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PEDIATRIC & ADOLESCENT MEDICINE REPORTS™

The essential guide to developments in primary care for infants, children, and adolescents

Hepatitis A: Foodborne Outbreaks in Michigan and Maine—Should we Recommend Immunization?

ABSTRACT & COMMENTARY

Synopsis: *Two hundred thirteen cases of hepatitis A in schoolchildren in Michigan were shown by hepatitis A genetic analysis to be transmitted by specific batches of frozen strawberries grown in Mexico and processed in California. These were widely distributed for school lunch programs. Apparently, sporadic cases in Maine and other states were subsequently linked to the same source.*

Sources: Hutin YJ, et al. A multistate, foodborne outbreak of hepatitis A. *N Engl J Med* 1999;340:595-602; Koff RS. The case for routine childhood vaccination against hepatitis A. *N Engl J Med* 1999;340:644-645.

Hutin and associates on the national hepatitis a investigation Team of the Centers for Disease Control and Prevention (CDC) investigated a large outbreak of cases of hepatitis A that occurred in February and March 1997. The cases occurred almost exclusively in schoolchildren and school employees in two counties in Michigan. There had been no cases of hepatitis A reported in these counties in the preceding year. Information was obtained concerning how often a subject ate school lunch and which food items were eaten during seven school days, beginning 32 days before the peak incidence of the disease. The same information was obtained from an equal number of randomly selected classmates who did not develop hepatitis A. During the same period, 39 cases were reported from Maine and similar dietary information was obtained. In both Michigan and Maine, there was a strong association between consumption of strawberry shortcake and the subsequent development of hepatitis A. Polymerase chain reaction analyses of viruses isolated from patients in Michigan and Maine revealed an identical sequence of RNA indicating that the same virus caused the disease. It was possible to trace the strawberries responsible for the Michigan outbreak. The same RNA sequences

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were also found in small numbers of cases that occurred in the same general period in several other states that had received the same batches of frozen strawberries. These berries had been grown in Mexico, processed and frozen in California, and distributed though the Department of Agriculture for school lunch programs. The source of contamination was found to be probably related to unsanitary field conditions in Mexico.

In an accompanying editorial, Koff points out the increasing vulnerability of individuals in the United States to hepatitis A infections because of a falling rate (30%) of naturally acquired immunity and the increasing importation of vegetables and fruits from less developed countries where hepatitis A is endemic. The Advisory Committee on Immunization Practices (ACIP) of the CDC has recommended hepatitis A immunizations for some high-risk groups, including persons traveling or working in underdeveloped countries where hepatitis A is endemic, homosexual males, patients with chronic liver disease, and Native and Alaskan Americans. However, universal immunization has not yet been advocated. Koff believes that the time is appropriate to institute universal hepatitis A immunization in U.S. children.

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COPY EDITORS: Michelle Moran, Neill Larmore, Holland Johnson.

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Questions & Comments

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COMMENT BY HAL B. JENSON, MD, FAAP

Although we have two highly effective and safe inactivated hepatitis A vaccines, they are not licensed for use in children younger than 2 years of age because of reduced efficacy. This age limit and the cost of yet another childhood vaccine have dampened enthusiasm for universal hepatitis A immunization. Hepatitis A infection most frequently occurs as sporadic cases but also occurs in the clusters and outbreaks such as these in Michigan and Maine described by Hutin et al that continue to occur and place a strain on public health departments. Physicians must provide postexposure immunoglobulin prophylaxis, and parents must take time from home and work to obtain medical care following exposure. These cases exemplify the dilemma we face to determine whether the currently available vaccines are appropriate for universal use.

In his accompanying editorial, Koff points out the increasing vulnerability of individuals in the United States to hepatitis A infections because of a falling rate (< 30%) of naturally acquired immunity, higher susceptibility of younger persons, and the increasing importation of vegetables and fruits from developing countries where hepatitis A is endemic and sanitation may be suboptimal. The ACIP has recommended routine hepatitis A immunization for persons traveling to or working in underdeveloped countries where hepatitis A is endemic, homosexual and bisexual males, persons with chronic liver disease, recipients of clotting factors, users of illicit drugs, as well as for children in high-risk communities such as Native and Alaskan Americans and children in selected areas of high hepatitis A endemicity. This approach has resulted in different hepatitis A vaccination recommendations in different areas. The state of Oklahoma recently enacted universal childhood hepatitis A immunization statewide for all children, and in the next year, Texas is implementing hepatitis A immunization for 32 counties along the Texas-Mexico border.

However, universal immunization for the entire country has not yet been recommended. Koff believes that the time is appropriate to institute universal hepatitis A immunization in U.S. children. These outbreaks demonstrate our vulnerability to global health problems despite never even leaving the confines of middle America. More and more of us, including children, are international travelers and would benefit from routine immunization in childhood.

We face barriers with hepatitis A vaccination that we have not let impede us with other vaccines. We instituted *Haemophilus influenzae* type b vaccination with the original polyribosyl ribitol phosphate (PRP) vaccine while the conjugate vaccines were being developed, to at least pro-

vide protection for all children 2 years of age and older. While combined hepatitis A and B vaccination beginning in infancy may be possible, the available hepatitis A vaccines are effective in children 2 years of age and older. We implemented universal hepatitis B vaccination in childhood even though hepatitis B, like hepatitis A, is predominantly a disease of adults and, therefore, the full public health benefit may take years or decades to realize. It is certainly appropriate to vaccinate early in life, against both hepatitis B and hepatitis A, to provide maximum protection. We have instituted universal vaccination against varicella, which, like hepatitis A, has a high rate of complete recovery but is associated with approximately 80-100 deaths annually in the United States.

We have the means by implementing universal hepatitis A vaccination to prevent much disease and even some deaths each year. However, it remains to be determined if we have the will and the determination. ❖

Detection of Congenital Cataracts

ABSTRACT & COMMENTARY

Synopsis: *A substantial proportion of children with congenital and infantile cataracts are not being detected by 3 months of age using currently recommended routine ocular examinations in the newborn period and again at 6-8 weeks of age. Dense cataracts must be treated surgically by 3 months of age to prevent visual loss.*

Source: Rahi JS, Dezateux C. National cross sectional study of detection of congenital and infantile cataract in the United Kingdom: Role of childhood screening and surveillance. *BMJ* 1999;318:362-365.

Recommendations concerning the ocular examination of newborns have been in place in the United Kingdom since the 1960s. These recommended inspections of the eyes of all newborns include an evaluation of the pupillary red reflex of all infants in the first week of life and then again at 6-8 weeks of age.

To determine the mode of detection and timing of ophthalmological assessment of a nationally represented group of children with congenital and infantile cataract, Rahi and Dezateux, members of the British Congenital Cataract Interest Group, conducted a study of 235 children born in the United Kingdom between October 1995 and September 1996 in whom a cataract was newly diagnosed. Thirty-five percent (83/235) were detected in the routine neonatal exam and 12% (30/235) were detected at the 6-8-week examination. Eighty-two children presented because of symptoms. In 91 cases, the child's caregiver had suspected an eye defect before the diagnosis of a cataract was made. Fifty-seven percent (137/235) of children had been assessed by an ophthalmologist by the age of 3 months, but one-third (78/235) were not examined by an ophthalmologist until after 1 year of age.

COMMENT BY RICHARD A. EHRENKRANZ, MD, FAAP

Although the definitive management of congenital cataracts is the province of a pediatric ophthalmologist, in most instances their recognition depends upon the pediatrician who performs the neonatal physical examination.

Congenital cataracts are recognized as an important, and potentially avoidable, cause of visual handicap throughout the world. There is a long list of conditions associated with cataracts in children, including infections, chromosomal disorders, and a large number of systemic conditions or metabolic disorders. The most common congenital cataracts are idiopathic. Regardless of the cause of the cataract, permanent visual loss can result from a dense, light-occluding, congenital cataract. Amblyopia is believed to result from light stimulus deprivation of the infantile retina, which prevents normal retinal images from forming and being transmitted to the visual cortex. It is believed that surgical treatment of dense congenital cataracts must take place within the first 3-4 months of life in order to prevent permanent blindness of the affected eye. Thus, early detection and referral to an ophthalmologist are of paramount importance.

The recommendations for neonatal ophthalmologic examination used in the United Kingdom at the time of this study are essentially the same as those followed in the United States.[1] Demonstration of a symmetrical red reflex in a newborn that is elicited by an ophthalmoscope, focused on the pupil from 12 in to 18 in, indicates that the passage of light though the cornea, lens, and vitreous is probably normal and especially indicates that the posterior eye structures are normal. Absence or asymmetry of the red reflex or dark spots should be a cause for concern. The chemical conjunctivitis and swelling evoked by prophylactic silver nitrate applications into the newborn's eyes may make it difficult to elicit a red reflex, but the increasing use of antibiotic eyedrops has made this much less of a problem. However, it is not uncommon to be unable to elicit an adequate red reflex—at least the first time around.

The results of Rahi and Dezateux indicate that currently recommended practices for the neonatal diagnosis of congenital cataracts fail to detect a significant number

of babies who are subsequently diagnosed as having cataracts, some of whom are permanently blinded. There is no reason to think that a similar situation is not also present in the United States. Unfortunately, their study was unable to tell in most instances whether failure to detect a congenital cataract was because an appropriate examination was not performed (an error of omission) or whether the exam was performed incorrectly (an error of commission). The short postnatal stays that are increasingly common today present a challenge to the pediatrician performing a complete neonatal examination, including the eyes. However, a successful assessment of the red reflex should be considered an essential part of the newborn exam. If it can't be successfully accomplished in the newborn nursery, it is certainly incumbent to do it in the first few weeks after birth. ❖

Reference

1. Committee on Practice and Ambulatory Medicine on Ophthalmology. American Academy of Pediatrics. Eye examination and vision screening in infants, children, and young adults. *Pediatrics* 1996;98:153-157.

Iron Supplementation and Psychomotor Performance

ABSTRACT & COMMENTARY

Synopsis: *Substitution of an iron-supplemented formula for an unsupplemented formula at 6-9 months of age in poor, inner-city children was associated with both improved hematological status and reduced the decline in psychomotor development in the second half of the first year of life.*

Source: Williams J, et al. Iron supplemented formula milk related to reduction in psychomotor decline in infants from inner city areas: Randomised study. *BMJ* 1999;318:693-697.

Williams and associates, from the university of Birmingham, United Kingdom, studied 100 5.7-8.6-month-old infants who were receiving non-iron-fortified cow milk formula because of the parents' choice. This was to determine the effect on hematological and psychomotor development of changing to an iron-supplemented formula. All children had a baseline developmental assessment using the Griffith scales as well as a hematologic assessment. They were then randomized to either continue non-iron-fortified formula or switch to an iron-supplemented formula. Developmental and hematologic assessments were repeated after 18 and 24 months. There were no significant differences in hemoglobin levels at the time of randomization, but after 18 months, 33% of the children receiving iron-fortified formula were anemic compared to only 2% of the iron-supplemented group. The two groups had Griffith general quotient scores that were not significantly different at randomization. Both groups showed a decline of test scores during the study, but the decline was significantly greater in the non-iron-supplemented formula group. By 24 months, the decrease in the mean scores of the unsupplemented formula group was 14.7, compared to 9.3 in the iron-supplemented group ($P < 0.02$).

COMMENT BY HOWARD A. PEARSON, MD, FAAP

Although I am not aware of a formal meta-analysis of the many articles relating psychomotor development and iron deficiency, most studies have concluded that there is a measurable effect on behavior and learning associated with iron deficiency and iron deficiency anemia in infancy, and that this can be improved, at least in the short term, with dietary iron supplementation. One of the nutritional triumphs of the last two decades has been the marked reduction of iron deficiency anemia in U.S. children,[1] and this improvement has generally been related to the increasing use of iron-fortified cow's milk infant formulas, often through the women, infants, and children (WIC) program for poor children. Despite these kinds of findings, there remains a reluctance on the part of some pediatricians concerning the routine use of iron-fortified infant formulas. The basis of this reluctance is really a little difficult for me to understand. There are many anecdotes that relate iron therapy and GI problems (pain, constipation, etc.). These symptoms are most frequent in women. A number of studies have shown no difference in GI symptoms of any sort in infants receiving iron-supplemented formula. I suspect that physician concern about GI intolerance to iron-fortified formulas is strongly age related. Older pediatricians are more likely to believe that iron-supplemented formulas are associated with GI symptoms and constipation. The younger generations of pediatricians, in my experience, usually prescribe an iron-fortified formula for infants who are not breast fed. In the study of Williams et al of poor, inner-city children in Birmingham, UK, the 33% prevalence of iron deficiency anemia in 18- to 24-month-old infants consuming non-iron-supplemented formulas is very similar to what was seen in poor, inner-city U.S. children before the widespread introduction of the WIC program in the early 1970s. The Williams study clearly corroborates that iron supplementation is effective in preventing iron deficiency anemia. The unique finding of the Williams study was its demonstration that iron supplementation was also associated with better (or at least less dete-

rioration of) psychomotor performance of treated children compared to nontreated children, suggesting a protective effect of iron treatment.

In another study where the effects of a novel form of iron supplementation were investigated, Adish and associates randomly divided a large group of Ethiopian infants into two groups.[2] In one group, a cast-iron cooking pot was provided to the family; the other group was given an aluminum cooking pot. After 12 months of follow-up, the children receiving food cooked in iron pots had significantly lower rates of anemia and better growth than children whose food was cooked in aluminum pots. This Ethiopian study may seem a bit exotic, but I well remember my observations in poor, rural children in Florida 30 years ago. I saw very little severe iron deficiency in that population, certainly much less than I encountered in New Haven, Conn, only a few years later. I also recall that in the rural homes of these poor children that I visited, almost invariably there was a large, black cast-iron pot on the wood stove, slowly cooking greens, a dietary staple. The bottom line is that infants need some form of dietary iron supplementation by 4-6 months of age to prevent progressive iron deficiency and its hematological and non-hematological consequences. ❖

References

1. Lozoff B, et al. Iron deficiency anemia and iron therapy effects on infant developmental test performance. *Pediatrics* 1987;79:981-995.
2. Adish AA, et al. Effect of consumption of food cooked in iron pots on iron status and growth of young children: A randomised trial. *Lancet* 1999;353:712-716.

Diagnostic Accuracy of Clinical Assessment of the Heart Murmur of Pulmonary Stenosis

ABSTRACT & COMMENTARY

Synopsis: *Although expert clinical examination is highly accurate for distinguishing pulmonary stenosis from non-pulmonary stenosis cardiac murmurs in pediatric patients, it is imperfect in assessing severity of stenosis.*

Source: Danford DA, et al. Pulmonary stenosis: Defect-specific diagnostic accuracy of heart murmurs in children. *J Pediatr* 1999;134:76-81.

In order to determine whether echocardiographic studies are necessary to confirm a diagnosis of pulmonary valve stenosis (PVS) made on the basis of physical examination by an expert pediatric cardiologist, Danford and associates enrolled 521 previously unevaluated patients who were referred for evaluation of a heart murmur. Sixty-two of these patients were ultimately proven by echocardiographic examination to have PVS (mild, 29; moderate, 27; and severe, 6). Specific difficulties in discriminating PVS from small ventriculoseptal defect (VSD) aortic valve disease, atrial septal defect, and innocent murmurs were identified. There was not a good ability to assess the severity of PVS or the presence of other cardiac defects on the basis of physical examination. Echocardiographic studies are indicated in children with a heart murmur suggestive of PVS.

COMMENT BY ALAN H. FRIEDMAN, MD, FAAP

This study is a well-constructed assessment of the ability of a clinical expert (in this case a board-certified pediatric cardiologist) to accurately diagnose pulmonary stenosis without using echocardiography. PS is one of the more commonly encountered cardiac pathologies leading to a heart murmur. The physical examination findings are usually marked by the auscultatory findings, which are typically distinctive: a systolic ejection-quality murmur at the upper left sternal border with variable radiation to the back. In addition, a systolic click is frequently appreciated. The echocardiogram is also a sensitive diagnostic tool for PS, and with the use of Doppler and color flow mapping techniques, detailed morphologic and hemodynamic information can be obtained. For example, Doppler tracings across the pulmonary valve can be used in the classification of PS in mild (< 25 mmHg) gradient across the valve, moderate (25 mmHg = valve gradient = 49 mmHg) or severe (= 50 mmHg valve gradient). Danford et al set out to determine the sensitivity and specificity of the expert clinical examination, presumably in part to determine if the relatively high cost of the echocardiographic study could be obviated.

Seven board-certified pediatric cardiologists enrolled a total of 521 consecutive, previously unevaluated pediatric patients who were referred because of a heart murmur in the study. The investigators prospectively recorded their clinical diagnosis and level of confidence in the diagnosis. When PS was suspected, the investigator committed to a pre-echo classification of mild to severe. Each subject then underwent a complete echocardiographic study with Doppler tracings.

The median age for all subjects was 2.87 years and there was a slight female predominance (52%). A total of 62 subjects (12%) were found to have PS, and of these, 29 (47%) had mild PS. The more severe the PS, the younger the patient tended to be at the time of first

evaluation. Sixteen (26%) of the PS subjects had additional defects found at echocardiography, including atrial septal defects, ventricular septal defects, aortic valve disease, patent ductus arteriosus, and double-outlet right ventricle, among others. These concomitant defects were not recognized prior to the echo. Of the 62 PS patients, 31 (50%) were thought to have PS as the sole pathology prior to echo, yielding a sensitivity of 50%. However, PS appeared on the differential diagnosis list of 14 additional subjects, where the cardiologist was less certain. Of the 459 subjects without PS, 15 were thought to have had PS by the pediatric cardiologist before the echocardiogram, yielding a specificity of nearly 97%.

Several interesting findings came to light when the ability of the cardiologist to accurately classify the PS severity based on the physical examination was examined. Of the four cases of severe PS in which the cardiologist included PS in the differential diagnosis, two were thought to be severe and two were thought to be mild. The majority of cases of mild PS and, for that matter, moderate PS were thought to be mild by the cardiologist when PS appeared on the pre-echo differential diagnosis list. Thus, there was not great success in differentiating whether PS was mild, moderate, or severe based on exam alone.

As Danford et al correctly point out, in order to conclude that echocardiography is not required to support or confirm a diagnosis made by a pediatric cardiologist, the expert examination: 1) must have a high sensitivity and specificity for the diagnosis; 2) errors resulting from reliance on the exam alone must not lead to harmful mismanagement; and 3) the severity of the disease must be graded accurately. It appears as if several of these criteria were not met in this study, most notably the inability to clinically grade the degree of PS. The cardiologists were good at recognizing that mild PS was mild, but much less accurate in discriminating moderate and severe PS from mild cases.

Other studies have shown that an expert clinical examination is effective in discriminating congenital heart disease from an innocent murmur. However, the results from this study suggest that recognizing concomitant heart disease in PS and distinguishing severe PS from mild PS can be difficult when the clinical examination is used in isolation. Thus, Danford et al conclude that an echocardiogram is justified and important in evaluating the child thought to have PS on the basis of physical examination. Incidentally, this study supports the published guidelines of the American Heart Association for the management of PS, which suggest that the echocardiogram provides vital information upon which plans for care and follow-up should be based.[1] ❖

Reference

1. Driscoll D, et al. Guidelines for evaluation and management of common congenital cardiac problems in infants, children, and adolescents. A statement for healthcare professionals form the Committee on Congenital Cardiac Defects of the Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation* 1994;90:2180-2188.

Special Feature

NSAIDs and Their Effects on Renal Function

By Thomas L. Kennedy, MD, FAAP

The use of nonsteroidal anti-inflammatory drugs (NSAIDs) in children has increased dramatically over the past few years. Ibuprofen is now routinely used as the antipyretic of choice by many pediatricians who feel it is more effective than acetaminophen. The NSAIDs class of drugs is quite diverse, technically includes salicylates, and now comprises more than 20 agents. Therefore, it is risky and unwise to make any generalizations for the entire group. However, the NSAIDs share in common the ability to inhibit cyclo oxygenase, an enzyme required for the synthesis of prostaglandins. It is this inhibition of prostaglandin synthesis that is responsible for both therapeutic and side effects. NSAIDs are generally safe and well tolerated by children. Most pediatricians are aware of the NSAIDs gastrointestinal effects, but fewer are aware of the potential for nephrotoxicity.[1] Although uncommon, adverse renal effects are varied; run the gamut from mild to severe; and include: 1) acute renal failure with or without oliguria; 2) chronic, insidious renal injury; 3) heavy proteinuria and the nephrotic syndrome; 4) tubulointerstitial nephritis; 5) disorders of salt and water metabolism; and 6) hyperkalemia.[2] Nevertheless, NSAID use in children is generally considered safe and well tolerated, particularly if dosed properly and given for short periods. For example, one report prospectively assessed renal function in a population of children admitted to the hospital with acute, febrile illnesses who received antipyretic therapy in one of three regimens: 12 mg/kg acetaminophen, 5 mg/kg/dose ibuprofen, or 10 mg/kg/dose ibuprofen.[3] The study found no evidence of increased renal impairment in the groups who received ibuprofen, even in those whose illnesses were associated with dehydration. However, individual increases in creatinine were not reported. Most reports of NSAIDs nephrotoxicity in children are single cases and the

role of NSAIDs is usually speculative. Renal injury has been encountered following large overdose and prolonged use but has also been reported with appropriately dosed, short-term therapy.

Because NSAIDs nephrotoxicity is the result of prostaglandin inhibition, it is important to appreciate the role of prostaglandins in the kidney. Under normal, euvolemic circumstances, there is little evidence that prostaglandins are either necessary or active in determining renal function. They are synthesized on demand and in response to specific stimuli and exert their effects only in close proximity to the site of synthesis. Thus, they may be considered autacoids, that is, substances whose action is local.

Renal prostaglandins are vasodilators, which, through their action on the afferent and efferent arterioles, help to preserve renal bloodflow and glomerular filtration rate (GFR), especially in the presence of hypovolemia and other conditions of underperfusion. Various renal prostaglandins (e.g., PGE2, PGF2I, and PGI2) are also active along different segments of the nephron, affecting solute and water transport. In the presence of vasoconstrictors such as catecholamines, angiotensin II, vasopressin, and endothelin, renal prostaglandin synthesis is stimulated in an attempt to preserve renal bloodflow and defend GFR. The vasoconstricting agents are commonly present in conditions with diminished effective arterial blood volume such as congestive heart failure, nephrotic syndrome, cirrhosis, severe hypertension, and sepsis. In these situations, renal prostaglandins are protective and oppose the intense vasoconstriction that could lead to renal ischemic injury. Use of NSAIDs in these circumstances will inhibit the synthesis of prostaglandins and could be potentially dangerous.

In certain situations (for example, chronic renal insufficiency) GFR may be chronically maintained or "propped up" by the action of renal prostaglandins, thus making the use of NSAIDs more risky. It has been reported in patients with sickle cell anemia, who routinely develop progressive evidence of renal dysfunction, that the baseline GFR (which frequently is normal to even greater than normal when assessed by creatinine clearance) is sustained by renal prostaglandin activity. Thus, decreases in GFR may be quite common with administration of NSAIDs.[4] A recent article describes the dramatic and unfortunate development of irreversible renal failure in an adolescent female with sickle cell disease who was receiving appropriate dose ketoralac therapy for the pain of a vaso-occlusive crisis.[5] This case was not associated with any of the recognized risk factors for NSAIDs nephrotoxicity such as hypovolemia. The conclusion that the renal failure was caused by ketorolac is speculative, although it was a reasonable diagnosis of exclusion. Simckes and colleagues recommend that children with sickle cell disease should have the doses of ketoralac adjusted to those administered to patients with renal insufficiency and even then that it should be used with care.

Renal prostaglandins also act to increase salt and water excretion. This natriuretic action makes sense physiologically since it helps to moderate the avid salt retention that occurs under conditions of hypovolemia and/or renal underperfusion and in the presence of activation of the renin-angiotensin-aldosterone system. Prostaglandin inhibition by NSAIDs predictably may lead to severe salt and water retention and the development of edema and/or hypertension. Furthermore, inhibition of renal prostaglandins may lead to unopposed action of antidiuretic hormone (there is normally a negative feedback of prostaglandin on ADH secretion), impaired free water excretion, and hyponatremia.

The final significant clinical effect of NSAIDs on renal function is the suppression of prostaglandin-dependent release of renin from the juxtaglomerular cells. The resulting hyporeninemic state leads to hypoaldosteronism and hyperkalemia, especially in patients with renal insufficiency.

The conclusion we should draw from all of this is that NSAIDs use in children is generally very safe, but that nephrotoxicity and renal-mediated adverse effects (e.g., hyperkalemia) are possibilities to consider, especially in certain high-risk groups. These include children with dehydration, hypovolemia, and other low-perfusion states; children with pre-existing renal compromise; and perhaps children with sickling hemoglobinopathies. ❖

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Brief Report

How Many Physical Exams are Necessary for Low-Risk Newborns?

Source: Glazener CM, et al. Neonatal examination and screening trial (NEST): A randomised, controlled, switchback trial of alternative policies for low risk infants. *BMJ* 1999; 318:627-631.

Although there is universal agreement that all newborn infants should be screened for physical abnormalities by a complete physical examination, there is little consensus or data concerning how frequent-ly this examination should be performed. The Aberdeen, Scotland, Maternity Hospital has had a longstanding policy of performing two neonatal physical examinations on low-risk newborns—one within 24 hours after birth and a second a few days later, prior to discharge. The need for and value of the second examination was questioned in the hospital. Glazener and colleagues carried out a two-year study that randomized low-risk newborns to receive either one or two neonatal physical examinations. Examinations were performed by hospital-based pediatricians, community-based medical officers, and senior pediatric house officers. Conditions likely to be detected by neonatal screening examinations were classified by the ICD-9 International Classification of Disease.

The one-examination group had a total of 4835 newborns, and the two-examination group consisted of 4877 newborns. Significantly fewer abnormalities were diagnosed in the hospital among the one-examination babies (8.3% vs 9.9%). The 1.6% difference had a confidence interval or difference of 0.3-2.7%. The larger number of abnormalities diagnosed at birth was primarily attributable to an excess in suspected hip abnormalities. This resulted in extra referrals to outpatient departments and orthopedic consultations. However, these extra visits did not result in more active management because similar numbers of infants in each group underwent splinting or operations. There was no difference in the number of heart abnormalities between the two groups.

Glazener et al conclude that there was no evidence of a net health gain from a policy of two hospital screening examinations. A single complete neonatal physical examination, combined with a repeat examination at 8 weeks of age, has similar results of detecting significant abnormalities as two neonatal examinations.

In the United States, most babies get only one examination in the hospital nursery because of short lengths of stays. This study should be of some reassurance that not many significant abnormalities are likely to be missed. —**rae**

CME Questions

22. Which of the following statements is true regarding hepatitis A infections?

a. They can be reliably prevented in children younger than 2 years of age with current vaccines.
b. Universal vaccination is currently advised in areas of high epidemicity for hepatitis A.
c. Specific viral strains responsible for local outbreaks cannot be identified with tests currently available.
d. They have a high rate of acute and long-term mortality.

23. Substitution of an iron-supplemented for an unsupplemented formula at 6-9 months of age to poor inner-city children:

a. has no effect on hematological performance.
b. is associated with an increase of gastrointestinal symptoms.
c. is associated with a lesser decrease in psychomotor performance than that in children continuing to receive unsupplemented formulas.
d. is nearly universally advocated by pediatricians.

24. The use of NSAIDs:

a. produces a measurable effect on kidney function in children with acute febrile illnesses.
b. may protect renal function in children with diminished blood volume.
c. may be used without concern in children with hemoglobinopathies or renal failure.
d. has no direct effect on prostaglandin synthesis.

25. Clinical assessment of a heart murmur that has the characteristics of pulmonary stenosis (PS):

a. is highly reliable in distinguishing PS from other cardiac lesions.
b. is highly reliable in assessing the severity of pulmonary stenosis.
c. does not require further assessment with ultrasonography and Doppler.
d. usually results in a cardiac diagnosis in addition to PS.

26. True statements about neonatal screening physical examinations include all of the following *except*:

a. a greater number of abnormalities are diagnosed when two vs. one neonatal exams are performed.
b. most of the difference between number of abnormalities detected are explained by hip abnormalities.
c. a greater number of cardiac abnormalities are detected when two examinations are done.
d. a larger number of hip abnormalities that require orthopedic interventions are found when two examinations are done.

In Future Issues:

Does Timing of PE Tube Placement Make a Difference?

Highchair Accidents