We continue to support the online Breastfeeding Friendly Consortium which is a partnership between the Virginia Department of Health (the main funder), the University of Virginia Department of Continuing Medical Education, and the Virginia Chapter of the AAP. The site contains 20 hours of continuing medical education, CERPs, and CEU’s at no cost to Virginia residents and at low cost for everyone else. It also has Maintenance of Certification activities for Part 2 and Part 4, also at low cost, and all related to breastfeeding. Check it out at: www.bfconsortium.org. To date, more than 40,000 individuals have completed the course. The curriculum has been used in all 50 states and 23 countries.

We have also been involved in other exciting activities! We were both presenters at the ILCA conference in Chicago, Natasha on a cultural panel, and Ann leading an Advanced Clinical Skills pre-conference workshop. Natasha is active in the Postpartum Support Virginia and speaks throughout the state on the subject and authored the ABM Clinical Protocol on the subject. Ann spoke at the Perinatal Conference in Roanoke about exclusive breastfeeding and the Ten Steps and is leading the revision of the ABM’s supplementation protocol. We both teach in the annual ABM What Every Physician Needs to Know About Breastfeeding which took place in Washington, D.C. in October.

Last but not least, the Virginia Coalition is working on a license plate!

Overall, things are hopping, but all in the right direction, and as they say in the South, we are “keepin’ on keepin’ on” spreading the word about the importance of breastfeeding and how healthcare providers in our state can support mothers and babies on their journey.
President’s • MESSAGE

Setting Course for a Bright Future

Begin with the end in mind is how Stephen Covey’s Best Selling book “7 Habits of Highly Effective People” begins. Covey argues to be successful one must know what they want to achieve before one can be successful attaining it. Of Covey’s seven habits “Begin with the End in Mind” is fundamental to his theory of what it takes to be successful. That is knowing what one wants to accomplish, starting with the final goal clearly in mind is crucial in planning to achieve it. This habit is important not only for an individual, but also for an organization such as ours.

Just like an individual, the chapter must know what it wants to achieve in order to maintain a concentrated effort. The chapter’s goal, the way I see it, is to be of value to its members so they can better provide healthcare to the children of the Commonwealth. This is achieved through active support and participation in four areas: Advancing healthcare for children; enhancing the education of pediatricians; maintaining a proactive advocacy; and fostering communications and interaction between pediatricians, their patients and payers.

In a broader sense, beginning with the end in mind means knowing where you’re at and where you’re coming from. Knowing what has been accomplished in the past and what is currently happening now creates a vision of the potential of what can be done in the Futures. Recognizing and honoring what others have accomplished, builds the sense what can be done. Two recent events that have impressed upon me how this is important to what we do in medicine, particularly what we do in pediatrics. As practicing pediatricians we set many examples. We are examples to our patients and their families. We are examples to the other areas of medicine. We are examples to the younger generations of pediatricians. This is demonstrated simply by what we do and by how we do it. This may not be something we often consider as we go about our daily lives. Our daily activities set the course of how pediatrics develops and is carry on into the future, sort of like setting the rudder of a boat. What the Virginia Chapter takes part in now leads to what it will be doing in the next five to ten years and longer. It is wonderful to imagine what we do today shapes where our chapter will be come 2026. As I recall the actions of previous chapter leaders addressing the issues when I first became involved in the chapter, it is easy to see why the VA-AAP is seen as a strong advocate of children and pediatricians. Under the leadership of previous chapter presidents and leaders, such as Tom Sullivan and Les Ellwood who concentrated on service and advocacy - their accomplishments directly influenced in what the chapter is currently doing.

So where are we now? From the present perspective, the future looks bright. The efforts of pediatricians are being recognized by others outside of pediatrics. At the MSV Annual Meeting this year, two pediatricians were among the six honorees recognized by The Medical Society of Virginia, for their exceptional service to medicine and their patients. The two were Dr. Robin Foster and Dr. Bob Gunther.

So where are we heading? In the last past two years under the leadership of Dr. Barbara Kahler, the chapter has been awarded several grants to enable the chapter to participate in studies that will impact how the practice of pediatrics is done. These grants support studies looking at a broad range of issues. One of the first grants studies, the practical use and guidelines of telemedicine in the pediatric office is being completed. Another study examines how to better incorporate the evidence-based recommendations of the Bright Future for well child visits and preventative screening. Other grants support the examination of how to improve HPV vaccination rates and how to improve the mental health care follow-up on children in foster care. Most recently the Chapter was chosen to be the recipient of a grant from the CDC through the national AAP to investigate how to improve appropriate antibiotic use for children. Powered by these grants and forward-thinking studies the chapter is emerging as a leader in advancing practical pediatric practice knowledge.

Anyone who has ever attended any of the pediatric conference in the state, the AAP NCE or the MSV Annual Meeting sees the many young physicians and residents just beginning their careers. These are future leaders of medicine and pediatrics who will be taking the chapter into the mid-21st century. They will follow the inspired directions set by those such as Barbara Kahler, Bob Gunther and Robin Foster taking the chapter to an even higher level of vital service. The physicians who are just beginning their careers are seeing what they can work toward what they can do for their profession and what pediatrics can mean for them. The achievements of our predecessors and the current leaders are not an end point at all, but rather a guiding light for a brighter future.

Respectfully,
Samuel T. Bartle, MD, FAAP President Virginia Chapter | American Academy of Pediatrics
VIRGINIA • PEDIATRICS

News You Can Use…

Senator Emmett Hanger Receives 2016 VA-AAP Child Advocate Award

Drs. Sam Bartle and Robert Gunther presented Senator Emmett Hanger with the VA-AAP Child Advocate Award during the Donald W. Lewis Pediatric Update 2016 on Saturday, September 17, 2016 in Williamsburg. The Virginia Chapter, AAP applauds Senator Hanger on his support of mental health and Medicaid funding, of decreasing tobacco use in the state, and of preventing obesity in children has been important and valuable for our patients and pediatricians in Virginia.

MSV Annual Meeting Update
Sandy, Chung, MD, FAAP
VA-AAP Vice President

Medical Society of Virginia held their annual meeting in Roanoke from October 14-16, 2016. Several resolutions proposed by physicians around the state were considered, discussed, debated and voted on by the MSV House of Delegates.

Some of the resolutions adopted included the following:

1.) Supporting the AMA’s recently amended policy calling for an end to recertification examinations,

2.) Supporting efforts to prevent physician burnout and suicide,

3.) Helping to protect Peer Review Committee processes,

4.) Supporting efforts to educate the medical community on and strengthen public health infrastructure to respond to health effects of climate change,

5.) Creating a group to study the problem of drug/opioid abuse among physicians and healthcare providers,

6.) Supporting legislative efforts to improve communication of visits from telemedicine providers to primary care providers, and

7.) Supporting efforts to improve physician training on end of life care and encouraging universal usage of advance care plans.

The Virginia Chapter, AAP held its annual Pediatric Breakfast during the 2016 MSV Annual Meeting where pediatrician delegates and the chapter lobbyist discuss current legislative issues. The Virginia Chapter continues to be an advocate and voice for the health of Virginia’s children and for the profession of pediatrics.
Childhood Sleep Disorders for the General Pediatrician: Are We Screening Effectively?

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Sleep problems affecting children are common, occurring at a rate of 37%, according to one study. Despite that surprising statistic, many pediatricians have had limited formal training in the diagnosis and treatment of many common sleep disorders. Sleep problems can be divided into respiratory and non-respiratory disorders. Sleep-disordered breathing (SDB) consists of both obstructive sleep apnea (OSA) and primary snoring, with prevalence rates of 1% to 5% for OSA and 5% to 12% for habitual snoring. Less commonly occurring respiratory disorders include central sleep apnea and sleep-related hypoventilation. Of the pediatric non-respiratory sleep disorders, behavioral insomnia of childhood is the most common, affecting 20% to 30% of infants and toddlers. Primary insomnia most often occurs in adolescents and children with developmental disabilities and has a prevalence of 5% to 20%. The other non-respiratory sleep disorders include circadian rhythm abnormalities, parasomnias (5% to 35% prevalence), restless legs syndrome, periodic limb movement disorder (combined prevalence of 2% to 8%) and central disorders of daytime sleepiness, i.e. narcolepsy and idiopathic hypersomnia (pediatric prevalence not well-established). Despite these known prevalence rates, research from one large pediatric primary care network showed that only 3.7% of patients received a sleep disorder diagnosis. In that study, the overall rate of both OSA and snoring combined was near 1%, suggesting under-recognition and under-diagnosis of sleep disorders by pediatricians.

Identification and treatment of sleep disorders is crucial, due to both short and long-term sequelae if left untreated. Nearly all studies examining the relationship between sleep-disordered breathing and cognition have discovered deficits in cognition or neuropsychological function in association with SDB. These deficits may include problems with learning, memory, visuospatial skills, language, verbal fluency, concept formation, analytic thinking, school performance, and executive functions. Behavioral abnormalities include hyperactivity, attention-deficit/hyperactivity disorder, daytime sleepiness, depression, and aggression. Improvements in learning and behavior may occur following treatment of OSA; however, these may be only partially reversible, perhaps if left untreated for too long. Cardiovascular effects of OSA in children include both left and right ventricular dysfunction. OSA is associated with increased left ventricular mass in children and, furthermore, improvements in right ventricular diastolic function have been shown to occur following adenotonsillectomy in children with OSA. Additionally, several studies have demonstrated that childhood OSA can affect autonomic regulation, brain oxygenation and cerebral blood flow, suggesting that OSA may put these patients at risk for cardiovascular health problems in the long term.

Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome, a 2012 clinical practice guideline published by the American Academy of Pediatrics, defines obstructive sleep apnea syndrome as a “disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction (obstructive apnea) that disrupts normal ventilation during sleep and normal sleep patterns.” This disorder of breathing must also be accompanied by symptoms or signs such as gasping or observed apneas, sleep enuresis, morning headaches, daytime sleepiness, attention-deficit/hyperactivity disorder, tonsillar hypertrophy, overweight, high-arched palate, failure to thrive, or hypertension, among others. The same guideline recommends that, as part of routine health maintenance visits, clinicians should ask whether a child or adolescent snores. If the patient snores on a regular basis (defined as at least 3 nights a week) and has any of the additional complaints or findings described, then a sleep study or polysomnogram (PSG) should be performed, or referral should be made to a sleep specialist or otolaryngologist for further evaluation. The American Academy of Sleep Medicine agrees that a PSG should be performed when the clinical assessment suggests a diagnosis of OSA. Once a diagnosis is established, the gold-standard for treatment in children with OSA is adenotonsillectomy, recognizing that other treatments exist and that surgery may not be indicated for all patients. The PSG helps to stratify severity and identifies children at risk for complications of surgery, persistence of OSA after surgery, and long-term sequelae of OSA. Even those with mild OSA on pre-operative PSG should have a repeat PSG performed if symptoms persist after surgery. Those with moderate to severe OSA on initial PSG should automatically have a repeat study performed to ensure resolution of sleep-disordered breathing, as even in the absence of symptoms, these patients are at higher risk of residual OSA following surgery.

There are also several non-respiratory indications for PSG in children. PSG may be indicated when there is clinical suspicion for periodic limb movement disorder (PLMD) or restless legs syndrome (RLS). A common pediatric sleep pitfall is confusion between PLMD and RLS, which are not the same entity. PLMD can only be diagnosed on PSG, as it requires a certain frequency of distinctive limb movements, combined with some clinical sequelae. RLS is instead a clinical diagnosis for which the finding of those limb movements can support, but not establish, a diagnosis. In addition, those with frequent parasomnias (sleepwalking, sleep terrors, etc.), epilepsy, nocturnal enuresis with a clinical suspicion of OSA or PLMD, or those with atypical or injurious parasomnias...
should also have a sleep study performed. Finally, an overnight PSG with multiple sleep latency test (a structured nap protocol) the following day is required for patients with suspected narcolepsy or for evaluation of excessive daytime sleepiness\(^1\).

At the VCU Center for Sleep Medicine, office visits are available for infant and pediatric patients referred for suspected sleep disorders with either a child neurologist, pediatric pulmonologist, or neurodevelopmental pediatrician, all board-certified in sleep medicine. Sleep studies are performed overnight at the Center for children aged 2 and up. Newborns up to age 2, and any patients with special needs (requirement for a lift for transfers, specialized nursing services, or tracheostomy and ventilator dependence requiring a ventilator titration PSG) have their studies performed under close observation in the hospital at VCU Medical Center. A dually-trained registered respiratory therapist (RRT) and registered polysomnographic technologist (RPSGT) is on staff and available to perform those more complex studies in the hospital. Our entire staff is well-trained in performing sleep studies on young patients and those with developmental delays such as Down syndrome and autism. In addition to non-surgical management of OSA, we care for infants and children with tracheostomy and ventilator dependence, as well as those who require non-invasive ventilatory support during sleep. We also diagnose and manage the full spectrum of non-respiratory sleep disorders. Our entire team of providers, nurses, office staff and sleep technologists welcome the opportunity to work with you to care for your patients.

References
Children's Hospital of The King's Daughters and the American Academy of Pediatrics, Virginia Chapter

Present

VIRGINIA PEDIATRICS NEWSLETTER
American Academy of Pediatrics – Virginia Chapter

Continuing Medical Education
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None.
Laparoscopic Gastroesophageal Dissociation in Neurologically Impaired Children with Recurrent Gastroesophageal Reflux Disease

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Gastroesophageal reflux is often seen in infants and children; however, it only causes problems in a small percentage of patients. Neurologically impaired children have a higher incidence of GER that is associated with various problems such as esophagitis and/or aspiration pneumonia. Many of these patients can be treated with acid suppression medication and/or reflux precautions. However, a subset of those children will fail these measures and need an antireflux procedure for their gastroesophageal reflux disease (GERD). Unfortunately, this patient population has a relatively high rate of recurrent reflux and loosening of their fundoplication. While a redo laparoscopic fundoplication is often possible, recent studies in Europe have questioned repeatedly performing an operation in this patient population that has failed at least once.

One operation that has been proposed as an alternative to redo fundoplication, particularly in neurologically impaired patients, is a gastroesophageal dissociation (GED), which can permanently cure GERD. Since it was first introduced by Dr. Adrian Bianchi from Manchester, England in 1997, several additional studies have demonstrated that it has relatively low morbidity and mortality, reduces the reoperation rate, as well as improves the quality of life (QOL) of the patient and caregiver. After the stomach is dissociated from the esophagus, intestinal continuity is restored with a Roux-en-Y esophagojunostomy and a jejunostomy, see Figures 1 and 2. While most patients are then fed via a gastrostomy tube with bolus feeds, patients can still take up to full feeds by mouth. In addition, most patients can be taken off acid suppression as acid exposure to the esophagus is permanently eliminated. Furthermore, medications can also be easily given into the gastrostomy tube.

Over the last 5 years, we have performed 20 laparoscopic gastroesophageal dissociations (LGED) in neurologically impaired children with excellent results. The patients have ranged from 14 months to 17 years of age and all had severe, recurrent GERD. All but two had undergone at least one fundoplication (max = 3). Patients weighed 7.9 to 57 kg (avg = 23.6 kg), length of stay ranged from 5–20 days (avg = 12 days), and estimated blood loss ranged from <5 cc to 450 cc (avg = 69 cc, median = 25 cc). Morbidity and mortality from the operation is relatively similar to our experience with redo laparoscopic fundoplication and typical results from other expert centers. There have only been 3 readmissions in this cohort of patients, one for a dislodged feeding tube and two with viral illnesses. Every patient had resolution of their GERD (follow-up avg = 11.5 months, max = 5 years) with minimal to no retching or vomiting. There was a decrease in healthcare visits for aspiration and respiratory illnesses (rated 5/5 from all 13/19 survey respondents), as well as improvements in perceived QOL of the patient (avg = 4.3/5) and caregiver (avg = 4.6/5).

In neurologically impaired patients with recurrent GERD, relying on long-term gastrojejunalostomy feeds or redoing the fundoplication often leads to other problems. Patients routinely have difficulty with gastrojejunalostomy tubes becoming displaced or clogged as well as the fact that feeds have to be given in a continuous manner. Furthermore, patients will often continue to have acid reflux that can expose them to cancerous changes of their esophagus over time as well as cause significant pain despite acid suppression. Also, a redo fundoplication is at high risk for failing again and can be associated with retching. After having a LGED, our cohort of patients had resolution of their retching and vomiting, a reduction in readmissions for reflux-related respiratory illness, and improved patient and caregiver QOL. These results mirror those seen and published in Europe. While it is a more challenging operation and associated with potentially significant complications, the morbidity and mortality is relatively low and is the only operation that will permanently cure GERD. Our retrospective study suggests that in neurologically impaired patients with severe, recurrent GERD, a LGED may be a viable alternative to traditional treatments. However, we have an ongoing prospective study that will further evaluate these patients and compare them to those who undergo a laparoscopic redo fundoplication. Hopefully, these results will help determine the best surgical option for this challenging patient population.

Objective: The reader will be able to review and highlight the potential role of Laparoscopic Gastroesophageal dissociation in recurrent GERD in neurologically impaired children.

ACGME Competencies: Medical Knowledge

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Poverty in Virginia’s Children
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The American Academy of Pediatrics (AAP) recently issued a policy statement that declared child poverty in the United States to be “unacceptable and detrimental to the health and well-being of children.”1 Across Virginia, poverty continues to be a tremendous problem for children. Fifteen percent of Virginia’s children live in households with annual income ≤100% of the federal poverty level (FPL; $24,300 for a family of four).2 Thirty-three percent of children live in “poor, near poor, or low income” (incomes up to 200% of the FPL) and 7% of families live in “deep poverty” (incomes up to 50% of the FPL).2

Poverty is not evenly distributed throughout the population. In Richmond’s poorest census tract the annual median family income is $10,000, while in the wealthiest census tract it is more than $175,000.3 Poorer neighborhoods tend to have greater levels of stress and violence, fewer safe parks for outdoor play, fewer grocery stores that sell affordable fresh fruit and vegetables, and higher levels of lead and airborne contaminants.

Poverty has been associated with low birth weight, infant mortality, language delay, chronic illness and injury. Indeed, colleagues at the VCU Center on Society and Health found a 20-year life expectancy difference between two nearby neighborhoods in Richmond (Figure 1).4 In collaboration with this Center, researchers at the Children’s Hospital of Richmond are working hard to understand how poverty affects child health and how we can best address health disparities that affect our community.

Examples of what you can do to help:
1. Vote for candidates and advocate for policies that help poor families.
2. Learn about resources for poor families in your community.
3. Screen for social determinants of health like housing and food insecurity in your clinic.
4. Make appropriate referrals for home visitation programs, medical-legal partnerships, Early Intervention and Head Start.
5. Promote early literacy through Reach Out and Read.

References
Despite the election year, kratom has been in the news recently.1 The Drug Enforcement Agency (DEA) was considering classifying kratom as a schedule 1 drug, but this action has been placed on hold. The DEA is allowing additional public comment until December 1st, when a final decision will be made. Currently unscheduled, kratom has many supporters who have managed to stall any scheduling because of the 1) reported therapeutic benefits and 2) the ease of performing research on an unregulated substance. Regardless of the legal status of kratom, some patients will continue to use and abuse it. It is helpful for pediatricians to stay informed about novel herbal agents in order to help their patients make efficacious and safe choices, recognize toxicity and offer appropriate treatment.

Kratom is plant material from the *Mitragyna speciosa* tree which grows in Southeast Asia. It is used for its stimulant (at low doses) and sedative/analgesic properties at higher doses. 2 The active alkaloids mitragynine and 7-hydroxymitragynine act on the opioid delta and mu receptors, causing euphoria, central nervous system and potentially respiratory depression. In the United States it has been listed as a drug of concern by the DEA and an emerging drug of abuse by the National Institute on Drug Abuse (NIDA).3 4 Kratom, like many other herbal products, has not been scientifically proven to treat any conditions as required by the FDA.

Naturally because of its properties, kratom is used as a drug of abuse, a treatment for opioid withdrawal and dependence, and for analgesia. From 2010 to 2015 calls to US Poison Centers about kratom increased from 26 to 263, a 100 fold increase.5 The youngest age was 2 months old.

Last month, the Virginia Poison Center at Children’s Hospital of Richmond, at Virginia Commonwealth University, had a call from a neonatologist reporting a 1 day old male with opioid neonatal abstinence syndrome. His mother had been taking kratom during her pregnancy. The child was treated supportively with benzodiazepines and morphine. Expectedly, the most common pediatric exposures are in teens. Two exemplary pediatric cases since 2010 were a 17 year old male taking kratom along with a muscle relaxant for recreation, who presented with confusion and hallucinations; and a 15 year old female who presented with bizarre behavior and sinus tachycardia after drinking a kratom tea. She was treated with intravenous fluids and benzodiazepines and recovered within 2-3 hours. However, there are cases of serious toxicity and fatalities in adults using kratom.6 7 Both patients were found deceased.

Anecdotally, kratom has therapeutic benefits, but clearly there are serious health risks. In this time of an escalating opioid epidemic, the pediatric population has already encountered kratom. In a case of respiratory depression, it is logical that naloxone can be used as a first-line agent in treatment. The Virginia Poison Center and the Children’s Hospital of Richmond, at Virginia Commonwealth University are available to assist at any time with questions on the diagnosis and treatment of kratom exposures.

References


Image credit: Mitragyna speciosa leaves By Uomo vitruviano - Own work, CC BY-SA 3.0, https://commons.wikimedia.org/w/index.php?curid=12196426
Primary care pediatricians frequently encounter anemia, neutropenia, or thrombocytopenia must diagnose the condition quickly and accurately. Those cytopenias may be acute or chronic, benign or malignant. One serious set of diseases that must be considered are the bone marrow failure syndromes, which may be inherited or acquired. Because some of the inherited bone marrow failure syndromes are associated with predisposition to leukemia or solid tumors, surveillance is critical and management complex. This article reviews the features that will help pediatricians differentiate potential bone marrow failure syndromes from other causes of cytopenia(s), as well as discuss the additional testing required to confirm specific disorders. To help pediatricians counsel families, the article also presents a general approach to management of bone marrow failure disorders in specialty care and options for genetic counseling.

**Bone marrow failure syndromes**

A wide range of primary and secondary causes can markedly reduce the number of circulating blood cells. More common are syndromic. Acquired bone marrow failure syndromes include aplastic anemia and myelodysplastic syndromes. Epidemiologic or registry databases suggest that collectively, primary bone marrow failure and myelodysplastic syndromes are more common than pediatric acute myeloid leukemia, which occurs with an incidence of ~7 cases per 1 million children/year.

Inherited bone marrow failure syndromes are caused by mutations in a growing list of genes. Most cases of pediatric acquired aplastic anemia in the United States are triggered by a poorly-defined environmental, infectious, or drug exposure; however, they are thought to result from immune-mediated destruction of blood stem cells. Pediatric myelodysplastic syndromes (many of which are termed refractory cytopenia of childhood) are a heterogeneous group of disorders. Some overlap with the inherited bone marrow failure syndromes, some are secondary to genotoxic damage, and others are truly de novo.

**Hematopoiesis simplified.**

The continuous production of highly specialized blood cells requires carefully regulated processes of stem cell renewal and differentiation. Less than a dozen hematopoietic growth factors drive these processes. Some act on multiple cell lineages, others on specific lineages (for example, erythropoietin and red blood cells, thrombopoietin and platelets, and granulocyte colony-stimulating factor and neutrophils).

A healthy bone marrow microenvironment provides physical and cellular support for the blood stem cells. Circulating blood cells have different life spans: lymphocytes ~ years, erythrocytes ~120 days, platelets ~ 7 days, and granulocytes ~2 days. The blood system must quickly respond to stress of hypoxia, bleeding, or infection by increasing erythrocytes, platelets, or neutrophils. If the available number of blood components is not sufficient for normal function, the problems arises, such as fatigue, bleeding,
When to suspect a bone marrow failure disorder

A physiologic buffer in the numbers of these blood components exists before signs or symptoms arise from a critical shortage: hemoglobin < 8 g/dl, neutrophils < 500/ul, and platelets <20,000/ul. Signs and symptoms include pallor, loss of energy or appetite, petechiae or bleeding, and fevers. Physical findings of both pallor and petechiae suggest a complex blood disorder, although a cause may ultimately be found to be benign and temporary (e.g. viral infection).

The history and physical examination remain essential. Age at presentation and presence of skeletal defects (such as short stature or thumb abnormalities), or abnormalities in skin pigmentation constitute important characteristics to raise the specter of an inherited bone marrow failure syndrome.

Most of the single lineage inherited bone marrow failure syndromes present during infancy: Diamond-Blackfan anemia, severe congenital neutropenia (Kostmann syndrome), and congenital amegakaryocytic thrombocytopenia. The multi-lineage bone marrow failure syndromes, such as Fanconi anemia, dyskeratosis congenita, and acquired aplastic anemia, present later in childhood.

Other historical clues that point to bone marrow failure syndromes include family history of blood disorders or cancers arising in early adulthood, low birth weight or failure to thrive, infections, and gastrointestinal problems. Exposure to drugs or exotic travel may point to acquired bone marrow failure.

A careful visual examination will illuminate a bone marrow failure syndrome. The patient may have skin with areas of hyper- or hypopigmentation, eczematoid rash, thumb or digit defects, poor formation of the teeth or nails, leukoplakia, and facial anomalies.

Developmental defects involving the heart, lungs, endocrine glands, or genitourinary tract are difficult to detect on examination and require either laboratory or imaging studies. As the differential diagnosis for a child with signs and symptoms of pancytopenia includes malignant diseases, lymphadenopathy and/or hepatosplenomegaly point to a malignant or even more infrequently metabolic storage disease.

Routine laboratory studies may be the first to suggest a bone marrow failure syndrome. A complete blood count reveals much information. One often-overlooked value is the mean corpuscular volume (MCV). In the presence of cytopenias, a high MCV suggests a stem cell disorder, while a normal MCV is consistent with acquired anemia or viral-mediated suppression. A small platelet volume is found in Wiskott-Aldrich syndrome, an immunodeficiency associated with thrombocytopenia. An examination of the blood smear might reveal dysplastic nucleus or abnormal cytoplasmic granules in leukocytes, which help to define the myelodysplastic syndromes.

The vast majority of single lineage cytopenias occurring outside infancy are self-limiting and may be due to infection, drug exposure, or be immune-mediated. Transient erythroblastopenia of childhood can be readily distinguished from Diamond-Blackfan anemia, if the affected child is older than 1 year and lacks congenital anomalies and macrocytosis. The presence of jaundice or dark urine indicates acute hemolytic crisis. Laboratory tests to diagnose a hemolytic anemia include positive antiglobulin tests (Coombs) and elevated values for lactate dehydrogenase, total and indirect bilirubin, and positive urinalysis. Immune-mediated thrombocytopenia (ITP) occurs in an otherwise healthy child and typically presents with severe thrombocytopenia (<10,000/ul).

Closing in on a diagnosis

If history, physical examination, and testing point to a bone marrow failure syndrome, additional testing will be required to establish an accurate diagnosis. A bone marrow aspirate and biopsy must be performed and be read by an experienced hematopathologist. Bone marrow specimens must also be analyzed for chromosomal breakage studies, cytogenetics, and immunophenotyping.

Abnormal alignment of chromosomes following their chemically induced breakage identifies most patients with Fanconi anemia. If the result is negative but suspicion remains high, then skin fibroblasts are obtained for chromosomal breakage studies.

Peripheral blood is analyzed for telomere length. Telomeres are the “cap” at the ends of chromosomes. A complex of proteins, DNA, and RNA control chromosome length. Shortening of the telomeres occurs as the cell ages, and in particular, shortened telomeres provide a marker for dyskeratosis congenita. To support a diagnosis of Shwachman-Diamond syndrome, serum trypsinogen, or pancreatic amylase can be studied.

Increasingly, next generation sequencing or gene set panels are being used to confirm the diagnosis. Most but not all of the etiologic genes have been identified. Fanconi anemia can be caused by 1 of at least 20 genes; at least 6 different genes each cause dyskeratosis congenita, Diamond-Blackfan anemia and severe congenital neutropenia.

The genes that cause the inherited bone marrow failure syndromes are quite variable. DNA repair pathways are affected in Fanconi anemia; whereas molecules controlling telomere maintenance are defective in dyskeratosis congenita. Genes encoding proteins that make up the ribosome, an organelle involved in protein synthesis, are mutated in Diamond-Blackfan syndrome. Severe congenital neutropenia can result from mutations in a neutrophil elastase, a transcription factor Gfi-1, the receptor for G-CSF, a mitochondrial protein Hax-1, a catalytic component of glucose-6-phosphatase, and WASP (the same gene that when mutated causes Wiskott-Aldrich syndrome). Not surprisingly, mutations in the receptor for thrombopoietin are associated with congenital amegakaryocytic thrombocytopenia. The precise function for the SBDS gene mutated in almost all cases of Shwachman-Diamond syndrome remains elusive. Increasingly being recognized as a inherited bone marrow failure syndrome is GATA2 deficiency, GATA2 is an important transcription factor for hematopoiesis. Affected children may have problems with warts and other infections, lymphedema, and myelodysplasia with abnormalities in chromosome 7.

Genetic counseling

Identification of the genetic basis for these disorders affords genetic counseling. Fanconi anemia is typically autosomal recessive. Dyskeratosis congenita and severe congenital neutropenia can be autosomal recessive, autosomal dominant.. Diamond-Blackfan anemia is autosomal dominant. Shwach-
man-Diamond syndrome is autosomal recessive. Some forms of these disorders may be X-linked.

**Need for multi-disciplinary care**

Establishing the precise diagnosis of an inherited or acquired bone marrow failure syndrome is critical for determining effective management of the pediatric patient. Hematology/Oncology treatment ranges from close monitoring to allogeneic stem cell transplantation. Because gastrointestinal, pulmonary, cardiac, neurocognitive, psychological, and endocrine disorders arise in children and adolescents with inherited bone marrow failure syndromes, pediatric specialists must be involved in their care. Pediatric surgical services may also be needed. Excellent laboratory services are also required for optimal care. These services may be found in only a few specialized children’s hospitals such as Children’s Hospital of Richmond, where I have organized a Bone Marrow Failure and Myelodysplastic Syndrome Clinic.

For patients with severe congenital neutropenia or Shwachman-Diamond syndrome, filgrastim should be administered to maintain an absolute neutrophil count greater than 500/ul. Transfusion support, along with a trial of prednisone, underlies the treatment of Diamond-Blackfan anemia. However, chronic use of transfusions and prednisone are associated with severe side effects. Toxicity also occurs with the use of androgens in Fanconi anemia.

Any bone marrow failure disorder may need stem cell transplantation, which provides a cure for marrow failure, but carries side effects of graft-versus-host disease, hormonal insufficiency, and infections associated with an immunocompromised host. Also, stem cell transplantation will not improve non-hematologic effects and may contribute to the development of secondary cancers.

Because the inherited bone marrow failure syndromes and myelodysplastic syndromes are associated with transformation to acute myeloid leukemia, frequent monitoring of peripheral blood counts is necessary, and in some cases, annual bone marrow aspirate examinations. Patients with Fanconi anemia, dyskeratosis congenita, and Diamond-Blackfan syndrome can develop epithelial cancers (particularly squamous cell cancers of the oropharynx and vulva) and osteosarcoma.

**Conclusions**

The key issue in diagnosing a cytopenia is to determine whether it is a self-limiting or a chronic disorder. An accurate diagnosis of the type of bone marrow failure syndrome is important in order to counsel families on the natural history of the disease and provide effective treatment. Management may range from monitoring to supportive transfusions to stem cell transplantation, and involves multiple specialties. The recent discoveries of the genetic causes for the inherited bone marrow failure syndromes permit genetic counseling and family planning.

**Further Reading**

Zika Virus: A Primer for Pediatricians

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In October 2015, what almost seemed to be a nuisance epidemic suddenly took an unexpected and devastating turn when an upsurge of infants born to mothers infected with Zika virus were noted to have marked microcephaly and other malformations. Over the ensuing months, what was initially noted as a possible association has since been confirmed to be causal; this has raised significant concern and drawn increased attention to Zika virus infections.

What is Zika virus?
The Zika virus was first recognized in 1934 in primates in the eastern hemisphere, and soon thereafter it was found to also infect humans. Thereafter the virus remained largely isolated to this area however in February 2014 it was detected in the western hemisphere. Zika virus is an RNA virus member of the Flavivirus family of vector-borne viruses, closely related to others in this family including Dengue, Chikungunya and West Nile. Because of this, Zika virus often demonstrates symptoms similar to these other viruses.

How is Zika virus spread?
Though the most common means of viral transmission is via mosquitoes, it is now well-known that infection may also occur via sexual contact, vertical transmission and transfusions as well. Though it has been detected in breast milk via sensitive PCR, it has not been demonstrated to be transmitted this way so mothers infected with Zika are still encouraged to breastfeed.

Where in the world is Zika virus found?
Although travel-associated infections (individuals returning with infection after travel to endemic areas) have been found in almost every country, local Zika virus transmission has been documented in all of Latin America, the Caribbean, and some of the Pacific islands. Local transmission has also been seen in the United States including 2 areas of Miami-Dade County (an area of Miami Beach and a neighborhood north of Miami). Local transmission has also occurred in all 3 US Territories (US Virgin Islands, American Samoa, Puerto Rico). Travelers to any of these areas are at risk of infection.

What are the primary symptoms of Zika virus infection outside of the neonatal period?
Most children, adolescents and adults who become infected with Zika virus remain unaware, as only ~20% of those infected become symptomatic. Symptoms generally last less than a week and the most common symptom in adults is a maculopapular pruritic centrifugally- spreading rash; though rash may also be seen in children, it is less common. Both adults and children often have low-grade fever, and children more often are seen to have nonspecific gastrointestinal symptoms. Other common symptoms seen in infected adults are a non-purulent conjunctivitis and moderate arthralgias, particularly of the small joints of the hands and feet; children do not present with these symptoms as often however arthralgias may be difficult to assess in younger children. For those who are infected and even amongst those who are symptomatic, Zika virus infection is seldom severe and only very rarely lethal (usually only in those with predisposing conditions). Of note, it is important to keep Dengue virus infections in the differential diagnosis when considering a Zika virus infection as this virus is endemic in many of the same regions, and some of the symptoms are similar (fever – though often higher in Dengue, rash – though more common in Zika and arthralgias, myalgias and headache- seen in both infections); however, Dengue virus infection may occasionally result in hemorrhage and shock, thus attention to the hemodynamic status and evidence of bleeding in sick patients returning from travel is essential. NSAIDs and aspirin need to be avoided as antipyretics in these patients until definitive exclusion of Dengue virus.

What treatment is available for Zika virus infection? Are there any ways to prevent infection?
At this time, there are no available therapies for Zika infection or vaccine to prevent it. Current recommendations center on the two most common means of transmission: mosquito bites and unprotected sex. Prevention of bites in endemic areas includes appropriate use of mosquito repellents and barrier clothing (long sleeved shirts and long pants) and actions to prevent exposure to mosquitoes including sleeping under nets when outdoors and emptying all standing water. Sexual transmission of Zika virus can be prevented by abstinence or the strict use of condoms throughout a sexual encounter (including vaginal, anal or oral sex).

What are the specific recommendations regarding the timing for the use of condoms continued on page 14...
during sex after Zika exposure or infection?
It is first important to recognize that Zika virus transmission may occur via sex whether an infected individual is currently symptomatic or not. Amongst pregnant women, condom use is essential for sex with a partner who lives in or traveled to an area with Zika, and should be used throughout the entire pregnancy. For individuals not residing in an endemic area, recommendations regarding condom use and planning a pregnancy are similar. For women and men who have been diagnosed with Zika virus or who have characteristic symptoms of Zika (even without confirmatory testing), the CDC recommends women wait at least 8 weeks after their symptoms first appeared and men wait at least 6 months after their symptoms first appeared to have unprotected sex/attempting conception. For men and women without symptoms of Zika virus but who had possible exposure from either recent travel or sexual contact, the CDC recommends waiting at least 8 weeks after the possible exposure before unprotected sex/attempting conception.

What findings may be seen in infants born to mothers infected with Zika virus?
At this time, the full spectrum of manifestations infants with congenital Zika virus infection may demonstrate is not completely known. Infants may have microcephaly, at times somewhat profound, and often other findings associated with fetal brain disruption sequence (redundant scalp skin, overlapping sutures, prominent occipital bone, and profound neurologic impairment) suggesting complete arrest of brain growth during fetal development. Other findings include other brain findings, the most common of which are ventriculomegaly and cerebral calcifications (mostly of the parietal and frontal lobes, basal ganglia and thalamus), hearing loss and other findings on neurologic exam (hyper-and hyptotonia, hyperreflexia, seizures), multiple variations of ophthalmologic findings and several orthopedic findings suspected to have originated from neurologic insults. Infected infants may also demonstrate growth restriction and some may die at any point during gestation.

How can Zika virus infection be diagnosed?
Serologic and RT-PCR testing are the primary methods for diagnosing Zika virus infection, and practitioners should seek guidance from their local Health Department for testing of suspected cases. Testing to be performed varies with timing of presentation with respect to symptom onset. Within the first week after onset of symptoms, Zika virus may be detected in the blood by PCR; however, it may be detected in urine for up to 2 weeks after symptom onset. From about the 4th day after symptom onset through about 12 weeks, IgM for Zika virus will be positive. Given significant cross-reactivity in IgM between flaviviruses, confirmation of Zika virus IgM antibody response with a plaque reduction neutralization testing (PRNT) must be performed; at this time, this testing is only available at the CDC (local Health Departments help to coordinate this testing). Beyond 12 weeks after symptom onset, testing is not recommended for non-pregnant individuals.

Infants suspected of congenital Zika virus infection should have RT-PCR of the blood and urine as well as IgM antibody testing performed within 2 days of birth. Blood should be obtained directly from the infant as cord blood is not recommended. It is recommended that any infant born to a mother with confirmed Zika virus infection during pregnancy or any infant with abnormal clinical findings consistent with congenital Zika and a maternal epidemiologic link with the virus be tested.

Is any other evaluation recommended for infants suspected to have congenital Zika virus infection?
The CDC has published an outline for assessment of both symptomatic and asymptomatic infants born to mothers with both suspected and confirmed Zika virus infection during pregnancy. All infants of mothers known to have sustained a Zika infection during pregnancy should have a complete physical exam, hearing screen and head ultrasound just after birth. If an asymptomatic infant born to a known Zika-infected mother is confirmed to have been infected with Zika virus, an ophthalmologic exam within the first month of life should be performed and additional follow-up should include close monitoring of head circumference, development and reassessment of hearing (infants of such mothers who are not infected do not need an eye exam but should have similar follow-up). Infants with findings consistent with congenital Zika infection should have the assessments listed previously but also labwork (CBC, metabolic panel, LFTs) but more detailed neuroimaging should be considered. If a symptomatic infant is confirmed to be infected, follow-up thyroid screening, neurologic exam and eye exams are recommended. Practitioners caring for infants with findings consistent with congenital Zika syndrome should consider consultation with Ophthalmologists, Infectious Disease specialists, Neurologists and Endocrinologists.

Comprehensive and up to date recommendations can be found at the CDC website: https://www.cdc.gov/zika/

References
VA-AAP Newsletter Registration and Evaluation Form  
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You have the opportunity to claim up to 1 AMA PRA Category 1 Credit(s) ™.

To claim CME credit, please complete the survey below, or you may also visit https://www.surveymonkey.com/r/VAAAPFall2016 and complete online.

NAME: __________________________________________________________________________________________

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For this activity, how many hours of CME are you claiming? ___________ (Max. 1 hours)

As a result of reading the articles, will you make any changes in your practice? □ Yes  □ No

Please list up to 3 strategies that you plan to implement as a result of reading the articles? (answer required for credit)

1. _______________________________________________________________________________________________
   _______________________________________________________________________________________________

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If you will not make any practice changes, did this activity reinforce your current practice of pediatrics? □ Yes  □ No

Please explain:

How could this activity be improved?

Future Topic Requests (optional):

Excellent                    Average                      Poor

____________________________________________________________________________________

Overall, how would you rate this activity?  5  4  3  2  1

This CME activity will expire on November 2017.
Please send form to: CME Office, 601 Children’s Lane, Norfolk, VA  23507
Please allow up to 8 weeks to receive your certificate.
Antenatal hydronephrosis, or urinary tract dilation (UTD), is one of the most commonly identified congenital abnormalities in utero thanks to excellent sensitivity of antenatal ultrasound technology. Unfortunately the classification and definitions of UTD are varied and do not currently offer significant predictive value to distinguish which cases will have a higher likelihood of urinary tract infections or require postnatal surgical intervention. Furthermore, nephrologists and urologists are currently unable to predict which of the mild and moderate cases will likely resolve postnatally. Over-identification of what will eventually become normal leads to an increased number of postnatal tests, follow-up visits with specialists, and high parental anxiety.

A multi-disciplinary consensus classification system for antenatal and postnatal UTD was recently created to address these issues in diagnosis and predictability. Dr. Tony Herndon, a pediatric Urologist who recently joined Dr. John Edmonson at CHoR Nephrology and Urology at VCU, was instrumental in the creation of the classification system. Dr. Cristin Kaspar, a third year Nephrology fellow with CHoR, presented her research on the validation of this UTD classification system at the Pediatric Academic Societies 2016 meeting, (manuscript submission pending). In this retrospective study of 203 consecutive patients, we found: (1) that antenatal ultrasound can be normal for UTD in the second trimester and develop as late as in the third trimester of pregnancy; (2) that the “Increased Risk UTD A2-3” classification, if present in the second trimester, is unlikely to normalize before birth; (3) resolution of UTD postnatally can occur in both “Low Risk UTD A1” and “Increased Risk UTD A2-3” classifications. Postnatally the UTD system was also able to differentiate severity of kidney and urinary tract abnormalities, specifically UPJ and UVJ obstructions and posterior urethral valves, but more research is needed in validating its ability to predict likelihood of UTI, urinary-tract related surgery, or positive vesico-ureteral reflux.

We at CHoR Nephrology and Urology at VCU are excited to continue research in these areas, and to be able to assist our families in evidence-based decision making. Families or primary care physicians wishing to know more about any prenatal kidney or urinary tract condition can contact our office for a consultation visit prenatally free of charge at 804-827-2264.

References
It is common for physicians to leave clinical practice for some period during their career and then seek to reenter clinical practice. Some physicians leave clinical practice to pursue a career opportunity or passion in a non-clinical setting. This could be either short-term (just a few years) or for an indefinite period. Examples range from taking an administrative position at a hospital, to pursuing a full-time research opportunity, to doing mission work abroad. Physicians who are considering a non-clinical career should keep the following key points in mind.

**Be Open to Possibilities**

Be aware that even a short-term sabbatical from clinical practice, can make it difficult and expensive to just step back into clinical practice. Be open to the possibility that your professional future may include going back into clinical practice even if at the moment it is not something you see yourself doing. Being prepared for that option is important.

**Licensure is a Privilege**

Licensure is a privilege. It is often hard to reinstate a license once it has expired. Before you leave clinical practice, contact your medical licensing board to find out about its policies regarding a leave of absence. Ask your board how it handles physicians who are not in clinical practice, if they issue limited licenses, and what statutes, if any, your state has in place to facilitate a return to practice. Overlooking regulatory issues can result in great difficulty and frustration when trying to reestablish a lapsed license.

**Explore Ways to Not Leave Clinical Practice Completely**

Find out if there are ways to not leave clinical practice completely. Explore opportunities to moonlight or take call at local hospitals, ambulatory centers, and other clinical venues during your absence as a way of maintaining some clinical activity. Look into the possibility of doing volunteer work. Remember that preparation for Maintenance of Certification (MOC) may require some clinical work to meet criteria for renewal of your specialty boards. Even some non-clinical roles in health care may require MOC status.

**Seek Advice from those in Non-Clinical Careers & Others**

Consult with physicians who have chosen non-clinical careers and learn what their experience has been like. Seek support and insights from individuals who have been in similar situations, as well as from others, both professionals and non-professionals (mental health professionals, clergy, colleagues, career advisors etc.).

**Stay connected with your Clinical Colleagues**

Stay connected with your clinical colleagues and maintain paths to reconnect with clinical practice opportunities. You may also need to ask a clinical colleague to serve as a mentor when you return to clinical practice. Consider staying on or joining hospital committees and maintaining contacts within your community that may be useful should you decide to reenter clinical practice.

**Keep Informed and Plan Ahead**

Keep informed about new developments in your specialty as well as any regulatory issues that may influence a return to clinical practice. Check with local hospitals, specialty boards, and state licensing boards about any changes in requirements that may affect your ability to reenter clinical practice. In the interest of patient safety, be prepared to have to demonstrate your clinical competence. On a regular basis consult resources like The Physician Reentry into the Workforce Project’s website at www.physicianreentry.org to remain up-to-date on physician reentry issues. Beware that returning to clinical practice is a process that takes planning. It may take a year or more to return to clinical practice depending on your specialty, state, and time away from clinical practice.

**Citation:** Stepping Away from Your Clinical Career? Key Points to Keep in Mind About Physician Reentry. American Academy of Pediatrics; The Physician Reentry into the Workforce Project, Elk Grove Village, Ill. 2016.
Pediatric General Assembly Day

Monday, January 30th, 2017 | 7:30AM - 2:00PM

Hilton Garden Inn | 501 E. Broad Street | Richmond, Virginia

Help the Virginia Chapter
make this the best
Pediatric General Assembly day ever!

Pediatric Resident Competition again this year!

Participating pediatric residency programs will be eligible for prizes for their participation such as a morning report breakfast.

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Contact: JChappell@ramdocs.org | More details to follow on website www.virginiapediatrics.org
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Name: ________________________________
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Please check: (□) VA Chap AAP member (□) Fellow (□) Faculty
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Other; please specify:

If you grew up in Virginia, where are you from? ______________________________

Are there specific child health issues that you are interested in working on this legislative session?

Have you ever met with your state or federal legislators before?

□ Yes □ No

Who are your legislators?

Senator:
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(Go to the “Who’s My Legislator?” section of the Virginia General Assembly webpage at http://conview.state.va.us/whosmy.nsf/main?openform to find out).

Return form to: Jane Chappell by fax 804/788-9987 or email to jchappell@ramdocs.org.
The Doctor Yum Project Goes on the Road!

The Doctor Yum Project team met up with Coach Mel in San Francisco for the American Academy of Pediatrics National Conference to spread the word about the work that we are doing in Fredericksburg. We were proud to talk about local families cooking at home together, our nine partner practices, and all the schools participating in the Doctor Yum Preschool Food Adventure.

Doctor Yum and Coach Mel also got to sign copies of their award winning book, “Raising a Healthy, Happy Eater” for doctors to use in their offices all across the globe!