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Cover art by Henry Knudtson, age 10
I have been doing some research for a presentation I need to make to some new leaders and I came across the above passage on gratitude. I wanted to share it with you.

Have you given much thought to the things you are grateful for? Recently with the chaos in Ukraine, the plane disappearance in the Far East, the gridlock in Washington, D.C., and the uncertainties in medicine, it is easy to forget about all the wonderful things that are a part of our lives. It is easy to get caught up in the day to day work of our practices and forget to take time to enjoy the good things we are a part of every day – those special encounters with the people who let us into their lives and the wonderful people we work with.

In March, I attended the American Medical Association (AMA) National Advocacy Conference. We heard from representatives of the federal government, leaders in Congress and people who help shape health policy in this country. There seemed to be a common theme to why nothing seems to be getting done. Ideology and self-interest, worry about getting elected, and pleasing the voters back home seem to be more important than actually figuring out how to solve some of the issues facing our country. Perhaps some of these leaders should think about being “grateful” – it might help them see past the petty differences and get something done. President Ronald Reagan said many times, “there is no limit to what a man can do or where he can go if he does not mind who gets the credit.”

Another speaker at the conference was a “futurist;” he spoke of the wonderful things to come and said they are going to come at us with a breakneck pace. Each new advancement makes the next several easier to obtain. The medicine we learn even today may not be the same in just a few years as we learn more and new things. Examples will be genomics and easier use of imaging technology. He talked about how it is important to have mentors and not just the traditional mentors, but a “reverse mentor,” someone younger than ourselves to help us and teach us the new technology and how to use it. This is something I had never thought about before – another reason to be grateful for the younger generation.

Federal advocacy was one of the main focuses of the conference. The issue this year was the sustainable growth rate (SGR) formula fix. We had an opportunity to talk with all three of our congressional delegates and asked them to support, and in some cases push leadership, to move forward with legislation that would once and for all remove the annual threat of significant cuts in physician payment. This would allow physicians to then focus on newer and better ways to care for patients and do so with quality and better costs. We heard from all of our congressional delegates that they want to, but they were not optimistic that it would get solved before the end of March (I hope when you read this in April it will be a thing of the past, not just pushed to the end of the year). Maybe as good as our congressional members are, they need to think about being “grateful” for serving us as we (and they) serve the people of South Dakota.

I want to remind you that our 2014 Annual Meeting is coming up May 30-31 in Rapid City. You should have received information about the meeting, and can read more about it at www.sdsmma.org. This is a great opportunity to meet with some old friends, make some new ones and learn more about your SDSMA. I hope to see you there.

I also want to let members know that Gregg Tobin, MD, passed away on March 7. Gregg will be missed by not only his family, but also the community he served. Please remember his family and the colleagues he worked so closely with.

Book Recommendations

Tuesdays with Morrie: An Old Man, a Young Man, and Life’s Greatest Lessons by Mitch Albom
Have a Little Faith by Mitch Albom
The Five People you Meet in Heaven by Mitch Albom
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“I never saw such weather,” Pa said. “It must be all of 40 degrees below zero and not a breath of air stirring. The whole world seems frozen solid.”

So says Charles Ingalls in Laura Ingalls Wilder’s children’s book, The Long Winter. I know this cold winter brought out those same feelings in many, including myself. Activities were curtailed, enthusiasm dampened and a general melancholy settled over me because the severity of the weather never seemed to break! Because of the long, cold winter, I was somewhat surprised when legislators displayed a hearty appetite for ice cream at our Alliance 2014 Day at the Legislature Feb. 6.

Since first-year students from the University of South Dakota Sanford School of Medicine were also in Pierre that day observing legislative procedures, several of them were able to give us a hand serving ice cream sundaes to legislators. We were also able to provide legislators with information about the Alliance’s award-winning state health project, Cribts for Kids. Alliance members from east and west participated in our Day at the Legislature. A special thank you goes to Grace Wellman of Sioux Falls for coordinating the ice cream event. Thank you also to Peggy Huber of Pierre, Suzanne Wiedel of Huron, and Kris Zimmerman, Sally Kelts and her daughter, Michelle Colbath of Rapid City for braving the unending cold weather and spending the day in Pierre. Despite the cold, it was a fun day for all of us! I also want to express gratitude to everyone who made a donation to the Cribts for Kids project to honor their favorite physician on National Doctors’ Day last month. This was a wonderful opportunity to recognize our state’s dedicated physicians and support our health project.

Now that spring is in the air, I don’t think it is too early to look ahead to the SDSMA and Alliance Annual Meeting at the Ramkota Hotel and Conference Center in Rapid City next month. Alliance activities kick off with the Medical Student Scholarship Fundraiser on Thursday, May 29. I encourage your attendance and participation because our donations support the next generation of physicians in South Dakota, and it is just plain fun! All Alliance members are also encouraged to attend our annual meeting and the officer installation luncheon on Friday, May 30. Renew old friendships, make some new ones, and catch up on Alliance activities at the same time. This is the time to celebrate the successes of the past year and set our goals for the coming year. I hope to see you there and ask you: what can we accomplish together?

Were you able to find the answer to the trivia question from last month? Dr. Crawford Long was the 26-year-old physician who first used ether to painlessly remove a tumor from the neck of a patient in 1842. He had been practicing in Georgia for less than a year, but his sympathy and concern for his patients caused him to search for a way to relieve their needless pain. The U.S. Congress declared March 30 to be National Doctors’ Day in 1958 – 56 years ago. The red carnation became the official flower for Doctors’ Day because it is the symbol of love, charity, sacrifice, bravery and courage.

South Dakota’s medical school first began as a two-year school in 1907, but do you know when they graduated their first class? Who was considered the very first graduate? Do you know what year the medical school graduated its first class of four-year students, the University of South Dakota’s very first medical doctors?
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Travis Liddell, MD

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Virtually everyone who reads this has experienced something so shocking or life-altering that you remember where you were the exact moment it happened. It can be hard to describe how you felt in that instant because it was so visceral. Words lack the depth necessary to do it justice. One of those times for me happened as my wife and I drove away from Boston Children’s Hospital having just been told our 2-year-old son wasn’t talking because he has autism. In that instant, the trajectory of not only his life but also his parents and future sibling’s was permanently altered.

As most physicians know, used indiscriminately, the “retrospectoscope” can be a dangerous tool. It all seems so obvious to us now – delayed speech, male gender, looking down straight lines, repetitive behaviors, sensory integration problems. How could two reasonably educated people miss it for so long? The answer isn’t that complicated. We are his parents, not his physician… he was our first child… “boys take longer to talk…” Two parts ignorance, one part denial. If it weren’t for his kind, astute, persistent pediatrician, who knows where we would be today. I’ve no doubt she suspected it all along and that is why she kept close tabs on him. She was the first to teach us about advocating for him through her example. We owe her a debt we can never realistically repay. Logically, her workup started with speech therapy evaluations and treatment. From there we proceeded to developmental pediatricians, neurologists, etc. After his initial developmental evaluation and a confirmatory second opinion, our focus shifted to acceptance and education of what we needed to do for him.

At the time my wife was lucky to have a program director that, without a moment’s hesitation, allowed her to take time off from her fellowship so she could begin the task of searching for and enrolling him in the services he needed. For those of you who have never had to face this, it is a daunting task and was a full time job. There is no centralized source to guide you. What we found was a patchwork of services knit together by a network of parents and organizations. They are mostly invisible to the general public but they are everywhere and care fiercely about these kids. How fortunate we were at that time to live in a state where the general consensus is that a timely, proactive approach is critical and where the resources seemed limitless. He was quickly enrolled in 30 hours per week of intensive home behavioral therapy. Once he was 3, the local school system assumed charge of his plan and continued it with vigor. He still faces daily challenges that typically developing kids do not. However, there is no doubt in our minds that because of this aggressive and early intervention, he became the kid he is today – full of unlimited potential and who will contribute many positive things to the world around him that he could not have without this help.

We all know the prevalence of autism spectrum disorder (ASD) is reaching frightening levels, more than doubling from one in 150 kids in the year 2000 to one in 68 by the last Centers for Disease Control and Prevention estimate recently released. We also know that until our understanding of this disease is more sophisticated, the best outcome for them currently is via early identification and aggressive, individualized intervention, because no two ASD kids are alike. If you haven’t already, there is no chance that you won’t have some meaningful interaction with one of these kids at some point in the near future. They are your kids, your grandkids, your neighbors and your patients. Sometimes the hardest part is to let yourself recognize it’s there.

There should be no shame or hesitation in searching for or receiving the diagnosis. So use our example of “success” and the observance of National Autism Awareness Month to spur you to become an advocate for these kids and their parents in whatever way you can – giving/raising money for research, referring kids for evaluation sooner rather than later, or lobbying for better health care coverage. Don’t let public opinion, school systems or politicians tell you they need less. We are the ones who know better. They need us and we will all be better for it in the long run.

About the Author: Jason Knudtson, MD, FACS, general/critical care surgeon, Sanford Surgical Associates.
Outstanding Teaching

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The Golden Apple Award is given in recognition of and with gratitude for continuing dedication, sacrifice and passion for excellence to the faculty member at each campus who most helped and inspired the third and fourth year medical students.

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"Dr. Fossum was an excellent instructor and a brilliant physician. He was always willing to help, and loved to teach. He was very approachable and eager to share his wealth of knowledge.”

Mark Lounsbery, DO
Sioux Falls Campus

"Really had a great experience with Dr. Lounsbery! Thanks! He is an excellent teacher, he took time for me.”

Byron Nielsen, MD
Yankton Campus

"Excellent teacher; probably the best attending I have had during my clinical years in that he taught and explained things very well and took the time to teach to his students. Great with his patients.”

Heidi Strouth, MD
Rapid City Campus

"Dr. Strouth took the time to teach, she is a great teacher. She is awesome.”

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Propafenone Associated Severe Central Nervous System and Cardiovascular Toxicity Due to Mirtazapine: A Case of Severe Drug Interaction

By Naveen Rajpurohit, MD, Sudeep Raj Aryal, MD; Muhammad A. Khan, MD; Adam T. Stys, MD; and Tomasz P. Stys, MD

Abstract
We describe a rare case of severe drug-drug interaction between propafenone and mirtazapine leading to propafenone toxicity. A 69-year-old Caucasian male taking propafenone for atrial fibrillation was prescribed mirtazapine for insomnia. Subsequent to the first dose of mirtazapine the patient experienced seizures, bradycardia and prolonged QRS as well as QTc intervals on EKG. The patient was admitted to the ICU and recovered after supportive management.

Propafenone is an established class IC antiarrhythmic drug commonly used in the treatment of atrial fibrillation. It is metabolized through the CYP4502D6 pathway. Five to 10 percent of Caucasians are poor metabolizers. Mirtazapine is a commonly prescribed antidepressant drug, which is also metabolized through and may modulate the CYP4502D6 pathway leading to altered metabolism of propafenone and possible adverse effects. In this case, toxicity was reversed once the offending drugs were discontinued. An extensive review of the literature revealed this to be the first described case of drug interaction between propafenone and mirtazapine.

Introduction
Drugs with narrow therapeutic index serve as a double-edged sword. They are efficacious within a narrow therapeutic range but may cause serious untoward effects when beyond this limited range. Apart from optimization of the individual dosage, drug interactions especially when used in conjunction with other drugs that affect the same metabolic pathways, determine final drug level and potential effects. Propafenone, a widely used effective antiarrhythmic drug has serious dose-related toxicity beyond its narrow therapeutic range. Propafenone toxicity is associated with fatal cardiac arrhythmias amongst other organ system involvement. Drugs that interact with propafenone to alter its pharmacokinetics may either decrease or increase its plasma concentrations leading to a lack of optimum therapy or serious adverse effects, respectively. We report a case involving a drug-drug interaction between propafenone and mirtazapine. A 69-year-old male on chronic propafenone therapy for paroxysmal atrial fibrillation, presented with new onset seizures and symptomatic bradycardia after taking the first dose of mirtazapine prescribed for insomnia.

Case Report
A 69-year-old Caucasian male with paroxysmal atrial fibrillation treated with propafenone 325 mg BID presented with new onset seizures which occurred while sleeping. The patient had a long-standing history of insomnia and had been sleeping only one to two hours per night for the last few days. About 12 hours prior to the seizure, he took a new prescription of 15 mg mirtazapine for insomnia. Other medications included aspirin 81 mg and multivitamins. His wife reported that she woke to discover her husband having seizure-like activity, with jerking of bilateral upper and lower extremities, lasting about a minute. He experienced two more seizure episodes en route to the hospital. The patient was noted to be hypotensive (systolic blood pressure about 50 mmHg) and bradycardic (heart rate in 30bpm). An electrocardiogram (EKG) done in the ED demonstrated atrial fibrillation, new right bundle branch block, QRS prolongation of 154ms and a corrected QT interval of 500ms (Figure 1). The patient was intubated and sedated for airway protection. He received intravenous epinephrine (1 mg), atropine (3mg) and dopamine drip was initiated for hypotension. With
this therapy the patient’s heart rate and blood pressure started to stabilize. Initial blood work revealed that levels of hemoglobin, urea, creatinine, electrolytes, magnesium, glucose, creatinine kinase, troponin T, liver function tests and coagulation markers were within normal limits.

His wife confirmed that the patient had not accidently or intentionally overdosed on any of his medications. Both propafenone and mirtazapine were considered as possible causes of this patient’s presentation; however, propafenone toxicity was felt to be the most likely culprit. Computerized tomography and magnetic resonance imaging of the brain were negative for any acute intracranial pathology. An electroencephalogram (EEG) showed moderate diffuse encephalopathy, most likely secondary to pharmacologic sedation. Continued fluid resuscitation with normal saline was performed and his hemodynamic status improved gradually. An EKG performed six hours after the admission showed sinus bradycardia (heart rate 50 bpm), QRS interval of 101ms and a corrected QT interval of 485ms. He was gradually weaned off the pressor support and extubated 36 hours after the admission. A subsequent EKG done 36 hours after admission showed sinus rhythm in the 70 bpm, a normalized QTc interval, and a narrow QRS complex.

Discussion

We describe a patient who developed serious cardiac and central nervous system toxicity primarily due to drug interactions between propafenone and mirtazapine.

One of the most important systems involved in metabolic drug interactions, especially with antidepressants, is the enzyme system comprising cytochrome P450 oxidase. Of the cytochrome P450 systems, CYP2D6 has received the most attention.2 Typical substrates for CYP2D6 are largely lipophilic bases including the majority of antidepressants, antipsychotics, antiarrhythmics, antiemetics, β-adrenoceptor antagonists (β-blockers) and opioids. The CYP2D6 enzyme activity varies significantly within a given population and depending upon the rate of drug metabolism, the population can be divided into ultra-rapid metabolizers (UMs), extensive metabolizers (EMs), intermediate metabolizers (IMs) and poor metabolizers (PMs). There is a considerable variability in the CYP2D6 allele distribution among different ethnic groups, resulting in variable percentages of PMs, IMs, EMs and UMs in a given population.3 Approximately 5 to 10 percent of Caucasians and up to 2 percent of persons of Asian and African descent lack this enzyme as a result of an autosomal recessive inherited defect. There are nine mutant forms of the CYP2D6 gene which are either inactive or poorly active.4 Individuals who are poor metabolizers exhibit greater bioavailability, greater plasma concentrations, prolonged elimination half-lives, and possibly exaggerated pharmacologic response from standard doses of drugs that are metabolized by CYP2D6 (Table 1).3,4 The human genome project has enabled gene analysis to determine individual variations to drug response and metabolism.5 This has ushered in an era of “personalized medicine,” based on genetic profiling with
availability of customized medical treatment according to the individual's genome. Gene analysis has enabled differentiating polymorphisms in the CY2D6 gene to select rapid and slow metabolizers. Therefore, based on an individual's drug metabolizing CY2D6 genetic machinery, a rapid metabolizing person is prescribed a higher dose and vice-versa, unlike the "average metabolizer" dosing of a drug by a pharmacy company.

Propafenone is a type IC antiarrhythmic agent that also has weak beta-blocking characteristics; it blocks sodium channels in both the activated and inactivated state. Metabolism of propafenone is genetically determined using the CYP2D6 enzyme system. About 10 percent of patients are PMs with a prolonged half-life of the parent compound. The half-life of the parent compound ranges from two to 12 hours, with a mean in the six hour range. The major metabolites of propafenone are 5-hydroxypropafenone (active) and N-debutyl propafenone. In vitro studies have shown that 5-hydroxypropafenone is at least as effective as a sodium channel blocking agent as the parent drug, whereas the N-desalkyl metabolite is less potent. As a beta-blocker, however, 5-hydroxypropafenone is less potent than the parent drug in animal studies. PMs are at an increased risk of developing central nervous system side effects while receiving propafenone, presumably due to the high plasma concentrations achieved in these patients. Also, the high propafenone concentrations attained in PMs might lead to clinically evident beta-blockade. It is possible that at the high plasma concentrations achieved in PMs, clinically important beta-adrenergic blockade will be observed that could contribute to both adverse reactions and antiarrhythmic efficacy.

Mirtazapine is a noradrenergic and specific serotonergic antidepressant (NaSSA), which is used for major depression, dysthymia, post-traumatic stress disorder and insomnia. It is primarily metabolized by the liver. The half life is 20-40 hours with time to peak concentration being close to two hours. There is relatively little data available on the effects of mirtazapine on the cytochrome P450 system and its potential to interact with other drugs in vivo. Although in vitro studies do not demonstrate inhibitory effects, mirtazapine is a substrate for CYP2D6, CYP1A2 and CYP3A4. It is a mild competitive inhibitor of CYP2D6. Thus in this case, administration of mirtazapine probably led to inhibition of CYP2D6 with a resultant increase in propafenone levels.

We propose that our patient was a poor metabolizer of propafenone, with a higher drug concentration and increased half-life of propafenone at baseline as compared to an extensive metabolizer. Mirtazapine administration led to competitive inhibition of CYP2D6, which further increased the level of propafenone by inhibiting the conversion of propafenone to its metabolite. The higher concentration led to severe toxicity manifested by seizures, bradycardia, prolonged QRS interval, prolonged QTc interval and RBBB. An alternative hypothesis of propafenone - mirtazapine interaction leading to elevated mirtazapine plasma levels – attributing to the patient's presentation was considered. A literature search revealed that significant side effects associated with mirtazapine were mainly somnolence, increased appetite, weight gain and rarely agranulocytosis. The drug was found to be non-toxic even at 30-50 times the standard dose. Reported side effects for mirtazapine do not include cardiac arrhythmia and seizures.

The diverse nature of the CYP2D6 enzyme activity responsible for propafenone metabolism necessitates cautious clinical use. The time to steady-state (optimal intervals at which efficacy can be assessed and dosage adjustments made) will vary in patients of different phenotypes. The dose increment should be gradual to avoid disproportionate rise in the plasma concentration due to saturation of the involved cytochrome P-450 isoenzymes. Last but not least, extreme caution is needed when instituting drugs for concomitant use with propafenone that are substrates for or inhibit the cytochrome 2D6 system, as they can inadvertently increase serum levels of propafenone and cause toxicity.

To our knowledge this is the first described case of drug interaction between propafenone and mirtazapine in the medical literature.

**References**


Please note: Due to limited space, we are unable to list all references. You may contact South Dakota Medicine at 605.336.1965 for a complete listing.

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Tomasz P. Stys, MD, Sanford School of Medicine of the University of South Dakota; Sanford Heart Hospital, Sioux Falls.
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Remember... AA&R Ask. Advise. Refer.
Identification of a Founder Mutation for Maple Syrup Urine Disease in Hutterites

By Amelia Mroch, MS, CGC; Laura Davis-Keppen, MD; Cindy Matthes; and Quinn Stein, MS, CGC

Abstract

Maple syrup urine disease (MSUD) is an organic acidemia detected on newborn screening. The condition has been reported with increased frequency in certain founder populations including Hutterites. We present a case of MSUD in a Hutterite boy. Mutation analysis was completed and identified a candidate founder mutation in the BCKDHB gene, specifically c.595_596delAG. Further testing of other Hutterites with MSUD is needed to determine whether additional mutations may exist.

Introduction

Maple syrup urine disease (MSUD) is an organic acidemia characterized by elevated concentrations of branched-chain amino acids (isoleucine, leucine and valine). Classic MSUD has its onset in the neonatal period with a noticeable maple syrup odor of cerumen and urine followed by poor feeding and lethargy. Progression to coma and respiratory failure can occur if left untreated.

As with other genetic conditions, founder mutations for MSUD are recognized and have been reported in several populations worldwide. Perhaps the most studied is that in the Mennonite population where the reported carrier frequency for MSUD is one in 10. Other proposed communities with founder effects for MSUD include the Portuguese Gypsies and Filipinos.

In South Dakota, the Mennonite population is quite small; however, the Hutterite religious cultural group is more populous. Hutterites are Anabaptists who emerged as a defined group in Austria in the 16th century. During the 1800s, 443 Hutterite individuals traveled from Europe to the plains of North America and established three colonies, or leuts: Schmiedeleut, Dariusleut and Lehrerleut. Over the last century, the number of Hutterites has grown to more than 40,000 throughout the Northern Great Plains of the U.S. and Canada.

Because they continue to live in reproductive isolation, there are more than 30 autosomal recessive syndromes documented to occur more frequently in the Hutterite population, including MSUD. Examples of other conditions include cystic fibrosis, Bowen-Conradi syndrome, cerebro-oste-nephro dysplasia, dilated cardiomyopathy with ataxia, Joubert syndrome-related disorder and limb girdle muscular dystrophy subtype 2H.

Specific Hutterite founder mutations have been identified in about half of these conditions. For example, a founder mutation in the CFTR gene known as M1101K has been found primarily in the Hutterite population. The founder mutation for MSUD, however, has not been reported to date. We document a case of MSUD in a Hutterite child with identification of a potential founder mutation.

Case Report

A Hutterite child presented to our facility on day 4 of life when he was identified as having MSUD on newborn screen (Table 1). On admission, the baby was described as “sleepy” and “lazy with feedings.” His urine was positive for ketones. At that time, he was started on formula specific to the treatment of MSUD.

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<th>Table 1. Newborn Screen and Follow-up Laboratory Values</th>
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<td>Leucine</td>
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At 3 months of age, his ability to consume full formula feedings had decreased and a gastrostomy tube was placed. At 29 months, he continued to receive the bulk of his nutrition through his tube. Development has been in the normal range up to this point. He walked at 13 months and had about five words at 18 months.

At 6 months of age, genetic testing was discussed and the family opted to proceed. Testing was performed in a clinical laboratory and identified a homozygous variant in BCKDHB defined as c.595_596delAG (p.Pro200Stop).

Family history includes several distant relatives with MSUD. Parents are double third cousins (Figure 1).

Discussion

There are three recognized genes that form the branched-chain alpha-ketoacid dehydrogenase complex: BCKDHA, BCKDHB and DBT. There is also a fourth gene, DLD, that interacts with the complex. Mutations in this gene result in a different phenotype from classic MSUD. It is not possible to biochemically distinguish which of the three genes contains the mutation leading to the disease.

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<thead>
<tr>
<th>Type</th>
<th>Gene</th>
<th>Chromosomal locus</th>
<th>Proportion of MSUD</th>
</tr>
</thead>
<tbody>
<tr>
<td>MSUD 1A</td>
<td>BCKDHA</td>
<td>19q13.2</td>
<td>45%</td>
</tr>
<tr>
<td>MSUD 1B</td>
<td>BCKDHB</td>
<td>6q14.1</td>
<td>35%</td>
</tr>
<tr>
<td>MSUD 2</td>
<td>DBT</td>
<td>1p21.2</td>
<td>20%</td>
</tr>
</tbody>
</table>

MSUD is termed classic or intermediate based on severity, which depends on residual branched chain ketoacid dehydrogenase activity. Those with classic MSUD present within the first week of life and have 0 to 2 percent enzyme activity, while those with intermediate MSUD have a variable age of onset and 3 to 30 percent enzyme activity. Rarely, affected individuals with decreased branched chain ketoacid dehydrogenase activity have intermittent symptoms or respond to thiamine. Infants with classic MSUD are identified by newborn screening, but intermediate forms of the disease may not be diagnosed until later.

Diet treatment should be started immediately upon an infant having an abnormal newborn screen result. Adequate calories must be provided to prevent catabolism. Breastmilk or standard infant formula must be discontinued and replaced with a medical formula free of the branch chain amino acids (BCAA). Serum levels of BCAA must be obtained frequently so that the formula mixture can be adjusted to keep levels in the therapeutic range. People with MSUD need to follow a life-long restricted diet that is very low in natural protein sources in order to limit intake of BCAA. Specialty low protein grain items (bread, pasta, baking mix) need to be consumed in addition to fruits and vegetables. Medical formula is also consumed life-long, and serves as the primary source of protein, contributing all of the other amino acids except BCAA.

With early diagnosis, individuals with MSUD can have normal growth and development. This requires regular and ongoing adjustment of therapy based on metabolic demands. Metabolic decompensation can occur with common illnesses, leading to neurologic deterioration. Because of the frequent monitoring and necessity of hospitalizations, it is understandable that a family would have an interest in genetic and carrier testing for classic MSUD.

In the child presented here, a reflexive approach was used for analysis of the DNA, with the mutation ultimately being found in the BCKDHB gene that causes MSUD type 1b. The Pro200Stop mutation is a recognized pathogenic change. In 2003, Henneke et al. reported this same mutation in a Spanish, as well as an Austrian, family. To our knowledge, no report exists documenting molecular testing for MSUD in the Hutterites.

Of interest, the Hutterites were originally founded in 1528 in a region called South Tyrol which was, at the time, a part of Austria. Shortly after, they fled this region, began communal living, and lived in reproductive isolation. There were several moves around Europe before migrating to the U.S. in the 1870s, but they continued to live in reproductive isolation. For this reason, it is thought that all North American Hutterities have descended from the original Hutterite individuals arising out of Austria. This scenario illustrates beautifully the population genetics concept of a founder mutation — a mutation that exists in an individual who then goes on to reproduce in a closed environment thereby passing on the mutation to a much higher frequency of that population than would be expected based on general population calculations.

The common ethnic origins could explain why the same mutation has been seen in both the general population of Austria and in the Hutterites of North America. The reproductive isolation would explain why the Hutterites have only one known mutation which has been passed on from generation to generation. Hutterites with MSUD are
likely to be homozygous for the mutation, while the Austrian family reported in Henneke et al. carried the Pro200Stop mutation in addition to a second, different mutation in those affected with MSUD.9

Beyond identification of the mutation in this child, an additional purpose in pursing testing was to address the family interest in carrier testing. At this time, carrier testing has been facilitated for four family members or their spouses (Figure 1).

After detecting the molecular cause of MSUD in the child described above, subsequent testing of an additional male with classic MSUD, a 21-year-old from a different Hutterite colony, detected the same BCKDHB c.595_596delAG homozygous mutation.

Knowledge of this mutation has the potential to allow for more widespread carrier testing in this population. Further testing of those Hutterites with MSUD is needed to ensure that there are no other sequence changes in their population; however, this result holds promise as a founder mutation in Hutterites.

Acknowledgments
We would like to thank the family for their interest in sharing this information with the medical community as well as for pursuing genetic testing to allow for carrier testing in more distant relatives.

REFERENCES


Please note: Due to limited space, we are unable to list all references. You may contact South Dakota Medicine at 605.336.1965 for a complete listing.

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Incidental Finding of Subclavian Steal in a Cardiac Catheterization for Acute Coronary Syndrome

By Noah Wiedel, MD, MS; Adam T. Stys, MD; and Tomasz P. Stys, MD

Abstract
The subclavian steal syndrome (SSS) refers to neurological symptoms that develop from a proximal subclavian artery occlusion. We present a case of an asymptomatic patient found to have subclavian steal (SS) on angiography. A brief literature review follows.

Introduction
Subclavian steal syndrome (SSS) results from retrograde blood flow in the vertebral artery that comes off distal to an occlusion or narrowing of the subclavian artery. Normally, the blood supply to the subclavian artery comes from the aorta. In the event of an occlusion to the proximal subclavian artery, flow to the distal part of the subclavian artery is supplied via the vertebral artery (Figure 1). Usually in such a case, there are no symptoms; however, with increased blood flow (for instance, during exercise of the arm), the blood flow diverted to the subclavian artery can result in inadequate perfusion of the CNS, resulting in SSS. Then symptoms include neurologic deficits ranging from presyncopy to memory impairment. Subclavian steal may also manifest itself as exercise intolerance in the ipsilateral upper extremity. Occlusion of the proximal subclavian artery is most commonly due to atherosclerosis, and more rarely Takayasu arteritis, thoracic outlet compression, or as a complication following repair of aortic coarctation. Differences in blood pressure in the upper extremities typically are present. We describe a case of asymptomatic subclavian steal in a man presenting with angina pectoris, resulting in a change of access site for coronary catheterization.

Case Report
A 62-year-old male presented with typical worsening exertional angina pectoris. Past medical history was significant for stenting of the right coronary artery six years ago, hyperlipidemia, and bilateral pulmonary emboli. His medications included simvastatin, cyclobenzaprine, clopidogrel, omeprazole, nebivolol, aspirin, nitroglycerine patch. Social history was significant for a 135 pack year smoking history. Blood pressure was 96/61 in the right arm, 125/76 in the left arm and heart rate was 77. The rest of cardiovascular exam was unremarkable. ECG showed sinus rhythm with nonspecific T wave changes.

Cardiac catheterization was attempted via right radial artery as the Allen’s test showed adequate dual blood supply to the hand and our lab is set up primarily for right sided access; however, the proximal right subclavian artery was completely occluded (Figure 2). A radiolucent jet originating at the right vertebral artery ostium was seen.
with the background of injected radiopaque contrast in the subclavian artery (Figure 3 and 4), consistent with flow reversal in the vertebral artery (SS). As the patient was neurologically asymptomatic, no subclavian artery revascularization was deemed necessary.

The coronary angiography was completed via right femoral artery access. The patient was found to have a total chronic occlusion of right coronary artery with a trial of medical management instituted.

Discussion

SSS refers to diverse neurological deficits resulting from the above described blood flow pattern. These include presyncope, syncope, disequilibrium, hemianopia and other visual disturbances. On physical exam, pulsus parvus et tardus may be found, as well as difference in blood pressure on upper extremities. Ultrasonography, computed tomography angiography, magnetic resonance angiography and classical angiography can be used to confirm the diagnosis. SS is most frequently asymptomatic and is suggested by a 20 mmHg difference of blood pressure in left and right brachial artery.

In our case, as the Allen’s test was satisfactory, the attempt at cardiac catheterization via right radial artery provided the diagnosis of SS. Our lab is set up for primary right radial access, though it can switch to the left side without a problem. The added value of the peripheral disease information in such a case may make the operator choose this approach. The decision on proceeding with femoral artery access next versus contralateral radial artery approach depends on operator’s preference.

There are no specific screening guidelines for SS, but left versus right brachial BP differences discovered in a thorough physical examination help with the diagnosis. Finding SS does not change management of other cardiovascular diseases, except for the access for cardiovascular procedures. In SSS with significant symptomatology, a referral for revascularization is reasonable. These patients can benefit from percutaneous or surgical intervention to alleviate symptoms.3
A diagnosis of subclavian steal is associated with increased mortality, typically from cardiovascular events, rather than neurological sequelae. Atheromatous disease is most commonly the etiology and its occurrence elsewhere is common. As such, a finding of asymptomatic subclavian steal syndrome may lead practitioners to prioritize cholesterol goals. Patients with severe, symptomatic subclavian steal may undergo percutaneous or surgical intervention to alleviate symptoms. 

REFERENCES


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Cystic Fibrosis (CF) is an autosomal recessive life-limiting disease affecting approximately 30,000 individuals in the U.S. and 70,000 worldwide. The underlying defect in CF is due to mutations in the cystic fibrosis transmembrane conductance regulator (CFTR) gene, which is responsible for encoding the CFTR protein found in the apical membrane of the epithelial cells. The CFTR protein transports sodium across membranes and works in conjunction with the epithelial sodium channels to maintain salt, fluid, and pH homeostasis in various epithelial membranes. In the lungs, the dysfunction of CFTR protein leads to dehydrated airway surface liquid and impaired mucociliary clearance resulting in a continuous cycle of infection, inflammation, and progressive lung disease. The most common bacterial pathogen is *Pseudomonas aeruginosa*, which eventually results in long-term infection. Other common bacteria include methicillin sensitive *Staphylococcus aureus* (MSSA), methicillin resistant *Staphylococcus aureus* (MRSA), *Haemophilus influenzae* and, less frequently, *Stenotrophomonas maltophilia*, *Achromobacter species*, and *Burkholderia cepacia*. Pancreatic insufficiency results from ductal mucosal obstruction with dehydrated secretions leading to chronic fibrosis and/or fatty replacement of the gland, resulting in little to no release of digestive enzymes. This leads to fat malabsorption, fat-soluble vitamin deficiency and poor growth if left untreated. The CFTR protein is also found in other areas of the body, but clinical manifestations are primarily seen as a result of CFTR dysfunction in the lungs, exocrine pancreas, gastrointestinal tract, sweat glands and vas deferens.

CF is further complicated by other comorbid conditions including, but not limited to, gastroesophageal reflux disease (30 percent), sinus disease (30 percent), asthma (25 percent), cystic fibrosis related diabetes (20 percent), osteoporosis (15 percent), depression (12 percent), and liver disease (10 percent). Treatment of CF includes addressing all aspects of disease; however, this article will primarily focus on the pharmacologic treatment of pancreatic insufficiency and maintenance treatment for pulmonary disease.

**Diagnosis and Epidemiology**

CF has an incidence of approximately one in every 3,000 live births with a carrier rate of one in 25 Caucasians of northern European decent. Newborn screening for CF is performed in all 50 states. South Dakota began routine newborn screening in 2006. Diagnosis of CF is confirmed by sweat chloride testing or measurement of nasal potential difference and should be performed only at accredited CF centers. With earlier diagnosis and the development of new treatment options, the average life expectancy has increased dramatically over the last several decades, reaching a predicted median survival of 41.1 years based on the 2012 Cystic Fibrosis Foundation (CFF) registry data (Figure 1). There are approximately 110 CF patients seen at the South Dakota CF center at Sanford USD Medical Center and Sanford Children’s Hospital.

**Genetics**

More than 1,900 mutations of the CFTR gene have been identified. However, the most common mutation is F508del with homozygotes and heterozygotes accounting for 87 percent and 47 percent of CF patients, respectively.
The most common mutations have been classified into five categories based on type of CFTR dysfunction (Table 1). In some cases the CFTR protein is completely absent, which typically correlates with the most severe form of the disease; whereas mutations that result in some residual CFTR function may be associated with less severe expression of CF. When mutations from two different classes are present, disease severity is very unpredictable. Within our state, the second most common mutation is M1101K. This mutation is unique to the Hutterite population and is in the same class as F508del (Class 2). Of note, over 10 percent of CF patients in South Dakota carry at least one copy of M1101K.

**Pancreatic Enzyme Replacement**

Approximately 90 percent of patients with CF have pancreatic insufficiency which requires treatment with pancreatic enzymes, supplemental vitamins and high-fat/high-calorie dietary intake. Current Food and Drug Administration (FDA)-approved pancreatic enzyme products are porcine derived and include a combination of lipase, proteases and amylase. Dosing is either weight-based in older children or adults or based on units of lipase per gram of fat for those infants and younger children who are formula or breast fed. Dose adjustments are based on symptoms of malabsorption which may include oily, loose, frequent or very foul smelling stools or a lack of weight gain. All enzyme products are labeled with the enzyme manufacturer’s proprietary name and a number that indicates lipase component. The majority of brands available come as capsules containing small enteric-coated spheres that prevent the enzymes from immediately interacting with oral mucosa and instead are activated in the duodenum when the pH is greater than 5.5. The capsules can be swallowed whole or opened with the beads sprinkled in food that do not have too high of a pH, such as applesauce, in order to prevent release of the active component prematurely, and should be taken with meals and snacks. The FDA-mandated pancreatic enzyme manufacturers to submit new NDAs by 2010 to prove safety and efficacy, resulting in a change in product availability and the elimination of generic enzymes. Current approved brands include Creon, Pancrease, Pertzye, Ultresa, Zenpep and Viokace. It is important to note that Viokace tablets are not enteric coated. It is also important to note that enzymes are not AB rated so they cannot be automatically substituted. In addition, acid suppressing agents may be added to help maximize enzyme efficacy due by preventing premature acid degradation. Adverse effects due to pancreatic enzymes are rare, however, due to purine content in enzymes, hyperuricemia or hyperuricuria may occur. Additionally, there have been rare reports of fibrosing colonopathy and colonic strictures attributed to long-term high dose enzyme therapy (greater than 2500 units/kg/dose).

**Vitamin Supplementation**

Fat-soluble vitamin supplementation is frequently needed to maintain adequate levels of vitamins A, D, E and K. There are CF specific vitamin formulations available which contain higher amounts of the fat-soluble vitamins compared to traditional multivitamins. These products are supplied as softgels, chewable tablets or drops and include AquADEKs, Vitamax, Libertas ABDEK Multivitamin and MVW Complete Formulation Multivitamin. Adjustments in dose are based on serologic measurement of vitamin levels. Additional high dose supplementation of individual fat-soluble vitamins may be required to achieve normal vitamin levels.

### Table 1. Classification of Mutations

<table>
<thead>
<tr>
<th>Class</th>
<th>Mutation Effect</th>
<th>Percent of pts&lt;sup&gt;a&lt;/sup&gt;</th>
<th>CFTR Expression</th>
<th>Channel Function</th>
<th>Common Mutations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td><strong>Protein synthesis defect</strong>: CFTR is not made (nonsense mutations)</td>
<td>10%</td>
<td>Little to none</td>
<td>Little to none</td>
<td>W128X G542x R553X R1162X</td>
</tr>
<tr>
<td>2</td>
<td><strong>Folding/Trafficking defect</strong>: CFTR is made but not “folded” correctly or is not “trafficked” to surface</td>
<td>88%</td>
<td>Little to none</td>
<td>Little to none</td>
<td>F508del N1303K I507del M1101K</td>
</tr>
<tr>
<td>3</td>
<td><strong>Gating defect</strong>: CFTR is present but not active</td>
<td>4%</td>
<td>Normal quantity</td>
<td>Little to none</td>
<td>G551D G551S S1255P S1251N</td>
</tr>
<tr>
<td>4</td>
<td><strong>Narrow channel</strong>: CFTR is present but reduced function</td>
<td>&lt;2%</td>
<td>Normal quantity</td>
<td>Some Cl- activity</td>
<td>R117 R347P R334W</td>
</tr>
<tr>
<td>5</td>
<td><strong>Splicing defect</strong>: CFTR production is variable</td>
<td>Rare</td>
<td>Reduced quantity</td>
<td>Normal Cl- activity</td>
<td>3120+G A 3849+10kbC T</td>
</tr>
</tbody>
</table>

<sup>a</sup> Percent of patients with at least one allele from that class.

---

Pharmacology Focus
**Pharmacology Focus**

**Pulmonary Therapies (Tables 2 and 3)**

Multiple modalities are used to treat the viscous mucus, bacterial burden and inflammation in the airways. Educating patients on appropriate cleaning of nebulizer equipment, storage and handling of medications and order of administration is important. Inhaled medications should be administered in the following order: 1) bronchodilators, 2) hypertonic saline, 3) dornase alfa, and 4) inhaled antibiotics. Additionally, a form of airway clearance is recommended for all patients with pulmonary disease and may include high frequency vest therapy, chest physiotherapy or other devices such as the Acapella device.

**Bronchodilators**

In vitro studies suggest bronchodilators may also increase ciliary beat frequency and stabilize mast cells. Despite these effects, there is no robust data to suggest bronchodilators result in significant long-term improvements in lung function or delay disease progression. The 2013 CF guidelines for chronic medications, published by Mogayzel and colleagues, do not recommend for or against long-term use. However, bronchodilators continue to be prescribed routinely and may help with tolerance of other inhaled therapies.

**Mucus Thinners**

Medications targeting mucus viscosity are a mainstay of CF treatment and include dornase alfa and/or hypertonic saline. N-acetylcysteine (Mucolyst) was widely used for decades prior to the introduction of dornase alfa in 1993, however, studies have failed to demonstrate benefit and it is no longer recommended.

Recombinant human deoxyribonuclease alfa (Pulmozyme) or dornase alfa thins mucus by cleaving extracellular DNA.
left behind by lysed neutrophils in the airways. Dornase alfa is approved for all patients 6 years and older; however, safety and efficacy data for use in infants and children exists. There is little guidance as to when dornase alfa should be initiated; however, presentation with symptoms such as cough, frequent respiratory infections or other evidence of pulmonary congestion often provokes initiation of this treatment at an early age. Dornase alfa has been reported to result in statistically significant improvement in FEV$_1$ with relative increases of 5.8 to 7.3 percent in those with moderate disease and reductions in pulmonary exacerbations in those with mild disease. Dornase alfa is well tolerated with voice alteration being the most common reported side effect. 

Sodium chloride 7 percent solution (HyperSal) is a hypertonic saline that draws fluid into the airway surface liquid and decreases viscosity. Additionally, patients often cough with this treatment which may provide a mechanical loosening of airway mucus. While hypertonic saline is not FDA labeled for CF, it is recommended for patients 6 years of age and older. In addition, safety data exists for administration of this drug in infants and children. In clinical studies, statistically significant relative increases in FEV$_1$ predicted of 4 to 8 percent have been reported. While results from comparator studies between dornase alfa and hypertonic saline have demonstrated mixed results, dornase alfa has exhibited more robust data supporting its use. 

Inhaled Antimicrobials

Currently, P. aeruginosa is the only bacterium which warrants eradication and chronic suppression therapy based on published literature. FDA-approved inhaled antibiotics include tobramycin inhalation solution (TIS; TOBI, Bethkis), tobramycin inhalation powder (TIP; TOBI Podhaler), and aztreonam lysine inhalation solution (AZLI; Cyston). Treatment with inhaled anti-pseudomonal medication is indicated in patients with established P. aeruginosa infection regardless of disease severity. Other inhaled antibiotics such as colistimethate sodium are sometimes utilized, but, due to the lack of compelling data, are not recommended. Inhaled antibiotics achieve extremely high sputum concentrations that are not safely attainable with systemic antibiotic administration. One caveat to this is the presence of mucus plugging in the airways, which would prevent inhaled antibiotics from reaching all targeted areas. Long-term studies have not demonstrated significant risk for antibiotic resistance.

Clinical trials of TIS performed in patients with moderate to severe disease have reported statistically significant increases in FEV$_1$ of 8 to 12 percent relative to baseline. Regardless of disease severity, statistically significant reduction in pulmonary exacerbations results from chronic TIS therapy in patients with P. aeruginosa. Additionally, TIS has been proven effective for eradication of first time P. aeruginosa culture in CF patients when administered twice daily for a minimum of 28 days. TOBI Podhaler and Bethkis were approved in 2013 based on non-inferiority and pharmacokinetic studies compared to TOBI brand TIS. AZLI was approved in 2010, 13 years after TIS had been FDA approved. The AZLI studies primarily included patients who had previously been on chronic TIS therapy which, in theory, may have blunted treatment effect; however, statistically significant improvements in FEV$_1$ were still observed (6.3 to 10.3 percent) along with statistically significant reductions in pulmonary exacerbations. Additionally, a single 18 month comparator trial reported a statistically significant lower exacerbation rate and improved lung function in the AZLI group compared to TIS. The standard dosing regimen for inhaled antibiotics includes 28 days on drug followed by 28 days off in a rotating fashion. The effect of continuous administration of inhaled antibiotics with 28 days of TIS/TIP followed by 28 days of AZLI in a rotating fashion, eliminating an “off” period is being evaluated. This method is already employed in many patients who require frequent antibiotics or have declining lung function. The most common side effects reported with TIS include sputum discoloration, voice alteration, cough, and tinnitus. Adverse effects with TIP are similar but include a higher incidence of cough. Side effects of AZLI may include cough, wheezing, nasal congestion, bronchospasm and rash.

Anti-inflammatory Agents

Airway inflammation is a component of CF lung disease and various medications have been studied to address this aspect of CF with little success. Oral steroids have been proven beneficial but the risks outweighed the benefits. Inhaled steroids and leukotriene receptor antagonists have failed to provide benefit in the absence of a reactive airway disease, and various alternative supplements have failed to produce significant changes. Only two agents, azithromycin and high-dose ibuprofen, have evidence to suggest benefit.

Azithromycin is thought to elicit its effects through reduction of interleukins, tumor necrosis factor, and neutrophil production and oxidative bursts; and through inhibition of alginate production, which is instrumental
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in *P. aeruginosa* biofilm formation. Overall, small yet statistically significant improvements in FEV₁ (3.6 to 6.2 percent) have been reported along with statistically significant reductions in antibiotic courses, with the greatest benefit observed in those with established *P. aeruginosa*. Prior to initiating therapy it is important to rule out the presence of atypical mycobacterium, *Burkholderia cepacia* complex and liver disease or elevated liver function tests. The most studied dose is 250 mg for patients weighing less than 40 kg and 500 mg three times weekly for those with weights greater than 40 kg. The most common adverse effects reported were gastrointestinal.

Despite CF guidelines recommending high-dose ibuprofen (20 to 30 mg/kg/dose twice daily), less than 6 percent of patients meeting criteria are prescribed this therapy. The proposed mechanism is inhibition of neutrophil migration, adherence and release of lysosomal enzymes. Close examination of the literature reveals benefit was observed only in a small sub-group of patients (n=24) between the ages of 6 to 13 in the largest trial by Konstan, et al. Subsequent studies were small and retrospective in nature, and not all studies demonstrated benefit. Greater than 50 percent of patients who start high-dose ibuprofen stop due to adverse effects. Though uncommon, major gastrointestinal bleeding, overt hemoptysis and acute renal failure have been reported. Ibuprofen plasma levels must be measured and maintained between 50 to 100 mcg/ml as concentrations below this range may actually exacerbate pulmonary inflammation via increasing neutrophil influx. Adverse effects may include upset stomach, increased risk for gastrointestinal bleeding, tinnitus and renal toxicity.

**CFTR Modifiers**

Ivacaftor (Kalydeco) is the first drug to target the underlying cause of CF and was FDA approved in December 2012 for CF patients with a G551D mutation. In February 2014, ivacaftor received expanded approval to include eight additional class 3 mutations. Ivacaftor potentiates the CFTR protein on the surface of epithelial cells, resulting in increased chloride ion transport in patients with class 3 gating mutations which account for only 3 to 4 percent of CF patients. In patients with one G551D mutation, ivacaftor increased FEV₁ percent predicted from baseline by 18 to 21 percent, higher than any other medication studied to date. Additional outcomes included statistically significant reductions in exacerbations, increases in weight, improvements in quality of life scores and drastic reductions in sweat chloride concentration. Patients must have one of the specified class 3 mutations and be 6 years of age or older (Table 3). Adverse effects include headache, cough and liver function test evaluations. Ivacaftor is primarily metabolized by CYP3A. Dose adjustment is necessary when combined with CYP3A inhibitors, and CYP3A inducers can substantially decrease ivacaftor levels. Patients are instructed to avoid grapefruit juice and Seville oranges, which are CYP3A inhibitors, and St. John’s Wort which is a CYP3A inducer. It is important for providers to notify the patient’s CF care team when initiating new prescription medications that could interact with ivacaftor.

**Research Pipeline**

Though a cure may not be found in the near future, the introduction of medications targeting the CFTR protein is a major advancement. Currently, there are 12 promising agents in clinical trial phases ranging from inhaled antibiotics to CFTR modifiers for those with other mutations. Ongoing studies are also evaluating faster delivery devices, optimal treatment strategies, methods for improving adherence, and impact of various nutrition and airway clearance approaches.

**Conclusion**

CF is a very complex disease involving multiple organ systems requiring complex treatment regimens tailored to each patient’s disease severity, airway microbiome and comorbid disease states. Interdisciplinary care and communication amongst all care providers are required to ensure optimal care is provided to patients.

**REFERENCES**

9. Cystic fibrosis patient registry South Dakota Center. Please note: Due to limited space, we are unable to list all references. You may contact South Dakota Medicine at 605.336.1965 for a complete listing.

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The belief in a right to patient privacy is almost as old as the medical profession itself. Early physicians in the fourth century B.C., following the teaching of Hippocrates, enshrined this value in the Hippocratic Oath: “Whatever I see or hear in the lives of my patients, whether in connection with my professional practice or not, which ought not to be spoken outside, I will keep secret, as considering all such things to be private.”

Respected through the centuries, this ancient ethic is now embodied in the mandates of HIPAA Privacy, which extends itself into the electronic age through HIPAA Security.

Securing protected health information (PHI), such as the PHI created, transmitted and maintained through electronic health records (EHRs), requires heightened awareness and diligence – not only to protect patient confidentiality, but also to protect your organization from steep penalties resulting from breach. The following sections describe how physicians can best support efforts to protect PHI.

Data on the Move

PHI physically on the move is greatly vulnerable to breach by theft or loss. Theft and loss of PHI is the number one reason for breach in America. Pay attention to when and how you carry high volumes of patient information, whether paper or electronic – laptops, boxes of charts, data backups, thumb drives, patient schedules, reports and even smart phones – then make sure to implement appropriate safeguards and use common sense. Treat PHI as a valuable possession and keep it out of sight, even when in a locked vehicle. Make a habit of storing PHI in your trunk before reaching your destination. Don’t leave your laptop bag unattended in an airport or other public area. And remember, if you use encryption and a strong password for your electronic device, you may be protected by safe harbor and creates a high risk of breach if lost or stolen.

Enforce Unique User IDs and Strong Passwords

The second-highest cause for breach is unauthorized access or disclosure. While often resisted, two very basic protections can guard against unauthorized access or disclosure: 1) the enforcement of unique user identifications (IDs) and 2) the use of strong passwords.

Of note, your information and activities within an EHR are subject to discovery during litigation. If you share user IDs or passwords, the integrity of your documentation for patient care may be challenged and declared inaccurate. So never share your user ID or password, period.

Smart Phones and Bring-Your-Own-Devices (BYODs)

Adoption of mobile devices such as iPhones, iPads, BlackBerrys and Android devices is fast-growing among health care providers. Smart phones are wonderfully convenient tools for communicating with staff and reviewing schedules, patient summaries and other clinical references.

While allowing privately-owned devices to be governed by organizational policy can feel vulnerable and invasive, it is the HIPAA-covered entity’s obligation to create safeguards for PHI within its protection, regardless of whether PHI is accessed by a personally-owned or organization-owned device.

Be aware and plan accordingly for managing common risks associated with mobile devices:

Authentication – Authentication methods for mobile devices, especially unmanaged ones, are subject to user discretion, including the ability to disable PINs or passwords. In the absence of authentication, anyone in possession of the device can potentially access PHI stored within or access it through an app with saved credentials, such as email.

Encryption – Mobile devices are not encrypted by default. Even when screens are locked with a PIN or password, unencrypted PHI stored on the device is not protected by safe harbor and creates a high risk of breach if lost or stolen.

Wi-Fi Connections – Without the proper safeguards, Wi-Fi connections leave devices vulnerable to others also connected to the network. This is especially true of public Wi-Fi networks where cyber thieves may access data on your device without your knowledge.

Loss and Theft – Mobile devices are particularly vulnerable to loss and theft because of size and portability.

A solid approach to managing risk surrounding mobile devices is to start with a written and enforceable policy that addresses both personally-owned and entity-owned devices. Include guidelines such as acceptable use of devices, security procedures, reporting processes for loss or theft and steps necessary to wipe confidential data before changing ownership. Consider technology controls such as remote wipe capabilities and location functions. Enforce use of PINs or passwords for screen locks, and encrypt as necessary.

This article includes material originally published by MMIC. MMIC is the largest policyholder-owned medical liability insurance company in the Midwest and is always thinking ahead to find ways to protect clients through risk financing, improving patient safety and physician well-being, and reducing the risks associated with information technology.
Much has been written about the estate tax law changes that provide higher exemption amounts and portability. For most people, estate planning has been greatly simplified, but in several situations, planning issues remain.

Current law imposes estate tax on wealth transfer exceeding a person’s “exemption amount.” Portability allows a widowed spouse access to any unused exemption amount belonging to his or her deceased spouse. If a widow(er) remarries, however, this exemption is lost. So, if Steve and Emily are married and Emily dies, she can leave $5.34 million free of estate tax. Any unused exemption would be available for Steve to shelter gifts he makes during his lifetime or at his passing. If Steve later meets Julie and is thinking of remarriage, he might not realize the significant planning opportunity. Tax law limits portability to his most recent spouse. Upon his marriage to Julie, Steve will forfeit access to Emily’s unused exemption. If Steve has a large estate ($5-6 million), he should strongly consider gifting property before the wedding.

Portability requires the first spouse to die to make an election by using IRS form 706. Historically, few estates filed an IRS form because it is time consuming and expensive. Consider a second marriage where the couple keeps their finances separate and plans for their respective children to inherit their wealth. In situations where one, but not both, of the spouses is wealthy, the portability election is tremendously valuable to the wealthy side, but has no value and is only an expensive nuisance to the other side. If the less-wealthy spouse passes away first, the challenge becomes convincing the estate to make the portability election even though it only benefits the other family.

In second marriages, spouses often use qualified terminable interest property (QTIP) trusts to transfer assets. In the example, assume when Julie passes away, she leaves $5 million in a trust to benefit Steve so long as he lives. At his death, the assets go to her children. This QTIP trust triggers no tax at Julie’s death because it qualifies as a marital gift. Later, when Steve passes away, assume he has $10 million to transfer to his children. He can shelter $5.34 million using his exemption amount. Remember, Julie’s exemption is still available to shelter up to another $5.34 million. The question is whether to allocate her exemption to shelter the $5 million transfer to her family or to Steve’s family. Tax law actually gives Steve’s executor the power to allocate Julie’s exemption. With a 40 percent estate tax rate, this becomes a $2 million debate.

These are just three planning issues that remain under the changed law. Things are certainly better, but not perfect. You still should consult with your estate planning attorney.
The majority of patients admitted to a hospital, treated in an emergency room or cared for in observation status will receive new medications or have changes made to their existing ones. This process is a significant source for potential adverse interactions, inadvertent omission of needed medications, unnecessary duplicate therapies or incorrect doses. These medication inconsistencies can happen at any point in direct care or in transition to another setting or provider. Medication reconciliation represents a method of maintaining consistent dosing throughout the continuum of care. It remains equally crucial to continue this consistency in ambulatory care where patients frequently obtain prescriptions from multiple providers or may have limited literacy or understanding of how their medicines are to be taken.

Accurate, safe and timely medication administration has been a goal of many organizations. Adverse drug events present the single greatest harm to patients in hospitals according to the Institute for Healthcare Improvement. The National Patient Safety Goal is an effort on the part of the Joint Commission to address this issue. The Agency for Healthcare Research and Quality provides information on adverse drug events. This is similar to the Partnership for Patients, a public and private collaborative within the Centers for Medicare & Medicaid Services (CMS) that also offers information on this process.

The reduction of outright or potential adverse drug events remains a challenge requiring constant attention. The most common implicated meds include warfarin, insulin, opioids, oral hypoglycemics, anti-platelet agents and digoxin. CMS attempts to track adverse event data from these agents. The Patient Safety and Clinical Pharmacy Service Collaborative PSPC is a resource within the Health Resources and Service Administration for organizations and facilities to engage in this common effort including pharmacists.

The involvement of clinical and commercial pharmacists, as well as computerized physician order entry and electronic prescribing, encourages a team-based approach to help medication administration be safe and timely. Multiple prescribers in various settings, as well as the liberal use of over-the-counter medications and supplements, makes the challenge of medication safety and reconciliation daunting. Helping patients, especially those with cognition and compliance issues, maintain accurate medication intake will require an extensive teamwork approach to this issue in the future.

Severe drug adverse reactions are unpredictable, complex and relatively uncommon. Those who prescribe should anticipate problems and avoid complacency. Patient safety remains the major concern in this area. Quality medication delivery involves ongoing accurate, up-to-date orders and using prescriptions and orders that avoid the use of abbreviations. Appropriate use of narcotics, anti-psychotics and antibiotics has come to the forefront. The future use of these often helpful but potentially harmful agents will likely be monitored more closely. Potentially inappropriate medications in older adults are listed in Beers Criteria, which can serve as a resource for prescribers. Please contact The South Dakota Foundation for Medical Care if you would like more information on medication safety.
SDBMOE Board News

By Margaret B. Hansen, PA-C, MPAS, Executive Director
South Dakota Board of Medical and Osteopathic Examiners

SDBMOE Member Update
Gov. Dennis Daugaard has re-appointed Mary S. Carpenter, MD, and appointed Laurie B. Landeen, MD, and Ms. Deborah Bowman for three-year terms as members to the Board of Medical and Osteopathic Examiners.

Renewal Cycle for South Dakota Medical Licenses Continues Until March
If you are experiencing difficulties with your online renewal application, please contact the SDBMOE staff for assistance. The staff is also collecting feedback regarding the renewal application in an effort to make improvements in the future. The following information was published in last month’s article and is being reprinted as it contains helpful renewal material.

The South Dakota Board of Medical and Osteopathic Examiners (SDBMOE) is requesting additional data with licensure renewal applications this year. This effort is part of a state and nationwide effort to identify information about health care professionals including academic training history, where and what services are provided to patients as well as other demographic information. Gov. Dennis Daugaard made improving the availability of health care providers in rural South Dakota a key provision of his South Dakota Workforce Initiative (SD WINS). This effort has been approved by the South Dakota Legislature.

A recent Associated Press story also highlights the problem in South Dakota. Nineteen of South Dakota’s 66 counties lack a primary care physician. Information gathered by the SDBMOE from the new questions on the renewal applications may provide assistance in determining how effective the rural recruitment efforts have been, and how to adjust those efforts to meet anticipated needs. The SDBMOE will ask the questions of all their regulated professions to provide a more detailed look at which health care personnel are available to serve the needs of South Dakotans.

Frequently Asked Questions (FAQ) that the Board Staff is fielding this year:

Q: You already have my medical school information. Why do you need it again?
A: The academic training information is not yet available in an electronic format; therefore, for this year, licensees will need to provide the month and year of completion for medical school and post graduate training.

Q: How do I answer the “how many weeks worked” question?
A: Use the actual number of weeks worked. For example, if you took two weeks of vacation, the answer would be 50 weeks.

Q: The “Please provide more details...” box comes up after one of my answers. If I made a mistake, can I type: “I hit the incorrect answer” in the explanation field?
A: Yes, that would be an appropriate response. If more details are needed for this or any other explanation, you will be contacted.

Q: The individuals that I supervise are not correctly listed. What do I do to change this?
A: Please send an email with the necessary corrections and the board staff will assist you. You can see who you are supervising at any time by doing a Licensee Look-Up on the Board website.

Q: Why are you only asking about the American Board of Medical Specialties (ABMS) board certification? Shouldn’t the American Osteopathic Board (AOA) certification be asked as well?
A: At this time, only the ABMS is being requested as it is the only board certification organization mentioned in the South Dakota medical practices act. Also, this question was already part of the information technology (IT) system so it was relatively easy to include in the renewal application. Next year the ability to ask about AOA certification may be available.

REFERENCES
2. SDCL 34-12G.
My grandmother Axie died at 99 having lived a blessed yet tragic life. As a young girl, she lost her father to some illness. As time passed, her mother, struggling to raise four children, lost a second husband, and then a third. When the third husband died, Axie’s mother, out of desperation, put some of her children into an orphanage. After all this, my grandmother grew up separated from her family. Obviously, she held no grudge since during a very successful professional life as a federal judge, Axie cared for her aging and then dying mother, an orphan made good.

The history of adoption is as old as humankind, with family members raising children orphaned by death, or war, or economic destruction. The middle ages introduced the concept of orphanage when babies were left at the door of monasteries, and were raised in the institution of the church.

But much of what the world knows of adoption and rules to protect orphans actually stems from the orphan trains of the U.S. in the late 1800s. The story begins with the U.S. Civil War and the increase in immigration at the time. The coincidence resulted in orphanage-over-crowding and huge numbers of homeless children roaming the streets of east coast urban cities. A group of religious leaders spearheaded a solution by shipping them on trains to the rural west.

Over the next 70 years, something like 250,000 orphaned, abandoned or homeless children were placed on trains and sent to western rural foster families working the land. The largest mass relocation of children to ever occur, this helped establish foster care in America and brought many lost youth into families where discipline and love gave them a chance for a reasonable life. At the same time, however, many of these children were indentured and exploited rather than adopted, and were made to become farm laborers and household servants.

Because of this social experiment, the need to protect children from abuse developed, and the best example was the Minnesota adoption law of 1917 requiring background checks of families with follow-up after placement. More foster homes were encouraged instead of institutional orphanages after this, and the American trend for adoption to ensure the best interest of the child spread globally.

Presently in the U.S., not every orphan is adopted, with something like 130,000 children now awaiting adoption. Another little Axie out there needs a parent.
Greetings and happy spring from all of us at DAKOTACARE! I believe we now deserve some outstanding weather for our perseverance over the last few months of downright horrid conditions.

For the last few years, at this time of the year, I have written about the need for all physicians to continue our efforts in combating cardiovascular disease, still the number one cause of death in the U.S. and South Dakota. I plan to do the same now. Please review the map below which delineates statewide 2011 county-by-county death rates secondary to vascular disease (e.g., heart disease and stroke). It doesn’t look good to me, so what can we do?

We need to continue our efforts in preventing these events, via more aggressive encouragements to our patients to follow the ABC’S (remember these from last year?) listed below. By controlling these risk factors we could reduce risk of heart attack or stroke by more than 80 percent. Only when we all work together can we impact the devastation these medical conditions have on our patients and loved ones.

A – Appropriate aspirin therapy  
B – Blood pressure control  
C – Cholesterol management  
S – Smoking cessation

I want to remind you as well about the CDC-sponsored initiative entitled Million Hearts (http://millionhearts.hhs.gov). This national initiative’s goal is to prevent 1 million heart attacks and strokes by 2017. Million Hearts brings together communities, health systems, nonprofit organizations, federal agencies and private sector partners from across the country to fight heart disease and stroke. I believe this laudable goal deserves our full attention and support. Cardiovascular mortality and morbidity may not directly impact your practice, but I guarantee it has impacted your network of family and friends.

Finally, another annual shameless plug asking you to consider supporting and/or participating in a Heart Walk somewhere in South Dakota this summer. These events are sponsored by your American Heart Association, through financial donations by corporations (such as DAKOTACARE, sponsor of the Healthy Change Award) and are integral to the AHA’s national campaign to get America moving. These are non-competitive walks which celebrate those who have made lifestyle changes while encouraging many more to take the pledge to live healthier lifestyles while raising the dollars needed to fund life-saving research and education, advocate for health and save lives. They will be occuring in the following locations:

Pierre – Saturday, Sept. 20 – Main Street Square  
Rapid City – Saturday, Sept. 13 – Hyde Stadium  
Sioux Falls – Saturday, Aug. 23 – Falls Park

I hope to see you there!

Correction

An editorial error caused the lead quotation to be left out of the DAKOTACARE Update in February. An important reference to this quote was made near the end of the article on goals, values and principles and its absence may have caused confusion. The emphasis may now be lost in context, but the quotation reads: “A people that values its privileges above its principles soon loses both.”  – Dwight D. Eisenhower

South Dakota Medicine regrets the omission.
Autism spectrum disorder (ASD) is a complex and pervasive neurodevelopmental disorder that affects one in 88 children. The rate of diagnosis has been increasing every year for decades with no indication of abating. Despite the fact that more individuals are being diagnosed with ASD, there remains much work to be done in providing effective screening procedures that result in early identification and treatment of the disorder. Early identification of ASD is critical in providing children with access to early behavioral and communication intervention, which are the only evidence-based treatments that have been shown to improve outcomes. Additionally, co-occurring conditions can be identified and treated.

While every person diagnosed with ASD displays the same general symptoms of persistent deficits in social communication and social interaction, along with restricted, repetitive patterns of behavior, interests, or activities, the disorder truly defies generalization. The type of onset, presentation of symptoms, level of cognitive, language, and adaptive functioning, and severity of symptoms vary widely from case to case. Often, there are co-occurring conditions that can mask or mirror the symptoms of ASD, such as seizure disorders, anxiety and mood disorders, intellectual disabilities, language delays and challenging behaviors that make it difficult to adequately identify. However, all children with ASD develop symptoms before the age of 3, and ASD can be reliably diagnosed by experienced clinicians by 18 months.

Despite the advances in knowledge and practice in recent years, the reality remains that many children are not identified until well after their third birthday, and subsequently are not receiving early intervention at a developmentally critical age. The American Academy of Pediatrics (AAP) recommends that all children be screened specifically for ASD during their regular well-child visits at 18 months and 24 months. One of the identified issues has been not adopting a systematic process for utilizing standardized screening instruments in addition to surveillance. Other factors include the following: perceived lack of time on the part of practitioners, and waiting until a parent raises concerns with their child’s developmental progress. It has been documented that parents often will not raise these concerns unless they are prompted. Additionally, many children are not brought in for routine well-child visits due to living in remote or underserved areas, and are only seen in emergency room or urgent care settings or for immunizations.

While many practitioners do utilize a systematic surveillance and screening protocol, this alone is not sufficient to effectively identify all children who need to be referred for a comprehensive diagnostic evaluation in a timely manner. For many children who only receive health care services for other illnesses, injuries, or immunizations, screening at these visits is critical. A brief review of the history will reveal those children who have not yet been screened, and the screening can be provided at the same visit. Practitioners cannot wait for parents to voice their concerns independently.

The following steps have been recommended by the AAP and the Centers for Disease Control and Prevention (CDC):

1. The first step is for parents to complete the screening tool.
2. The second step is for the clinician to score and review.
3. If the child screens positive, immediate action is required. The provider discusses results and concerns with parents, performs more specific medical and developmental assessment and/or refers for further assessment, and provides anticipatory guidance.
4. The final step is referral to Birth to 3 early intervention services if the child is under 3 years, or to their local school district special education office if the child is 3 years or older.

Commonly used and validated screening measures for ASD include the following:

- Modified checklist for autism in toddlers, revised (M-CHAT-R/F);
- Ages and stages questionnaire: Social emotional (ASQ:SE);
- Communication and symbolic behavior scales (CSBS);
- Parents’ evaluation of developmental status (PEDS);
- Screening tool for autism in toddlers and young children (STAT).
Most of these can be administered in as little as five minutes and are available for free or very little cost. Additionally, screening procedures for ASD can be billed using the CPT code 96110.

It is important to remember that a child who shows up positive on an ASD screening tool may exhibit signs associated with developmental delay and ASD; however, a positive screen does not indicate that the child meets the diagnostic criteria. These children must be referred for a comprehensive evaluation. Typically, the best place to start the referral is Birth to 3 early intervention services for children under the age of three, and to the local school district for children age 3 and older. Early identification and intervention is the key to increased positive outcomes for children with ASD.

REFERENCES


Please note: Due to limited space, we are unable to list all references. You may contact South Dakota Medicine at 605.336.1965 for a complete listing.

About the Author:
Eric G. Kertz, PhD, director of clinical operations and community services; assistant professor of pediatrics, University of South Dakota Sanford School of Medicine; Center for Disabilities.
REGISTER NOW!

SOUTH DAKOTA STATE MEDICAL ASSOCIATION

2014 ANNUAL MEETING

WHAT: 2014 SDSMA Annual Meeting

WHEN: May 30-31

Friday, May 30 - Educational sessions, exhibitors, Young Physician Mixer, Celebrating Medicine Reception, Presidential Banquet, Awards & Scholarships

Saturday, May 31 - SDSMA PAC Breakfast, Council of Physicians meeting

WHERE: Ramkota Hotel & Conference Center, Rapid City

And don’t miss the golf tournament and the SDSMA Alliance Medical Student Scholarship Fundraiser on Thursday, May 29!

We hope to see you there – register today!

To register, please return the form you received in the mail, or visit www.sdsma.org.

April is autism awareness month, and in keeping with the spirit of the month, we would like to make you more cognizant of this important neurodevelopmental disorder. According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), published in May 2013, the neurodevelopmental disorders now include intellectual developmental disorder or intellectual disability, communication disorders (one of which is called social “pragmatic” communication disorder), ADHD, specific learning disorder, motor disorders (i.e., Tourette disorder) and autism spectrum disorder. These disorders are not new to the medical literature but some have been renamed and their boundaries redefined. In fact, last year was the 70th anniversary of Leo Kanner’s article “Autistic Disturbances of Affective Contact” in The Nervous Child.¹

South Dakota has a strong connection to the man who first described this disorder (originally known as Kanner Syndrome) and who later became the father of child and adolescent psychiatry. Leo Kanner was born in Austria, served in World War I, was educated in Germany where he received his medical degree from the University of Berlin in 1921, served as the volunteer assistant, Charite Hospital, University of Berlin from 1920 to 1924 when he emigrated to Yankton to work at Yankton State Hospital as a senior physician. While there, he published numerous journal articles which were noticed by Adolf Meyer, the chief of psychiatry at Johns Hopkins University School of Medicine. In 1928, Dr. Meyer invited him to be the Commonwealth Fund Fellow in Psychiatry, and by 1930 he had established the first child psychiatric clinic in the U.S. at the Johns Hopkins Hospital’s Harriet Lane Home for Invalid Children. In 1935, he published the specialty’s first textbook, Child Psychiatry.²

However, autism’s acceptance as a distinct disorder separate from childhood schizophrenia, attachment disorder or intellectual disability took more than 30 years to sort out. Over the course of 15 years of study of his patients at the Johns Hopkins Child Psychiatry Clinic, Kanner believed that these patients had two cardinal features in common – a profound “autistic aloneness” and an “obsessive insistence on the preservation of sameness.” Many of his associated findings still hold true. He believed that it was “inborn,” or genetic, which has been substantiated by twin studies.³ He felt that it was seen more frequently in males (eight out of 11 of his original patients were boys), which have been substantiated by prevalence studies. Five of his patients had large heads and several were somewhat clumsy. One “interesting common denominator in the backgrounds of these children” is “they all come from highly intelligent families,” many of whom “had a great deal of obsessiveness in the family background.” Kanner also felt that they related better to concepts than to people. Many of these characteristics were also highlighted by the Austrian pediatrician, Hans Asperger, in his paper Die ‘autistischen Psychopathen’ im Kindesalter, published in German in 1944.⁴

Though it was briefly mentioned in the Diagnostic and Statistical Manual of Mental Disorders, Second Edition (DSM-II), it wasn’t until 1980 when the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III) was published that autism was first included as a diagnosis. The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) in 1994 (and the revision in 2000) listed six domains used to define autism and included Asperger syndrome for the first time.

The DSM-5 was published in May 2013 with new criteria for the diagnosis of autism. It no longer includes Asperger syndrome. In order for a child to be diagnosed with autism, they must meet five criteria. First, there must be persistent deficits in social communication and social interaction across multiple contexts, currently or by history. Second, there must be at least two restricted, repetitive patterns of behavior, interests, or activities, currently or by history. Third, there must be symptoms present in the early development period. These symptoms may not fully manifest until later stages or may be masked by learned strategies later in life. Fourth, these symptoms must significantly impair social, occupational or other areas of functioning. Last, these symptoms must not be better explained by intellectual disability or global developmental delay alone. The DSM-5 lists multiple examples for each criterion and requires the examiner to rate the severity of symptoms.

There are multiple tools that can be used to screen...

SOUTH DAKOTA STATE MEDICAL ASSOCIATION
ANNUAL MEETING

MAY 30 - 31, 2014
BEST WESTERN RAMKOTA HOTEL & CONFERENCE CENTER • RAPID CITY, SOUTH DAKOTA

FEATURED PRESENTATIONS
FRIDAY, MAY 30

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<tr>
<th>Prescription Drug Abuse and Diversion</th>
<th>The Future Has Arrived - Medical School Update</th>
<th>Prescription Drug Abuse Panel - Utilizing a Team Approach to Treating Patients, Not Criminals</th>
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<tbody>
<tr>
<td>Christopher T. Dietrich, MD</td>
<td>Mary D. Nettlemann, MD</td>
<td>Christopher T. Dietrich, MD</td>
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<tr>
<td>The Rehab Doctors</td>
<td>Dean, USD Sanford School of Medicine</td>
<td>MD, Moderator</td>
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<td>Kevin Bjordahl, MD, SDBMGE Member, Avera Medical Group Milbank</td>
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<td>Personal Measurements for Success &amp; Fulfillment</td>
<td>Patient and Team Communication Tools</td>
<td>Marc Boddicker, MD, Advanced Dermatology Center</td>
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<td>Kent Kramer, Chief Investment Officer, Foster Group</td>
<td>Robert S. Thompson, Director of Education, MMIC Group</td>
<td>Randy Jones, Executive Director, South Dakota Board of Pharmacy</td>
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<td>Team-Based Care</td>
<td>Shaping the Future of American Health Care</td>
<td>Craig Ulthe, MD, Sanford Family Medicine</td>
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<td>Charles E. McBride III, MD</td>
<td>Robert M. Wah, MD, President-Elect, AAMA Board of Trustees</td>
<td>John Wenande, Special Assistant Attorney General, South Dakota DCI</td>
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JOIN US FOR THESE OTHER EXCITING EVENTS!

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<tr>
<th>Day</th>
<th>Event</th>
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<tr>
<td>Thursday, May 29</td>
<td>Alliance Medical Student Scholarship Fundraiser</td>
<td>7 pm</td>
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<tr>
<td>Friday, May 30</td>
<td>Networking Lunch</td>
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<td>Young Physicians Mixer &amp; “Celebrating Medicine” Reception</td>
<td>6 pm</td>
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<tr>
<td></td>
<td>Presidential Banquet with Awards &amp; Scholarship Recognition</td>
<td>7 pm</td>
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<tr>
<td>Saturday, May 31</td>
<td>SDSMA PAC Breakfast Forum with U.S. Senate Candidates</td>
<td>7:30 am</td>
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For more information and to register, visit www.sdsma.org.
children for autism in your practice. It is recommended by the American Academy of Pediatrics (AAP) that children be screened at 18 months and 30 months, although screening can occur at the routine 24-month visit and if concerns arise, followed-up at 30 months. Early screening is important as early intervention can improve outcomes for children diagnosed with autism. Screening can begin as early as 6 months with the communication and symbolic behavior scales; developmental history, infant and toddler checklist (CSBS-DP-ITC), which is valid for children from 6-24 months. The modified checklist for autism in toddlers, revised (M-CHAT-R/F) was validated in the journal Pediatrics in January and can be used for screening and follow-up in children from 16-30 months. Both of these screening tools are public domain, are free to use and can be downloaded online. For older children, numerous other assessment tools such as the autism spectrum rating scale for ages 2-5 and ages 6-18 (ASRS), the Gilliam autism rating scale, third edition (GARS-3), the childhood autism rating scale, second edition (CARS-2) and the Asperger syndrome diagnostic scale (ASDS) can be purchased. These checklists are for both parents and caregivers/teachers. At all well-child visits, developmental surveillance and screening is recommended with prompt referral to Birth to 3 Connections, your local school district or educational cooperative if any concerns or red flags arise. Milestones that are concerning are the following: lack of babbling by 9 months, failure to respond to their name by 12 months, lack of single word use by 15-18 months or no pointing, gaze monitoring (joint attention) or pretend play by 2 years.

Since the services required by children with this developmental disorder work best when provided early, need to be intensive, and thus, are expensive, the standard of care has been a multidisciplinary or team evaluation. Professionals with knowledge about genetics, normal child development, sensory development, speech and language development and social and emotional development are important members of the team. A comprehensive developmental history is obtained, autism-specific questionnaires are completed by individuals who have direct interactions with the child and the autism diagnostic observation schedule (ADOS) is administered. In South Dakota, such teams are found in our schools or educational co-ops and are considered “educational teams” or in medical facilities such as Sanford Children’s Specialty Clinic and are considered “medical teams.” The Center for Disabilities in Sioux Falls and Children’s Care Hospital and School Outpatient Rehabilitation Center have “hybrid” teams in that they include educational, therapy and psychological professionals, but they also include a child and adolescent psychiatrist. Such evaluations are expensive and are time intensive. Thus, good screenings in primary care providers’ offices will expedite appropriate referrals, comprehensive evaluations, evidenced-based early intervention and medical management of coexisting conditions.

The demographics of South Dakota present unique challenges to the early identification and treatment of children with autism spectrum disorder. Because many communities in our state lack developmental surveillance and screening, we asked Dr. Eric Kurtz, the director of clinical services at the Center for Disabilities, to summarize some simple and effective screening tools and techniques to identify these children as early as possible. But if evidence-based treatments are not made available to children identified as autistic, screening and evaluation will have little impact on their ultimate outcome. The second practice parameter for the assessment and treatment of children and adolescents with autism spectrum disorder was just published in the Journal of the American Academy of Child and Adolescent Psychiatry in February 2014. They nicely outlined the clinical presentation and course, epidemiology, etiology, differential diagnosis, and co-morbidities and made three recommendations on assessment and four recommendations on treatment. As a state, we will not be able to meet these practice parameters without the cooperation of our primary care providers, pediatric subspecialists, child and adolescent psychiatry providers, early intervention teams, school districts and educational cooperatives. Because of limited educational resources, there has been a push to use health care resources to provide the evidenced-based treatment services that these children need. Some discussion and proposed legislation has occurred this year in the South Dakota Legislature.

REFERENCES


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About the Authors:
Jerome M. Blake, MD, associate professor of pediatrics, Sanford School of Medicine of the University of South Dakota; practicing developmental behavioral pediatrician, Sanford Children’s Specialty Clinic, Sioux Falls.
Jacqueline Berner, MD, second-year pediatric resident; plans to pursue a fellowship in developmental behavioral pediatrics.
1. Recommended Immunization schedule for persons aged 0 through 18 years – United States, 2014.

These recommendations must be read with the footnotes that follow. For those who fall behind or start late, provide catch-up vaccination at the earliest opportunity as indicated by the green bars in Figure 1. To determine minimum intervals between doses, see the catch-up schedule (Figure 2). School entry and adolescent vaccine age groups are in bold.

<table>
<thead>
<tr>
<th>Vaccine</th>
<th>Birth</th>
<th>1 mo</th>
<th>2 mos</th>
<th>4 mos</th>
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<th>9 mos</th>
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<th>18 mos</th>
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<tr>
<td>Diphtheria, tetanus, &amp; acellular pertussis (DTaP&lt;7 yrs)</td>
<td>1st</td>
<td>2nd</td>
<td>3rd</td>
<td>4th</td>
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<td>Tetanus, diphtheria, &amp; acellular pertussis (Tdap&lt;10 yrs)</td>
<td>1st</td>
<td>2nd</td>
<td>3rd</td>
<td>4th</td>
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<tr>
<td>Haemophilus influenza type b (Hib)</td>
<td>1st</td>
<td>2nd</td>
<td></td>
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<tr>
<td>Pneumococcal conjugate (PCV13)</td>
<td>1st</td>
<td>2nd</td>
<td>3rd</td>
<td>4th</td>
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<tr>
<td>Pneumococcal polysaccharide (PPSV23)</td>
<td>1st</td>
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<td>2nd</td>
<td>3rd</td>
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<tr>
<td>Inactivated poliovirus (IPV) (≤18 yrs)</td>
<td>1st</td>
<td>2nd</td>
<td>3rd</td>
<td>4th</td>
<td></td>
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<tr>
<td>Influenza (IV, LAIV) 2 doses for some: See footnote 8</td>
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<tr>
<td>Measles, mumps, rubella (MMR)</td>
<td>1st</td>
<td>2nd</td>
<td>3rd</td>
<td>4th</td>
<td></td>
<td></td>
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<tr>
<td>Varicella (VAR)</td>
<td></td>
<td>1st</td>
<td></td>
<td>1st</td>
<td></td>
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</tr>
<tr>
<td>Hepatitis A (HepA)</td>
<td>1st</td>
<td>2nd</td>
<td>3rd</td>
<td>4th</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Human papillomavirus (HPV2: females only; HPV4, males; females)</td>
<td>1st</td>
<td>2nd</td>
<td>3rd</td>
<td>4th</td>
<td></td>
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</tr>
<tr>
<td>Meningococcal (4-doses: MenB-4CMY-6 weeks; MenACWY-D&lt;9 mos; MenACWY-CRM&lt;2 mos)</td>
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</table>

This schedule includes recommendations in effect as of January 1, 2014. Any dose not administered at the recommended age should be administered at a subsequent visit, when indicated and feasible. The use of a combination vaccine generally is preferred over separate injections of its equivalent component vaccines. Vaccination providers should consult the relevant Advisory Committee on Immunization Practices (ACIP) statement for detailed recommendations, available online at [http://www.cdc.gov/vaccines/acip/recs/index.html](http://www.cdc.gov/vaccines/acip/recs/index.html). Clinically significant adverse events that follow vaccination should be reported to the Vaccine Adverse Event Reporting System (VAERS) online [http://vaers.hhs.gov](http://vaers.hhs.gov) or by telephone (800-822-7970). Suspected cases of vaccine-preventable diseases should be reported to the state or local health department. Additional information, including precautions and contraindications for vaccination, is available from CDC online [http://www.cdc.gov/vaccines](http://www.cdc.gov/vaccines) or by telephone (800-232-4636). This schedule is approved by the Advisory Committee on Immunization Practices ([http://www.cdc.gov/vaccines/acip](http://www.cdc.gov/vaccines/acip)), the American Academy of Pediatrics ([http://www.aap.org](http://www.aap.org)), the American Academy of Family Physicians ([http://www.aafp.org](http://www.aafp.org)), and the American College of Obstetricians and Gynecologists ([http://www.acog.org](http://www.acog.org)).

NOTE: The above recommendations must be read along with the footnotes of this schedule.

For more information and to view footnotes, please visit [http://www.cdc.gov/vaccines/schedules/hcp/child-adolescent.html](http://www.cdc.gov/vaccines/schedules/hcp/child-adolescent.html)
**FIGURE 2. Catch-up immunization schedule for persons aged 4 months through 18 years who start late or who are more than 1 month behind — United States, 2014.**

The figure below provides catch-up schedules and minimum intervals between doses for children whose vaccinations have been delayed. A vaccine series does not need to be restarted, regardless of the time that has elapsed between doses. Use the section appropriate for the child’s age. Always use this table in conjunction with Figure 1 and the footnotes that follow.

### Persons aged 4 months through 6 years

<table>
<thead>
<tr>
<th>Vaccine</th>
<th>Minimum Age By Date 1</th>
<th>Minimum Interval Between Doses</th>
<th>Minimum Interval Between Doses</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Dose 1 to dose 2</td>
<td>Dose 2 to dose 3</td>
<td>Dose 3 to dose 4</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>birth</td>
<td>4 weeks</td>
<td>8 weeks and at least 16 weeks after first dose; minimum age for the first dose is 34 weeks</td>
</tr>
<tr>
<td>Rotavirus</td>
<td>6 weeks</td>
<td>4 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Diphtheria, tetanus, &amp; pertussis</td>
<td>6 weeks</td>
<td>4 weeks</td>
<td>6 months</td>
</tr>
<tr>
<td>Haemophilus influenza type b</td>
<td>6 weeks</td>
<td>4 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Pneumococcal</td>
<td>6 weeks</td>
<td>4 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Inactivated poliovirus</td>
<td>6 weeks</td>
<td>4 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Meningococcal</td>
<td>6 weeks</td>
<td>8 weeks</td>
<td>8 weeks</td>
</tr>
<tr>
<td>Measles, mumps, rubella</td>
<td>12 months</td>
<td>4 weeks</td>
<td>8 weeks</td>
</tr>
<tr>
<td>Varicella</td>
<td>12 months</td>
<td>3 months</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>12 months</td>
<td>6 months</td>
<td>4 weeks</td>
</tr>
</tbody>
</table>

### Persons aged 7 through 18 years

<table>
<thead>
<tr>
<th>Vaccine</th>
<th>Minimum Age By Date 1</th>
<th>Minimum Interval Between Doses</th>
<th>Minimum Interval Between Doses</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Dose 1 to dose 2</td>
<td>Dose 2 to dose 3</td>
<td>Dose 3 to dose 4</td>
</tr>
<tr>
<td>Tdap vaccines</td>
<td>7 years</td>
<td>4 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Human papillomavirus</td>
<td>9 years</td>
<td>Routine dosing intervals are recommended</td>
<td>6 months</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>12 months</td>
<td>6 months</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>birth</td>
<td>4 weeks</td>
<td>8 weeks (and at least 16 weeks after first dose)</td>
</tr>
<tr>
<td>Inactivated poliovirus</td>
<td>6 weeks</td>
<td>4 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Meningococcal</td>
<td>6 weeks</td>
<td>8 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Measles, mumps, rubella</td>
<td>12 months</td>
<td>4 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Varicella</td>
<td>12 months</td>
<td>3 months if person is younger than 13 years, 4 weeks if person is aged 13 years or older</td>
<td>4 weeks</td>
</tr>
</tbody>
</table>

**NOTE:** The above recommendations must be read along with the footnotes of this schedule.

For more information and to view footnotes, please visit http://www.cdc.gov/vaccines/schedules/hcp/child-adolescent.html
<table>
<thead>
<tr>
<th>Name</th>
<th>Specialty</th>
<th>University/Department</th>
<th>City, State</th>
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<tbody>
<tr>
<td>Megan E. Albertson (TRAN)</td>
<td>PGY2 (RADI-DIAG)</td>
<td>University of Nebraska Medicine Center</td>
<td>Omaha, Nebraska</td>
</tr>
<tr>
<td>Jon M. Christensen (ANES)</td>
<td>Mayo Clinic</td>
<td>Department of Anesthesiology</td>
<td>Rochester, Minnesota</td>
</tr>
<tr>
<td>Kelly L. Delaney (MEDICINEC)</td>
<td>Gundersen Lutheran Medical Foundation</td>
<td>La Crosse, Wisconsin</td>
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<tr>
<td>Adam W. Dell (PEDS)</td>
<td>Primary Children's Medical Center</td>
<td>Pediatric Program</td>
<td>Salt Lake City, Utah</td>
</tr>
<tr>
<td>Cara A. Drew (FAMP)</td>
<td>Siouxland Medical Education Foundation</td>
<td>Family Medicine Program</td>
<td>Sioux City, Iowa</td>
</tr>
<tr>
<td>Stephn W. Drywater (EMER)</td>
<td>Oklahoma University School of Community Medicine</td>
<td>Tulsa, Oklahoma</td>
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</tr>
<tr>
<td>Matthew R. Frank (TRAN)</td>
<td>Sanford Health Medical &amp; Student Education Fargo, North Dakota</td>
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</tr>
<tr>
<td>Derek J. Gearman (SURG-PRE)</td>
<td>Mayo Clinic Surgery Program</td>
<td>Rochester, Minnesota</td>
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<tr>
<td>Tyler C. Gillen (EMER)</td>
<td>Akron General Medicine Center Emergency Medicine Program</td>
<td>Akron, Ohio</td>
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</tr>
<tr>
<td>Elizabeth Hahn (OGYN)</td>
<td>Truman Medicine Center Department of OB/GYN</td>
<td>Kansas City, Missouri</td>
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<tr>
<td>Kayla M. Haines (FAMP)</td>
<td>Center for Family Medicine</td>
<td>Sioux Falls Family Medicine Program</td>
<td>Sioux Falls, South Dakota</td>
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<tr>
<td>Laura A. Hill (SURG-GEN)</td>
<td>Gundersen Medical Foundation</td>
<td>La Crosse, Wisconsin</td>
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<tr>
<td>Daniel V. Irwin (FAMP)</td>
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<td>Sioux City, Iowa</td>
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<tr>
<td>Benjamin J. Jensen (ANES)</td>
<td>University of Wisconsin Hospital and Clinics</td>
<td>Clinical Science Center</td>
<td>Madison, Wisconsin</td>
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<tr>
<td>Sarah N. Johnson (ORTHO)</td>
<td>Truman Medicine Center Department of Orthopaedic Surgery</td>
<td>Kansas City, Missouri</td>
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<tr>
<td>Brad R. Julius (MEDICINEC)</td>
<td>University of Iowa Hospitals and Clinics</td>
<td>Department of Internal Medicine</td>
<td>Iowa City, Iowa</td>
</tr>
<tr>
<td>Nicholas F. Kaufman (MEDICINEC)</td>
<td>Military Match – Air Force</td>
<td>San Antonio Uniformed Services Health Education Center</td>
<td>Fort Sam Houston, Texas</td>
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<tr>
<td>David M. Krause (MEDICINEC-PRE)</td>
<td>USD Sanford School of Medicine Department of Medicine</td>
<td>Sioux Falls, South Dakota</td>
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</tr>
<tr>
<td>David M. Krause (MEDICINEC-PRE)</td>
<td>IU Health University Hospital Department of Radiology</td>
<td>Indianapolis, Indiana</td>
<td></td>
</tr>
</tbody>
</table>
Jeb T. List (TRAN)  
Indiana University Health Ball Memorial Hospital  
Muncie, Indiana

**PGY2 (RADI-DIAG)**  
Creighton University Medical Center  
Department of Radiology  
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Des Moines, Iowa

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Sioux Falls, South Dakota

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Madison, Wisconsin

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Pathology Program  
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Maria S. Tracy (OGYN)  
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Indianapolis, Indiana

Tim Vanadurongvan (SURG-PRE)  
University of Colorado Denver School of Medicine  
Aurora, Colorado

Allison P. Watson (MEDICINEC)  
University of Minnesota Medical Center  
Minneapolis, Minnesota

Erin E. Williams (TRAN)  
Hennepin County Medical Center  
Minneapolis, Minnesota

**PGY2 (DERM)**  
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Department of Dermatology  
St Louis, Missouri

Tessa L. Winterton (OGYN)  
Wesley Medicine Center  
Department of OB/GYN  
Wichita, Kansas
### 2014 SDSMA PAC Membership

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<tr>
<td>Janice Knutsen</td>
</tr>
<tr>
<td>Roger Knutsen, MD</td>
</tr>
<tr>
<td>Karla Murphy, MD</td>
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<td>Thomas Murphy</td>
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<tbody>
<tr>
<td>Judy Allen</td>
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<tr>
<td>Robert Allen, MD</td>
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<tr>
<td>Anne Barlow</td>
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<td>John Barlow, MD</td>
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<td>Jean Bubak</td>
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<td>Mark Bubak, MD</td>
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<td>Carey Buhler, MD</td>
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<td>Darlene Buhler</td>
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<td>Mary Carpenter, MD</td>
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<td>Mark East</td>
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<tr>
<td>Dan Flynn</td>
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<td>Daniel Johnson, MD</td>
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<tr>
<td>Christiane Maroun, MD</td>
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<tr>
<td>Jean McHale</td>
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<td>Michael McHale, MD</td>
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<td>Mary Milroy, MD</td>
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<tr>
<td>Barb Smith</td>
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<td>Raed Sulaiman, MD</td>
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<tr>
<td>Deborah Kullerd, MD</td>
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<tr>
<td>Kay Lawler</td>
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<td>Patrick Lawler, MD</td>
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<td>Alan Lawrence, MD</td>
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<td>Arie Lawrence</td>
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<td>Claudette Margallo</td>
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<tr>
<td>Lucio Margallo, MD</td>
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<tr>
<td>Scott Maxwell</td>
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<tr>
<td>Jennifer May, MD</td>
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<tr>
<td>Karen McPherson</td>
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<tr>
<td>Scott McPherson, MD</td>
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<tr>
<td>Stephan Miller, MD</td>
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<tr>
<td>Janice Minder</td>
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<tr>
<td>Jim Minder, MD</td>
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<td>Mary Nettleman, MD</td>
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<td>Marlys Porter</td>
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<td>Richard Porter, MD</td>
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<td>Herbert Saloum, MD</td>
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<td>Linda Saloum</td>
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<td>Arisami Thambi-Pillai</td>
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<td>Thavvan Thambi-Pillai, MD</td>
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<td>Marilyn Van Demark</td>
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<td>Robert Van Demark, Jr., MD</td>
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<td>John Vidoloff, MD</td>
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<td>Carol Zielike, MD</td>
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<td>James Holloway, MD</td>
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<td>Donald Humphreys, MD</td>
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<td>George Jenter, DO</td>
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<td>James Keil, MD</td>
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<td>James Kerr, MD</td>
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<td>Donald Knudson, MD</td>
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<td>Richard Little, MD</td>
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<td>Roxana Lupu, MD</td>
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<td>George Maher, DO</td>
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<td>Teresa Marts, MD</td>
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<td>James McDouggall, MD</td>
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<td>Mary Miller, DO</td>
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<td>John Oliphant, MD</td>
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<td>Elizabeth Reiss</td>
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<td>William Rossing, MD</td>
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<td>Raymond Sherman, MD</td>
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<td>Gary Timmerman, MD</td>
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<td>Kynan Trail, MD</td>
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<td>Victoria Walker, MD</td>
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<td>Merritt Warren, MD</td>
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<td>Grace Wellman</td>
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<td>Robert Wenger, MD</td>
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<td>Thomas White, MD</td>
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<td>Jason Wickersham, MD</td>
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<td>Gregory Wiedel, MD</td>
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<td>Jeffrey Bergsbaken, MD</td>
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<td>Michael Eide, MD</td>
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<td>Dennis Knutson, MD</td>
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<th>Resident $50</th>
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<td>Kevin Smith</td>
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<td>Sarah Smith, MD</td>
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*Your SDSMA PAC membership is very important in order to elect political candidates who understand the practice of organized medicine in South Dakota. To donate to SDSMA PAC, please contact SDSMA Vice President Mark East at 605-336-1965 or m east@sdsm a.org.*
For Your Benefit:

**SDSMA Is Your Communications Link**

Information is essential to your continuing success and viability. You can stay up-to-date with key issues and events by logging on to the all-new SDSMA website, www.sdsmoa.org. The site has been completely redesigned to offer members brand new features and easier navigation.

As a member, you also receive the *South Dakota Medicine* every month and have online access to the journal. We also produce the bi-monthly *E-news*, quarterly *Council Comments*, weekly *InSession* during South Dakota’s legislative session, and Action Alerts to communicate health issues of importance to elected officials.

If you’d like more information about our communications programs give us a call at 605.336.1965, visit the SDSMA website at www.sdsmoa.org, or email Director of Communications Elizabeth Reiss at ereiss@sdsmoa.org. As always, thank you for your membership in SDSMA.

"For Your Benefit" is the SDSMA’s monthly update on programs and services available to physicians through their affiliation with the SDSMA.

Don’t forget to send in your favorite scenic photo for *South Dakota Medicine* front cover consideration. Send photos to ereiss@sdsmoa.org.

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### Online Registration Now Open for 2014 SDSMA Annual Meeting

The 2014 SDSMA Annual Meeting will be held at the Ramkota Hotel & Conference Center in Rapid City Friday, May 30 through Saturday, May 31. Online registration is open — simply visit www.sdsmoa.org and click the registration link on the calendar of events on the right-hand side of the homepage.

With presentations, discussions, networking opportunities and social and fundraising events, the SDSMA Annual Meeting is a great time to share ideas and learn from fellow SDSMA members, including medical students, residents, active physicians and honorary members.

Don’t miss the exciting lineup of presentations and speakers scheduled for Friday, May 30! Featured presentations include:

- “Prescription Drug Abuse and Diversion” – Christopher T. Dietrich, MD, The Rehab Doctors
- “The Future Has Arrived: Medical School Update” – Mary D. Nettleman, MD – Sanford School of Medicine
- “Shaping the Future of American Health Care” – Robert M. Wah, MD – American Medical Association

The SDSMA Center for Physician Resources will bring you three exciting presentations:

- “Patient and Team Communication Tools” – Robert S. Thompson, MMIC Group
- “Team-Based Care” – Charles E. McBride III, MD, Summit Medical Group
- “Personal Measurements For Success and Fulfillment” – Kent Kramer, Foster Group

In addition, a special panel about prescription drug abuse will be held. Christopher T. Dietrich, MD, will moderate the panel entitled “Utilizing a Team Approach to Treating Patients, Not Criminals.” The panel will include experts Kevin Bjordahl, MD, Mark Boddicker, MD, Craig Utne, MD, Randy Jones of the South Dakota Board of Pharmacy, and John Wenande, special assistant attorney general of the state Department of Criminal Investigation.

The SDSMA Annual Meeting is a benefit of your membership; most events are included with annual SDSMA dues. In addition to registering online, you can find a meeting schedule and more details about exciting events taking place during the Annual Meeting at www.sdsmoa.org.

Be sure to register today!

Those with questions may contact Laura Olson at lolson@sdsmoa.org or 605.336.1965.

*Source: SDSMA staff*
The Issue Is...

SDSMA Health Credentialing Bill Signed By Gov. Daugaard

A bill sponsored by the SDSMA has been signed by Gov. Dennis Daugaard. The bill establishes a timeframe in which physician and provider credentialing must be completed by South Dakota’s health plans.

In response to a problem brought forward by members regarding the length of time it can take for providers to be credentialed for participation in a health plan’s provider network, the SDSMA pursued the legislation (HB 1157), which requires health insurers to make retrospective payment for all clean claims submitted by a health care professional after the credentialing period for covered services provided by the health care professional during the credentialing period.

Additionally, the legislation requires all health insurers or other entities responsible for the credentialing of health care professionals on behalf of the insurer to notify the applicant of its determination regarding a properly completed application for credentialing within 90 days of receipt of the application.

With one in four South Dakotans living in a primary care shortage area, we cannot afford to lose physicians to surrounding states as a result of their inability to be credentialed within a reasonable period of time. South Dakota now joins the many surrounding states that have timeframes to which health plans must adhere. Prior to this legislation, South Dakota had no such requirement in statute or regulation.

Source: SDSMA staff

SDSMA Representatives Attend AMA National Advocacy Conference

SDSMA President Daniel J. Heinemann, MD, SDSMA Delegate to the AMA Mary S. Carpenter, MD, and SDSMA President-elect Mary J. Milroy, MD, met with Sen. John Thune while visiting Washington, D.C., for the AMA National Advocacy Conference.

SDSMA President Daniel J. Heinemann, MD, President-elect Mary J. Milroy, MD, SDSMA Delegate to the AMA Mary S. Carpenter, MD, and SDSMA CEO Barb Smith attend the American Medical Association National Advocacy Conference March 4-6 in Washington, D.C.

The conference aimed to help empower physicians to be an advocate for patients, the medical profession and the future of health care. At the conference, attendees heard from political insiders, industry experts and members of Congress on current efforts being made in health system reform refinement and implementation. Attendees took part in discussions to help shape the future of the AMA’s advocacy efforts and its work to improve the health of the nation.

While in Washington, SDSMA representatives met with South Dakota’s Congressional delegation to discuss concerns about cuts to physician payments under the flawed Medicare sustainable growth rate (SGR). Concerns about the forthcoming expiration of the 1.0 floor on the Work Geographic Practice Cost Indexes (GPCI) that could mean a drop in Medicare physician payment of up to 3 percent were also expressed to Congressional representatives. Last, SDSMA members discussed federal funding for graduate medical education and asked Congressional representatives for support for increasing funding.

Source: AMA and SDSMA staff

"The Issue Is" is the SDSMA’s monthly update on key policy issues of importance to physicians.
A Physician’s Guide to Wealth Transfer Event
April 17 and April 22

The SDSMA Center for Physician Resources invites SDSMA members, residents and medical students to its free event, “A Physician’s Guide to Wealth Transfer.” This is the final program in the Center’s series on personal finances and will provide information and guidance on how to preserve the integrity of your assets when transferring to others.

A live event will be held at 7 p.m. at CJ Callaway’s April 17 in Sioux Falls, and a webinar will be held at 7 p.m. Central time April 22. The event is free and attendees may bring a guest. Dinner will be provided at CJ Callaway’s. Don’t miss it! Find links to register for both events at www.sdsma.org. A calendar of events is located on the right-hand side of the homepage. Those with questions about online registration may contact Elizabeth Reiss at ereiss@sdsma.org.

Additional resources including past presentations, legal briefs, and much more are available on the SDSMA website! You must be logged in to view SDSMA Center for Physician Resources content online.

Source: SDSMA staff

Retention, Transfer and Disposition of Medical Records

Under state law, medical records must be maintained for at least 10 years. In the case of minors, records must be kept until the child is 20 years old.

The HIPAA mandated privacy rules allow patients copies of their records for as long as they are maintained. After 10 years, records may be destroyed, but an index of all destroyed records must be maintained, and 30-day notice must be given to active patients prior to records destruction. The notice must give a deadline prior to which the records may be claimed.

Prior to destruction of the active patient records, the facility must prepare and retain an index with name of the individual whose records were destroyed, medical record number, date of birth, a summary of visit dates, the attending or admitting physician and diagnosis or diagnosis code.

Records may only be transferred to certain entities named in state law – to another physician, clinic, or other health care facility, a corporation organized for the purpose of operating a health care clinic, or a patient or patient’s representative who has been properly designated in accordance with state or federal law.

State law also specifically addresses the disposition of medical records in case of facility closure. If a facility closes, medical records must be properly maintained, but may be transferred to a different facility to do so.

For more information about medical records, download the SDSMA legal brief Retention, Transfer and Disposition of Medical Records at the all-new www.sdsma.org. Through the SDSMA Center for Physician Resources, the SDSMA delivers programs for members in the areas of practice management, leadership and health and wellness. You must be logged in to view legal briefs and other SDSMA Center for Physician Resources content online.

Source: SDSMA staff

Don’t forget to send in your favorite scenic photo for South Dakota Medicine front cover consideration.
Send photos to ereiss@sdsma.org.
# CME Events

Continuing Medical Education events which are being held throughout the United States (Category 1 CME credit available as listed)

## April 2014

- **April 9**  
  Internal Medicine Grand Rounds: "Implementation of Lung Cancer Screening"  
  12-1 p.m.  
  Sioux Falls  
  AMA PRA Category 1 Credit(s)™ available  
  Register online: www.usd.edu/cmee

## May 2014

- **May 5-9**  
  “Mayo Clinic Practice of Internal Medicine”  
  Mayo Clinic Siebens Medical Education Building  
  Rochester  
  AMA PRA Category 1 Credit(s)™ available  
  Register online: www.mayo.edu/cmee

- **April 17**  
  “Food Allergies for the General Pediatrician”  
  8-9 a.m.  
  Sanford USD Medical Center Schroeder Auditorium  
  Sioux Falls  
  AMA PRA Category 1 Credit(s)™ available  
  Register online: www.usd.edu/cmee

## June 2014

- **June 16-20**  
  “Mayo Clinic Internal Medicine Board Review”  
  Mayo Civic Center  
  Rochester  
  AMA PRA Category 1 Credit(s)™ available  
  Register online: www.mayo.edu/cmee

## DO YOU HAVE A CME EVENT COMING UP? WOULD YOU LIKE TO HAVE IT LISTED HERE?

**Contact:** Elizabeth Reiss,  
South Dakota Medicine,  
2600 W. 49th Street, Suite 200,  
Sioux Falls, SD 57105  
Phone: 605.336.1965  
Fax: 605.274.3274  
Email: ereiss@sdsmalog.org

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If not, contact us to reach over 2,000 physicians!

**CONTACT:**  
Elizabeth Reiss, South Dakota Medicine  
PO Box 7406, 2600 W. 49th Street, Suite 200  
Sioux Falls, SD 57117-7406  
605.336.1965  
E-mail: ereiss@sdsmalog.org
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- Desiree J. Lange, MD
- Michelle J. Bleile, MD
- Jacquelyn Choate, MD
- Shannon Gabriel-Criggs, MD
- Heather Peck, MD
- Jenny Starks, PA
- Kirsten Whalen, PA
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Help Shape the Future of Medicine in South Dakota

The South Dakota State Medical Association Foundation, the philanthropic arm of the South Dakota State Medical Association, is a tax-exempt 501(C)(3) non-profit corporation, was established to assist and support medical research, medical teaching and medical education at the Sanford School of Medicine.

On average, medical students graduate with $130,000 in debt. Contributions to the South Dakota State Medical Association Foundation provide financial assistance to students at the Sanford School of Medicine and are all designated for scholarships, grants and low-interest loans for students.

Any amount can be donated at any time throughout the year. If you have questions or want more information, please call Laura Olson at 605.336.1965.

Send Your Contributions Today To:
South Dakota State Medical Association Foundation
PO Box 7406
Sioux Falls, SD 57117-7406
www.sdsma.org

South Dakota State Medical Association Foundation
Shaping the Future of Medicine in South Dakota
Thank you for putting the ‘CARE’ in DAKOTACARE

In 1986, the physicians of our state created DAKOTACARE because they believed a health care plan should be locally owned and directed. Today, DAKOTACARE continues to improve on making healthcare coverage and services provided by South Dakota physicians a seamless process.

Your involvement is critical to making DAKOTACARE a success. Many South Dakota physicians are currently participating through various committees, work groups or in other capacities, helping to guide the business decisions of our organization. DAKOTACARE’s Medical Management Department, staffed with knowledgeable physicians, pharmacists and nurses work with you to provide quality health care to your patients.

Your ownership and insight puts the “care” into DAKOTACARE.
Be protected, stay cool.

We protect your peace of mind. It’s what we do for medical professionals and specialists. We know your organization is unique. We are too. MMIC provides medical liability insurance coverage, and delivers personalized peace of mind. It’s a movement and we’d love to have you join us.

Contact your independent agent or broker, or go to PeaceofMindMovement.com to see what MMIC can do for you.