

The Monitor

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From the Mind of the Chair

We are pleased to have a new issue of The Monitor to present to you in advance of the **AACC Annual meeting in Houston, TX July 28-August 1.**

A variety of features are included in this issue, including a reference interval corner highlighting obstetric practice changes impacting neonatal specimens in the lab ; excerpts from the literature; the next in our “ABC” didactic series, a piece on “R”-rickets; an enlightening interview with Greg Miller, past AACC Chair; this year’s Division award winners ; and upcoming meetings of interest.

We have a good variety of upcoming AACC Pediatric Maternal Fetal Division offerings at the Annual meeting in Houston, with dates and times provided to you in this issue.

Looking ahead, the International Federation of Clinical Chemistry and Laboratory Medicine (IFCC) World Lab meeting in Istanbul, Turkey is slated for 2014. Just preceding this meeting, the **XIIIth International Congress of Paediatric Laboratory Medicine (ICPLM) is the triennial meeting of the Task Force on Paediatric Laboratory Medicine (TF-PLM), June 20-22, 2014.** The focus of the ICPLM congress is to present a variety of themed symposia with the latest scientific and technological achievements in areas of paediatric clinical and diagnostic laboratory medicine. The meeting will take place immediately before the IFCC World Lab (which is scheduled for June 22-26, 2014). You can find more information about the Congress at:

<http://www.icplm2014.org/>

Two of our PMF members, Sihe Wang and Sharon Geaghan, are currently working on the scientific program.

We continue to value the scope and import of questions and issues in pediatric and maternal-fetal laboratory medicine presented to our listserv by all of you. Some of these important queries do not have an evidence-based answer, and remain unanswered on our listserv. In this year, I would like to see our Division leadership take such issues and make contributions to our field in advancement of science. We have commitment to this mission and will look to our members as well, to continue to help define best practice for our profession of clinical laboratory science and its application to health care.

Best wishes for a fulfilling summer and do consider joining us in Houston for one of our PMF activities.

Sharie Geaghan M.D.

Chair, AACC Pediatric Maternal Fetal Division

Reference Interval Corner

Sharon M Geaghan M.D.

Recent changes in obstetric practice: implications for laboratory specimens

Obstetric practice has varied widely in terms of the time before clamping the umbilical cord after delivery. The usual time of cord clamping after delivery may be 15-20 seconds. Delay of umbilical cord clamping provides additional placental transfusion to neonates, and can result in higher hematocrits. In preterm infants this is critically important, as frequent and extensive laboratory testing in neonatal intensive care units is the number one cause of blood loss and anemia. Overall, excessive phlebotomy “overdraws” in excess of the minimum requirement—a common occurrence in preterm infants—and typical weekly phlebotomy loss for a preterm infant during the first two weeks of life can be up to 30% of their total blood volume. Approximately the same volume is transfused to compensate for losses. RBC transfusions have the risk of incompatibility, suppression of the hematopoietic, transfusion reactions, viral infections, and, based on recent literature, poorer outcomes ¹.

Evidence base for delayed cord clamping

The American College of Obstetricians and Gynecologists (ACOG) and the American Academy of Pediatrics (AAP) have recently endorsed the practice of delay of cord clamping (30-60 seconds) for preterm infants. Widespread changes in obstetric practice follow ACOG guidance. The evidence base is a literature that has conclusively demonstrated that clamping the cord after 30-60 seconds increases circulating blood volume and red cell iron stores; reduces the incidence of anemia and therefore, the need for red blood cell transfusions. There is no current evidence base for delaying cord clamping after delivery of full term infants. Most importantly, the risk of intracranial bleeding is reduced approximately 50%.²

The number of newborns polycythemia increases with late cord clamping

To evaluate the effect of cord clamping time on outcomes, a randomized controlled trial of term neonates without complications was conducted in two obstetrical units in Argentina. Neonates were assigned to one of three groups: cord clamping at <15 sec (group 1), at 1 minute (group 2) or 3 minutes (group 3) and compared the venous hematocrit was measured 6 hours following birth. The hematocrits were 53.5% (group 1), 57.0% (group 2), and 59.4% (group 3). The prevalence of neonates with anemia, defined as hematocrits < 45% was low in the delayed cord clamping groups 2 and 3 (1.1 % and 0.0 % respectively) compared to 8.9% in the early cord clamping group. Importantly, the prevalence of polycythemia (hematocrit > 65%) was 4.4% in group 1 and 5.9% in group 2; but significantly increased in group 3, to 14.1%. No untoward effects due to these higher hematocrits were manifest. No increase in bilirubin levels or other deleterious clinical outcomes were evident in the delayed clamping groups, but in the early clamping group, prevalence of anemia was increased at 6, 24, and 48 hours. Maternal

effects were also evaluated: blood loss at delivery and hematocrit differences between delivery and at 24 hours were similar amongst the three groups³.

High neonatal hematocrits decrease over the first days of life

In a randomized, controlled study, the authors found that the number of polycythemic neonates with hematocrits > 65% dropped from 14.1% to 7.8% at 24-48 hours in the group that had 3 minutes of delay to cord clamping, who began with a mean of 59.4% hematocrit at 6 hours after birth³.

Ideal time for cord clamping is yet to be determined

Clinical studies have not established the optimum evidence-based time for cord clamping.

Neonatal specimen issues are directly relevant to the clinical laboratory

The current and growing trend towards longer delays in cord clamping after delivery will, based on the published literature, lead to greater numbers of polycythemic newborns and consequent laboratory samples with higher hematocrits.

We can expect the following (see below):

- High hematocrit samples leave less sample after centrifugation for analysis (chemistries), and may lead to more quantity not sufficient (QNS) samples

- High hematocrit samples clot more readily and may lead to rejected samples

- High hematocrit samples are more viscous and may have more frequent clots in instrumentation

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The ABC's of Pediatric Laboratory Medicine- R is for Rickets

By Jumoke Oladipo, MD, DABCC, Clinical Chemist, Staten Island University Hospital, NY

Rickets is a disorder caused by inadequate mineralization of bone matrix at the growth plates that occurs in children before epiphyseal fusion. Decreased endochondrial ossification leads to excessive epiphyseal cartilage, growth failure and skeletal deformities. The growth plate continues to thicken despite the lack of mineralization due to continued growth of the cartilage of osteoid. There is also a general softening of the bones and widening of the metaphyses. Rickets and osteomalacia are often used interchangeably but osteomalacia is due to inadequate mineralization of bone matrix which can exist with or without rickets.

Rickets is thought to have its roots in Northern Europe in the 17th century although it was not well described till 1645^{1,2}. Subsequently, in 1650, Francis Glisson concluded that rickets was a disease of children with peak incidence between eighteen months of age and two and half years^{1,2}. He also proposed that rickets was neither contagious nor heritable but suggested a mystic nature to the etiology of rickets. Names by which rickets was known in the 17th and 18th centuries included gibbosus or cyrtosis (meaning curve), rachitis and the English disease³.

Rickets was very common in the New England colonies of the United States and in Europe where its etiology was attributed to deficient diet, unhygienic environment, lack of exercise and lack of exposure to sunlight.³ The late 19th and 20th centuries saw a tremendous expansion in the knowledge of rickets; early animal studies linked rickets to the absence of a dietary fat which was believed to be closely linked to vitamin A⁴. It was not until 1922 that McCollum *et al* characterized this dietary fat as vitamin D⁵. It was also discovered that sunlight and cod liver oil were essential in the prevention and treatment of rickets.

The association of rickets with seasonal variations (higher incidence in winter months), dietary deficiency, skin pigmentation and exclusive breastfeeding led to diet fortification and improved therapy with subsequent eradication of rickets in the United States by the mid 1900s. Thereafter, rickets was considered to be a disease of the developing countries until reports of its re-emergence in the United States in recent years with majority of the affected infants being dark skinned and residents of the northern latitudes^{5,6,7}. Currently, rickets is considered not to be limited to developing countries nor a disease of the past.

Etiology and Epidemiology:

Rickets can be due to either predominant calcium or predominant phosphate deficiencies. The most common causes of calcium related rickets (Calcipenic) are due to vitamin D abnormalities with nutritional deficiency topping the list. Others include rickets of prematurity, vitamin D dependent rickets, chronic renal failure, congenital Vitamin D deficiency and intestinal malabsorption syndromes. Obesity has also been documented as an increasing cause of vitamin D deficiency due to sequestration of vitamin D in adipose tissue⁸.

Phosphopenic rickets results most commonly from renal phosphate wasting (phosphaturia). Causes could be hereditary (XLH - X-linked hypophosphatemia, ADHR - autosomal dominant, ARHR - autosomal recessive variants) or acquired (intrinsic renal tubular diseases and tumor induced rickets). Drug induced rickets due to prolonged use of antiepilepsy drugs (phenytoin, carbamazepine and phenobarbital) has been reported; these reports were mostly in institutionalized children⁹, while reports in ambulatory children have not found evidence of rickets^{10,11}. Reports of more recent experience with the classic antiepileptic drugs are not available for children because, typically, these drugs are no longer used as first-line agents in pediatric epilepsy with the exception of neonatal seizures in which phenobarbital and phenytoin are still used.

Vitamin D deficiency is particularly prevalent among exclusively breastfed dark skinned babies, because in addition to the skin pigmentation, breast milk has very low levels of vitamin D. Surprisingly, rickets have been reported in sun rich areas with prevalence rates as high as 24%¹². Dark skinned persons will require a higher amount of exposure to sunlight compared to light skinned persons for an equivalent amount of vitamin D. Urban environments also have challenges with obtaining adequate UV-B exposure due to fear of violence, work schedules, prolonged use of television, video games and computers by children and use of sunscreen to prevent skin cancer.

The true incidence of rickets in the US is unknown since this is not a reportable disease however; several studies have suggested the re-emergence of nutritional rickets in the US in recent years especially in areas with reduced sunlight^{6,8,13}. There has also been a resurgence of rickets in the UK and major European countries especially among immigrants from South Asia, Africa, Afro-Caribbean and Middle East (figure 1). In recent times, the promotion of breastfeeding has been associated with an increase in the incidence of rickets especially amongst breastfed dark skinned children¹⁴. The greatest burden of this disease however remains in Africa, the Middle East and Asia where disease prevalence are reported to be between 10 - 70%¹⁵.

Shock rise in rickets in kids

By STAFF REPORTER

Published: 12 Nov 2010

[Add a comment \(11\)](#)



MORE than one in five kids are showing signs of bone disease rickets as cases explode, say doctors.

Experts were stunned to find 20 per cent of children have the disease and cases of the condition — traditionally linked to poverty — are not concentrated in kids from any particular background.

Figure 1: Resurgence of rickets made news headlines. This excerpt is from an article based on research conducted in Southampton, UK by Professor Nicholas Clarke, consultant orthopaedic surgeon at Southampton General Hospital and professor of paediatric orthopaedic surgery at the University of Southampton.

Summary of Ca and Vit D Homeostasis:

A proper balance between bone mineral deposition and resorption has to be maintained for adequate bone growth. The parathyroid gland through its calcium sensing receptor is the major organ responsible for the maintenance of circulating calcium levels. Release of parathyroid hormone (PTH) leads to

metabolic effects that include increase in osteoclastic bone resorption, increase in renal tubular reabsorption of calcium and increased excretion of phosphates. There is also up-regulation of 1,25-dihydroxycholecalciferol (1,25DHCC) which is responsible for producing active vitamin D. One of the most important actions of active vitamin D is to increase the intestinal absorption of calcium.

Vitamin D metabolism has been discussed extensively by Dr. V Grey in the 2006 online edition of the monitor¹⁶; in summary however, the major organs involved in vitamin D metabolism are the skin, parathyroids, intestines, kidneys and bone. Vitamin D is responsible for normal mineral ion concentrations and is required not only to prevent rickets but also for many cellular and neuromuscular functions. It also has skeletal and extraskkeletal effects. It promotes the gene for calbindin, a calcium binding protein that transports calcium across the enterocyte. It also increases the production of calcium channels along the intestinal walls permitting calcium movement from the enterocyte into the circulation. Vitamin D is essential for the actions of PTH on osteoclasts and stimulates the production of osteocalcin and osteopontin (calcium binding proteins) by osteoblasts. It can be concluded that PTH is the early responder in controlling circulating calcium concentrations whereas vitamin D is the late responder in maintaining a normal mineralized skeleton.

Pathophysiology

A study by Fraser et al in 1967¹⁷ concisely explains the three classic stages of rickets. In stage I, there is hypocalcemia caused by vitamin D deficiency, development of secondary hyperparathyroidism, upregulation of 1 α hydroxylase enzyme, and increase in calcium intestinal absorption. This stage may be asymptomatic or may manifest as convulsions. In stage II, normocalcemia, hyperaminoaciduria, hypophosphatemia and hyperphosphaturia with evident clinical rickets is present. Stage III is a progression of stage II but with recurrence of hypocalcemia, convulsions and severe skeletal manifestations. Bone disease starts in the late phase of stage I. The three stages may not be present in all patients.

Placental transfer of 25OHD (25 hydroxyvitamin D) is protective for the first three months of life and symptoms begin to appear thereafter. Congenital rickets is very rare and is usually due to severe maternal calcium deficiency.

Vitamin D dependent rickets - 1 (VDDR-1, pseudo vitamin D deficiency) is caused by an autosomal recessive defect in 1- α hydroxylase. This is a very rare disorder with the exception of a French Canadian population in Quebec with a prevalence rate at birth of 1 in 2916 and a carrier rate of 1 in 27¹⁸. Calcitriol (1,25OH₂D) resistant rickets (CRR or vitamin D resistant rickets) is due to an autosomal recessive defect in the calcitriol receptor resulting in absent vitamin D signaling.

The past few years has broadened our knowledge of phosphate metabolism, now we know that intestinal absorption of phosphate is independent of vitamin D. The renal system is responsible for phosphate balance by tubular reabsorption and excretion controlled by phosphatonins and PTH. Fibroblast growth factor 23 (FGF23) has been recognized as a major phosphatonin that regulates renal phosphate reabsorption. FGF23 is produced mainly by osteocytes and acts on the renal tubular cells to

prevent the reabsorption of phosphate thereby leading to increased phosphate excretion. Increased FGF23 signaling leads to excess renal phosphate loss and down regulation of 1α hydroxylase while deficient signaling leads to phosphate retention^{19,20}. One of the common mutations in XLH involves the phosphate regulating gene on the X chromosome (PHEX gene), which increases FGF23 levels and promotes phosphate wasting; there is usually no associated hypocalcemia. Autosomal dominant hypophosphatemic rickets (ADHR) was the first disease associated with FGF23 abnormalities and this is due to amino acid substitutions at the cleavage sites making the molecule resistant to cleavage, having a longer half life and increased circulating levels promoting phosphate wasting. Autosomal recessive hypophosphatemic rickets (ARHR) is rare and is due to a defect in the DMP 1 gene (Dentin Matrix Protein 1), which is highly expressed in osteoblasts and osteocytes. Acquired renal phosphate wasting in children with mesenchymal tumors is also linked to the secretion of FGF23 by these tumors²¹.

Non-phosphatonin causing phosphaturic conditions include hereditary hypophosphatemic rickets with hypercalciuria (HHRH), Fanconi syndrome and isolated intrinsic renal tubular diseases like cystinosis and tyrosinemia. HHRH is caused by a primary defect in phosphate transport involving mutations in the SLC34A3 gene which encodes for NaPi-IIc protein (Na dependent phosphate co-transporter in the proximal convoluted tubule)²². The body responds appropriately by increasing 1α hydroxylase and active vitamin D concentrations. The importance of this comes to play in the treatment of this condition in which phosphate supplementation is all that is needed for correction.

Clinical Features (refer to Figure 2):

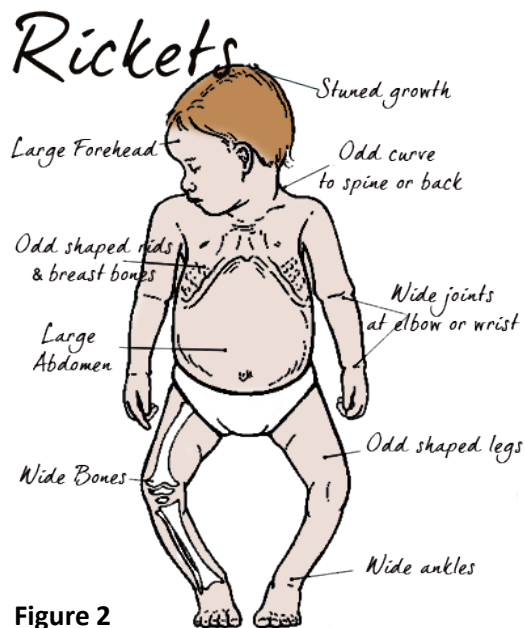


Figure 2

Most of the features of rickets are skeletal in nature. The deformities found largely depend on the age at presentation and the severity of the disease. Craniotabes (softening of the cranial bones) is a common finding. A characteristic finding in the chest is the rachitic rosary finding which is widening of the costochondral junctions giving it a beadlike feel on palpation. There is enlargement of the wrists and ankles and the characteristic Harrison groove, which is a horizontal depression along the lower anterior chest wall due to pulling in of the softened ribs by the diaphragm on inspiration. Other bony deformities are frontal bossing, scoliosis, lordosis, kyphosis, genu valgum symptoms include failure to thrive, muscle weakness,

fractures and listlessness. Children may present with tetany, seizures and cardiac failure^{23,24}.

Diagnosis

Diagnosis of rickets is usually based on the clinical features, biochemical analysis and radiologic studies. Radiologic findings are beyond the scope of this review, but is important to evaluate the skull, limbs and joints.

Biochemical and genetic tests are carried out to determine the etiology of rickets which are important to determine treatment modalities. The initial laboratory tests usually carried out in a child with rickets would include serum calcium, phosphorus, alkaline phosphatase, parathyroid hormone, 25-hydroxyvitamin D, creatinine and electrolytes. Serum 25-hydroxyvitamin D levels are the best indicator of vitamin D status. 1,25-dihydroxyvitamin D is not a useful test to determine if a child is vitamin D deficient and is frequently misordered. Vitamin D insufficiency and deficiency states have been defined as levels between 20 and 29 ng/mL and below 20 ng/mL respectively. Levels below 10 ng/mL indicates severe deficiency states. Studies suggest that peak calcium absorption occurs at levels of 32 ng/mL and maximal PTH suppression is not reached until levels of 30-40 ng/mL²³. In 2011, the Institute of Medicine (IOM) committee concluded that serum 25-hydroxyvitamin D levels of 16 ng/mL cover the requirements of half of the population and levels of 20 ng/mL cover 97.5% of the population²⁶. This created some controversy, as the levels were lower than those previously recommended.

Other investigations may be carried out depending on the suspected etiology of the rickets. Measurement of urinary calcium may be important in hereditary hypophosphatemic rickets with hypercalciuria. Amino acids and glucose in urine should be ordered if fanconi syndrome is suspected. Measurement of FGF23 has been proposed as a routine laboratory investigation to assist in determination of the etiology of hypophosphatemic rickets. Even though there are many commercial kits available for the assay of FGF23, none is FDA approved yet. Renal phosphate wasting is confirmed by very low total phosphate reabsorption and Tmp/GFR ratio (Maximal tubular reabsorption of phosphorus per glomerular filtration rate).

Table I: Biochemical findings in common causes of rickets

Disorder	Ca	Pi	PTH	25-(OH)D	1,25-(OH) ₂ D	ALP	Urine Ca	Urine Pi
Vitamin D Deficiency	↔, ↓	↓	↑	↓	↔, ↓	↑	↓	↑
VDDR - 1	↔, ↓	↓	↑	↔	↓	↑	↓	↑

CRR (VDDR -2)	↔,↓	↓	↑	↔	↑↑	↑	↓	↑
XLH	↔	↓	↔	↔	R↓	↑	↓	↑
Tumor Induced Rickets	↔	↓	↔	↔	R↓	↑	↓	↑
Fanconi Syndrome	↔	↓	↔	↔	R↑	↑	↓,↑	↑
HHRH	↔	↓↓	↔,↓	↔	↑, R↓	↑	↑	↑

↔, Normal; ↓, decreased; ↑ increased; R↓, relatively decreased; ↑↑, extremely increased; R↑, relatively increased; VDDR, vitamin D dependent rickets; CRR, calcitriol resistant rickets; XLH, X linked hypophosphatemic rickets; HHRH, hereditary hypophosphatemic rickets with hypercalcemia.

Ca, serum calcium; Pi, serum phosphate; PTH, parathyroid hormone; ALP, Serum Alkaline phosphatase.

Prevention of Vitamin D deficiency Rickets

Vitamin D deficiency rickets is preventable but remains a challenge. It is still uncertain if the prevention is due to the direct effects of vitamin D on the skeleton or indirectly through the correction of mineral ion concentration, the overall effect of adequate vitamin D levels remains very beneficial.

Most foods except for oily fish contain very little vitamin D unless artificially added. Prevention of vitamin D deficient rickets boils down to two approaches, adequate UV-B exposure and oral intake. There is still controversy as to how much exposure to sunlight should be allowed since sunlight exposure has been known to be a major risk factor for the development of skin cancer. Adequate dietary supplementation is about the only safe way to prevent this disease. The latest revision of requirements by the IOM in 2011 recommends 400 IU/day during the first year of life and 600 IU/L beyond the first year. In North America, fortification of cereals and infant formula with vitamin D is enforced and vitamin D supplementation is required for all breastfed babies. Increase in calcium intake is a factor that needs to be considered especially in areas where calcium deficiency has been identified as a major cause of rickets. Public health programs in the UK have recorded success in public awareness and a reduction in the incidence of symptomatic vitamin D deficiency²⁵.

The resurgence in the diagnosis of rickets is multifactorial, these include greater awareness among pediatricians, better laboratory methods for vitamin D assessment with an upsurge in the laboratory diagnosis of vitamin D insufficiency/deficiency, lack or block to sunlight exposure and increased breastfeeding. Since rickets occurs in children less than two years of age, optimizing vitamin D status of women in child bearing age and in their young offsprings is the method of choice.

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Excerpts from the Literature

Articles of interest compiled by the editorial board. Please welcome our new member of the editorial board, **Joely Straseski, Ph.D, DABCC, FACB.**

Powdered blend of polyunsaturated fatty acids (PUFAs) supplementation normalizes docosahexaenoic acid (DHA) and arachidonic acid (AA) levels in patients with PKU.

Jans JJ et al, Mol Gen Metabol 2013, 109:121-124. (UG)

In untreated or poorly treated PKU patients, in addition to neurotoxicity by phenylalanine, it has been hypothesized that limited availability of docosahexaenoic acid (DHA, 22:6n-3) and arachidonic acid (AA, 20:4n-6) in the brain attribute to mental retardation and microcephaly. DHA and AA are structural components of neuronal cell membranes and are pivotal for brain development and retinal function. A typical PKU diet provides low saturated and polyunsaturated fat intake and have a poor LC-PUFA status in plasma and erythrocytes as compared to healthy controls. In addition, phenylalanine metabolites phenylpyruvate and phenyllactate inhibit the endogenous synthesis of DHA and AA, and thus lowering their plasma levels in PKU patients.

In this study, the investigators measured plasma and erythrocyte DHA and AA levels in 54 patients with PKU, after supplementation with fish oil or the fatty acid supplement KeyOmega (a powdered blend of DHA and AA). In PKU patients concentrations of DHA and AA in erythrocytes and plasma were below the reference range. Although, supplementation with fish oil, DHA levels reached normal range, the AA concentrations did not reach to the normal values in these patients. In contrast, both DHA and AA levels increased and reached reference values upon supplementation with KeyOmega. The authors concluded that KeyOmega offers additional benefit over fish oil since both AA and DHA status are normalized in PKU patients supplemented with KeyOmega.

A systematic review of statistical methods used in constructing pediatric reference intervals.

Daly CH, Liu X, Grey VL, Hamid JS. Clin Biochem (2013). (JS)

Establishing reference intervals is often an arduous but critical step in new test development or validation. Pediatric populations present unique challenges that can make this task even more complicated. This article by Daly et al. discusses the need for a standardized statistical approach when establishing pediatric reference intervals. They review the current literature and investigate the different statistical methods used to construct pediatric reference intervals. The authors examine common reporting methods for the detection of outliers, partitioning into relevant groups, and statistical approaches to determining upper and lower reference limits. They also discuss concerns regarding sample size (more or less than 120), parametric vs. non-parametric statistics, and the importance of outlier detection and confidence limits.

Inclusion/exclusion criteria resulted in 22 original articles for their review. Striking variation was observed in the statistical methods used in these articles. Examples of methods include parametric, non-parametric, robust, parametric fractional polynomials and visual assessment of means and standard deviations. Fifty-nine percent of these studies used statistical methods that were outlined in the CLSI guidelines for reference interval determination. The vast majority of studies performed partitioning (96%), but not all of these tested for differences between the partitions (76%) or collapsed insignificant partitions (69%). Twenty four percent of studies that performed partitioning did not apply statistical methods to test whether the partitions were appropriate. The majority of studies did not perform tests to identify outliers in their data sets (59%). Less than 20% reported confidence intervals for their ranges.

The authors discuss the importance of providing confidence intervals and properly determining appropriate partitions and detecting outliers. Overall, they recommend the bootstrap method to determine confidence intervals by re-sampling of the data and the robust method for small sample sizes.

This topic is extremely important and the field will benefit from a discussion of, and guidelines for, the standardization of interval determinations in specific populations such as pediatrics. As large collections of pediatric reference interval data continue to be collected (e.g., CALIPER, CHILDX, KiGGS and the National Children's Study), establishing standardized criteria specific to this challenging patient population will be critical to interpretation across studies.

Interview with A Distinguished Colleague: Dr.Greg Miller

Sharon Geaghan, MD



I had a chance to catch up with Greg via a virtual interview, and he shares his insights with you as the second in a series of conversations with distinguished colleagues in our discipline

Q1. How did you come to the career decision to choose Clinical Chemistry as your profession?

I was lucky that a graduate student ahead of me learned about clinical chemistry and got me interested. Otherwise I would be mired in some drab research lab and would have missed all the excitement of clinical laboratory medicine.

Q2. Did you have a mentor and if so what did he/she teach you?

My mentor was Hanns-Dieter Gruemer with whom I trained at the Ohio State University Hospital when it was one of the top fellowship programs in the country. One of the important things he taught me is not to “polish the polish” meaning that a lab test only has to be as good as is needed for its use in patient care; we now call this “fit for purpose.” Another key learning experience was the importance of networking with colleagues. I still have collaborations with people I met during my training days at OSU and those friendships have led to many other productive collaborations over the years.

Q3. For newer chemists, do you have any pearls of wisdom for career development ?

One of the recommendations I give people is to make yourself useful to your employer and to any professional commitment. A job and a career are about how you can contribute to a team effort. Try to find a mentor, who may be at your institution or elsewhere, to help you find opportunities to get involved and to network with colleagues. Volunteer for projects and be sure your contribution is meaningful, correct and on time. There is no shame in asking for help; in fact it is foolish not to do so and will build trust. Just make sure you deliver on commitments, the rest will take care of itself.

Q4. What is your most enjoyable part of your professional work?

At this point in my career, I most enjoy creating opportunities for younger laboratory professionals to get engaged in contributing to advances in our field. I also get a lot of satisfaction from working on projects that will improve laboratory medicine on a national and international level. For example, I am co-chairing, with my good friend and colleague Gary Myers, AACC’s initiative to create the International Consortium for Harmonization of Clinical Laboratory Results (see www.harmonization.net). And there is my day job at Virginia Commonwealth University Medical Center that is as much fun today as when I started 35 years ago. I think we are all attracted to this field because of our desire to provide high quality laboratory service to patients and for public health. Doing that is quite satisfying.

Q5. What is the hardest part of your professional work?

The most challenging part is maintaining competence with advances in science and technology. There are so many exciting professional activities that it is a challenge to keep all the balls in the air and find time for self-learning. However, for a typical type A baby boomer, what more can one ask for?

Q6. The next generation of chemists have been characterized as looking for work-life balance; do you have advice for them in managing that balance from your experience?

Based on my previous response, I may be the wrong guy to ask. Even us more senior folks have had to deal with work-life balance, it is not really new. However, finding the balance is a challenge that each person must grapple with and we all have different expectations. I remind myself that one must be healthy and happy to be professionally productive over the long haul; so it is essential to make time for activities to ensure those attributes in life. I consider myself lucky that I really enjoy my professional work so it is easy for me to spend a lot of time on it. Learning organizational skills, how to prioritize and how to manage time can help to fit in as much as possible.

Q7. What developments would you most like to see occur in the field, over the next 5 years?

Medicine is increasingly dependent on evidence based practice guidelines. Many of those guidelines depend on laboratory results. I would like to see clinical laboratory professionals be part of the clinical society guidelines development teams. In addition, achieving more uniform and harmonized results from different laboratory measurement procedures would be a significant improvement in the quality of health care.

AACC Division Poster Winners

Congratulations of the winners of the Pediatric and Maternal Fetal Division 2013 poster session awards. There was a tie for both awards. Please check out their posters at the annual meeting!

Best posters:

Mohammed Attaelman Ph.D., Quest Diagnostics Nichols Institute, Valencia, CA

Development and Validation of an Improved Chemiluminescent Assay for Inhibin B

Victoria Bevilacqua M.S. Hospital for Sick Kids, Toronto, Ontario,Canada

Biological Variation and Quality Specifications for 38 Biochemical Markers in a Pediatric Population

Student and Young Faculty Awards:

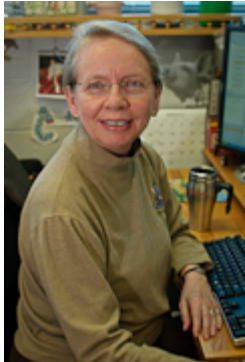
Jeffrey W. Meeusen Ph.D. , Mayo Clinic, Rochester, MN

Amniotic fluid glucose provides superior sensitivity in identifying subclinical intra-amniotic infection compared with gram stain and bacterial culture

Sarah Delaney (Ph.D. Candidate), Hospital for Sick Kids ,Toronto, Ontario, Canada

Novel Insights and Reference Intervals of Cardiac Troponin I in a Healthy Neonatal and Pediatric Population

Award for Outstanding Contributions to Pediatric and Maternal-Fetal Clinical Chemistry



Peggy Borum, Ph.D. University of Florida, Gainesville Food Science and Human Nutrition Department, Director, Metabolic Assessment Laboratory

Dr. Borum describes her teaching program as focussed on the biochemical foundations of the metabolic status found in different physiological states, and how an understanding of the biochemistry naturally leads to interventions to maintain health or to treat disease. Congratulations Dr. Borum!

2013 AACC Annual Meeting



Once again, it is time for the AACC Annual Meeting. Here are some sessions of interest for members of the Pediatric and Fetal Maternal Division.



Indicates a ticket is required for the session

Sunday July 28

Opening Plenary: Wallace H. Coulter lecture by C. Ronald Kahn, MD

“Deconvoluting the Metabolic Syndrome at a Molecular Level”



AACC University:

Laboratory-Driven Testing Algorithms: A Strategy to Improve Ordering Accuracy, Efficiency, and Utilization

Monday July 29

Plenary: John Mastick, PhD, “Challenging the Dogma: A New View of the Genomic Programming of Human Development”



Roundtables:

Anti-Mullerian Hormone (AMH): A Piece of the Infertility Puzzle

Diagnosis of Propionic and Methylmalonic Acidemias

Symposia:

Prenatal Testing of Maternal Plasma by Next Generation Sequencing: Is the Future Now?

Laboratory Challenges in Suspected Metabolic Disease: Newborn Screening and Beyond



Short courses/Interactive Workshops:

Old and New Limitations in Immunoassays: A Focus on hCG, TSH, and Patient Risk

State of the Art Serum Tumor Marker Testing for Women’s Cancers

Practical Approaches to Reference Interval Validation

Special Events:

Pediatric Maternal Fetal/Industry/Clinical Translational Science Divisions Joint Mixer and Abstract/Poster Awards

Tuesday July 30

Plenary: Bruce Hollis, PhD, and Jo Ann Manson, MD, DrPH, “The Vitamin D Debate: Is Enthusiasm Outpacing Evidence?”



Roundtables:

Limitations in hCG Point-of-Care Testing

Role of Therapeutic Drug Monitoring in Pediatric Cancer Chemotherapy

Drug Abuse Among Children and Adolescents: Beyond Traditional Drugs of Abuse

Symposia:

Human Chorionic Gonadotropin: A Complex Molecule with Changing Clinical Roles

Use and Misuse of Troponin Assays in Adult and Pediatric Hospitals

Pediatric Reference Intervals: Challenges and Recent Advances



Short courses/Interactive Workshops:

Utilization and Standardization of Laboratory Allergy Testing

Special Events:

Industry- AACC Pediatric and Maternal Fetal Division Dialogue on Advancing Pediatric Reference Intervals

Wednesday July 31

Plenary: Stuart Schreiber, PhD, “Patient-Based Therapeutics Discovery”



Roundtables:

Utility of Allergen-Specific IgE Testing in Allergic Disease

Diagnosing Ectopic Pregnancy: Current Challenges and Future Prospects

Symposia:

The Hunt for Accurate Diagnosis of Celiac Disease

Update in Metabolic Diseases



Short Courses/Interactive Workshops:

Controversies in ANA and Celiac Disease Testing

Thursday August 1

Plenary: Jeffrey Gordon, MD, “Dining in with Trillions of Fascinating Friends”

Symposia:

Sepsis: Is It the Bug or Is It the Host?

Save the Date: ICPLM 2014

The ICPLM 2014 will be June 20th-22nd, 2014, prior to the IFCC-WorldLab in Istanbul, Turkey. The exciting symposium programme is described below. Please save the dates for this exciting pediatric focused meeting which would be ideal for both the specialist and non specialist laboratory medicine

professional. The names of the plenary speakers will be announced in Spring 2013 and full detail in September.

<http://www.icplm2014.org/>