR (r = -0.41). The T-CAR levels were 55.7 ± 12.4 and 57.6 ± 12.1 mM, and the F-CAR levels 42.7 ± 9.9 and 44.4 ± 9.9 mM in the VPA-treated and control groups, respectively. Thus, there was no significant difference in T- or F-CAR levels between the VPA-treated and control groups. Plasma ammonia levels were the same in the two groups: 9.9 ± 6.2 and 9.4 ± 11.8 mM in the VPA-treated and control groups, respectively. There was no significant correlation between blood ammonia and either T- (r = +0.024) or F-CAR (r = -0.026).

Conclusion. Children on a regular diet ingest a sufficient amount of CAR that more than meets their daily CAR requirement. The level of neither T- nor F-CAR in patients with epilepsy and without severe neurologic or nutritional problems being treated with VPA appeared to be affected by VPA therapy. Because the blood CAR level depends on nutritional condition rather than on blood VPA concentration, CAR deficiency caused by VPA is not likely to occur in this population. The usefulness of supplementation of CAR for this type of patient with epilepsy, therefore, must be reevaluated carefully. Pediatrics 1998;101(5). URL: http://www.pediatrics.org/cgi/content/full/101/5/e9; carnitine, valproate, valproic acid, epilepsy, liver dysfunction, hyperammonemia, lipid metabolism, Reye's syndrome, handicap, malnutrition.

e10 ABSTRACT. Symptomatic Splenic Hamartoma: Case Report and Literature Review. Teresa C. Hayes, MD; Howard A. Britton, MD; E. Bruce Mewborn, MD; Dean A. Troyer, MD; Victor A. Saldivar, MD; and Irving A. Ratner, MD. An 11-year-old girl with low-grade fever, night sweats, thrombocytopenia, and an 8-year history of progressive splenomegaly underwent an elective splenectomy. Pathologic diagnosis was multiple splenic hamartoma. The patient’s symptoms resolved after the splenectomy. Since first described by Rokitansky in 1861, ~140 cases of splenic hamartoma have been described in the literature. Most of the splenic hamartomas were discovered incidentally. A minority of these lesions were associated with hematologic symptoms such as pancytopenia, anemia, and thrombocytopenia. Only 20 of the reported cases of splenic hamartoma occurred in pediatric patients. However, compared with the adult patients, nearly half of these cases in pediatric patients was associated with symptoms. Splenectomy and partial splenectomy have relieved these symptoms. With advances in imaging, splenic hamartomas are being discovered with increasing frequency. A multimodal radiologic work-up has enabled some cases of splenic hamartoma to be diagnosed preoperatively. Inclusion of this benign entity in the differential diagnoses of symptomatic splenomegaly in a pediatric patient is important in the preoperative management and counseling of the patient and family. In patients who have discrete lesions, consideration of this entity preoperatively may avoid total splenectomy. Pediatrics 1998;101(5). URL: http://www.pediatrics.org/cgi/content/full/101/5/e10; splenic hamartoma, pancytopenia, hypersplenism, splenomegaly, hemangiomas.

e11 ABSTRACT. School-age Follow-up of Prophylactic Versus Rescue Surfactant Trial: Pulmonary, Neurodevelopmental, and Educational Outcomes. Robert A. Sinkin, MD; Bonnie M. Kramer, PhD; Joan L. Merzbach, MSW; Gary J. Myers, MD; John G. Brooks, MD; Donna R. Palumbo, PhD; Christopher Cox, PhD; James W. Kendig, MD; Charles E. Mercier, MD; and Dale L. Phelps, MD. Background. Exogenous surfactant replacement has improved survival and reduced pulmonary complications of prematurity. Improved early outcomes for infants of <30 weeks' gestation treated with a strategy of prophylactic versus rescue surfactant, if needed, were demonstrated in a multicenter, randomized trial conducted between 1985 and 1988. We reevaluated a subset of survivors from this trial to determine the pulmonary and neurodevelopmental outcomes at school age.

Methods. At 4.5 to 8 years of age, all survivors from one of the three centers were located, and 96% were evaluated. The original randomization included stratification by center and followed an intention-to-treat methodology in assessing the efficacy of prophylactic versus rescue treatment with surfactant. The follow-up test battery included a health-assessment questionnaire, spirometry, 88% saturation test, neurologic examination, and the McCarthy Scales of Children’s Abilities (MSCA) and the
Conners’ Parent Rating Scale–48. Educational achievement was determined by school class placement and teachers’ reports of achievement.

Results. Of the 192 children originally enrolled, 154 survived. Evaluations were performed on 148 of these infants. An abnormal pulmonary history was found in 45 (30%) of the children: 16 (22%) in the prophylactic group and 29 (39%) in the rescue group. Formal pulmonary function was evaluated in 81 children; 29 (78%) in the prophylactic group and 33 (75%) in the rescue group were considered abnormal. No significant differences were found between the two groups on either cognitive or motor subscales of the MSCA, the Conners’ Parent Rating Scale–48, the neurologic examination, the education services received in school, or the teacher ratings of below-average academic performance. Intelligence scores measured on the MSCA were low–normal for both groups. Some level of educational assistance was being provided to 72 (49%) of the cohort studied, and both groups had below average educational performance and increased needs for educational assistance.

Conclusions. Prophylactic surfactant administration to infants of <30 weeks’ gestation was associated with fewer long-term clinical pulmonary complications than assignment to rescue administration. Formal pulmonary testing at school age did not reveal significant differences between treatment groups in those infants who could be tested. There also were no group differences found on neurologic, cognitive, behavioral, or educational assessments at school age. Pediatrics 1998; 101(5). URL: http://www.pediatrics.org/cgi/content/full/101/5/e12; follow-up, newborn, premature, surfactant.

e12 ABSTRACT. Predictors of Mortality From Fires in Young Children. Seth J. Scholer, MD, MPH; Gerald B. Hickson, MD; Edward F. Mitchel, Jr, MS; and Wayne A. Ray, PhD. Background. In the United States in 1994, fires claimed 3.75 lives per 100 000 child years and accounted for 17.3% of all injury deaths in children <5 years of age.

Objectives. To conduct a historical cohort study that uses maternal demographic characteristics to identify young children at high risk of fire-related deaths, thus defining appropriate targets for prevention programs.

Methods. The cohort consisted of children born to mothers who resided in the state of Tennessee between 1980 and 1995. Information was obtained by linking birth certificates, 1990 census data, and death certificates. Children were eligible for the study if they were <5 years of age at any time within the study period and if key study variables were present (99.2% of births).

Birth certificates provided information on maternal characteristics including age, race, education, previous live births, use of prenatal care, and residence (in standard metropolitan statistical area). Child characteristics included gender, gestational age, and birth type (singleton/multiple gestation). Neighborhood income was estimated by linking the mother’s address at the time of birth to the 1990 census (block group mean per capita income).

The study outcome was a fire resulting in at least one fatality (fatal fire event) during the study period, identified from death certificates (coded E880 through E889 in the International Classification of Diseases, 9th rev). We calculated the fatal fire event rate corresponding to each stratum of maternal/child characteristics. We assessed the independent association between each characteristic and the risk of a fatal fire event from a Poisson regression multivariate analysis.

Results. During the study period, 1 428 694 children contributed 5 415 213 child years to the cohort: there were 270 deaths from fire (4.99 deaths per 100 000 child years) and 231 fatal fire events. In the multivariate analysis, factors associated with greater than a threefold increase in fatal fire events included maternal education, age, and number of other children. Compared with children whose mothers had a college education, children whose mothers had less than a high school education had 19.4 times (95% confidence interval [CI], 2.6–142.4) an increased risk of a fatal fire event. Children whose mothers had more than two other children had 6.1 times (95% CI, 3.8–9.8) an increased risk of a fatal fire event compared with children whose mothers had no other children. Children of mothers <20 years of age had 3.9 times (95% CI, 2.2–7.1) an increased risk of a fatal fire event compared with children whose mothers were ≥30 years old. Although both maternal neighborhood income and race were associated strongly with increased rates of fatal fire events in the univariate analysis, this association did not persist in the multivariate analysis. Other factors that were associated with increased risk of fatal fire events in the multivariate analysis were male gender and having a mother who was unmarried or who had delayed prenatal care.

The three factors associated most strongly with fire mortality were combined to create a risk score based on maternal education (≥16 years, 0 points; 13 to 15 years, 1 point; 12 years, 2 points; <12 years, 3 points); age (≥30 years, 0 points; 25 to 29 years, 1 point; 20 to 24 years, 2 points; <20 years, 3 points); and number of other children (none, 0 points; one, 1 point; two, 2 points; three or more, 3 points). The lowest-risk group (score <3) included 19% of the population and had 0.19 fatal fire events per 100 000 child years. In contrast, highest-risk children (score >7) comprised 1.5% of the population and had 28.6 fatal fire events per 100 000 child years, 150 times higher than low-risk children. Children with risk scores >5 contributed 26% of child years but experienced 68% of all fatal fire events. If the fatal fire event rate for all children had been equal to that of the low-risk group (risk score <3), then 95% of deaths from fires would not have occurred.

Discussion. Maternal education, age, and number of other children had strong and independent associations with fire-related deaths among young children. Taken together, these factors defined a steep risk gradient, where children in the highest-risk group had a fire-related mortality rate that was 150 times that of the lowest-risk group. From a public health perspective, maternal factors clearly define children who would be good candidates for prevention programs. There is an urgent need to develop prevention programs that can be shown to reduce fire-related injury in high-risk children. Pediatrics 1998;101(5). URL: http://www.pediatrics.org/cgi/content/full/101/5/e12; wounds and injuries, fires, socioeconomic factors, risk factors.

e13 ABSTRACT. Cat Scratch Disease Presenting With Peripheral Facial Nerve Paralysis. Robert S. Walter, MD, and Stephen C. Eppes, MD. Acquired peripheral facial nerve paralysis is a relatively common disorder that af-
ffects both children and adults. The most frequent non-
trauma-related etiologies in otherwise neurologically in-
tact patients are idiopathic (Bell’s palsy) and infectious,
which includes otitis media, herpes zoster, Lyme disease,
herpes simplex virus, Epstein–Barr virus, and Myco-
plasma pneumoniae.1–5

Cat scratch disease (CSD) is typically a subacute, re-
gional lymphadenitis caused by Bartonella henselae that
is seen in children and young adults. CSD most often has
a benign, self-limited course. However, 11% of CSD
patients may present atypically, most commonly with
Perinaud’s oculoglandular syndrome or acute enceph-
aloathy.6–11

We present a child with the first reported case of acute
facial nerve paralysis in serologically proven CSD with
typical lymphadenitis.

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**ADDITION**

A sentence has been added to the American Academy of Pediatrics statement from the Committee on
pages, as article e13. The following should be added under the heading, “Propranolol,” after the dosage but
before the note that was initially published:

*Note: Some practitioners have used up to 0.15 to 0.25 mg/kg for the treatment of refractory infundibular spasm.*

The electronic version of this article will include links indicating this addition.
Cat Scratch Disease Presenting With Peripheral Facial Nerve Paralysis
Robert S. Walter and Stephen C. Eppes
*Pediatrics* 1998;101:e13
DOI: 10.1542/peds.101.5.e13

The online version of this article, along with updated information and services, is located on the World Wide Web at:
/content/101/5/e13.full.html
# Cat Scratch Disease Presenting With Peripheral Facial Nerve Paralysis

Robert S. Walter and Stephen C. Eppes

*Pediatrics* 1998;101:e13

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