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CORRECTIONS
In Memoriam 2018: Philip James Eckhoff, MD. An incorrect photo was used in last month’s issue.
In Memoriam 2018: Buron O. Lindbloom, MD. Dr. Lindbloom was incorrectly listed as DO in last month’s issue.
South Dakota Medicine apologizes for these errors.
Market volatility, this year notwithstanding, tends to unnerv e even the calmest of investors. When the market is dropping, investors like holding assets that maintain their value. When the market is going up, they prefer assets that are going up, not just holding steady. So how do we get the best of both worlds? How do we know what to hold and how much to hold at any particular time? That’s a crucial question, but the answer does not need to be complicated.

The amount of portfolio assets an investor holds in cash/bonds (aka fixed income) should be determined by cash flow needs. Think of it as your lifeboat. If the market is tanking, the last thing you want to do to create cash is to liquidate stock. Ideally, those growth assets would be given time to recover. Buy low, sell high, not the other way around. Investors must be able to remain invested through some difficult market downturns to achieve better long-term results. Preservation assets like bonds help buffer these near term bumpy roads and reduce investor angst.

So what exactly are “cash flow needs”? Think of someone nearing or in retirement. Income from employment has or will be ceasing. Thus the portfolio becomes a source for maintaining a certain standard of living. In preparation for that time, individuals should take steps to have enough cash and bonds in their portfolio to cover approximately eight years’ worth of their distribution needs. Historically, this level of cushion has been enough to weather virtually all market corrections. If stock and/or real estate markets decline, this lifeboat of preservation assets provides ample liquidity to sustain us while we wait for an expected recovery.

What if you do not have any immediate cash flow needs from the portfolio? Depending on your time horizon, it may make sense to have an investment line-up exclusively allocated to the equity markets. If growth is the goal for the foreseeable future and there is an understanding that markets will go up and down, then an all-equity portfolio can be reasonable. Advantages of having some fixed income exposure include having “dry powder” for rebalancing, as well as a degree of diversification for risk management.

For example, when stock markets declined in February, investors with fixed income in their portfolios were afforded the opportunity to deploy additional capital into those equity markets, in effect, “buying low”. As the market turns upward in later time periods, the investor owns more shares that have been purchased at lower prices. Simple stuff.

Remember, buy low, sell high. This tactic is not based upon a prediction nor timing effort. That’s fools’ gold. Rather, it is predicated on what percent of your portfolio ought to be in the stock market versus out of the stock market. What does your plan suggest the portfolio needs to do to meet your individual goals? From there, you have no need to take more risk than necessary. Build a diversified, low-cost portfolio in accordance with your plans. Keep the lifeboat intact and stay diversified. Wait, did I already say that?

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Putting Patients First

By D. Bruce Eaton, MD

The importance of giving patients our full attention cannot be overstated. Unfortunately, due to the increasing distractions of EMRs and other administrative burdens, it can become a more difficult task than we care to admit, and these distractions can affect our relationships with patients. Study after study have shown that excessive administrative tasks divert physicians’ time and focus away from patient care, and may decrease our career satisfaction. The South Dakota State Medical Association continues to advocate for a reduction in the regulatory and administrative requirements that we must comply with. What can we do to increase our satisfaction, improve our patients’ experiences, and how can we work to find our jobs more gratifying? I’ve invited Dr. Bruce Eaton, internal medicine physician at Regional Health in Rapid City, to write about his experiences – and what solutions have worked for him in putting his patients first.

Christopher T. Dietrich, MD, SDSMA President and Physical Medicine & Rehabilitation Physician

We are all aware of the positive impact technology has on our lives. We have the ability to communicate instantly with anybody, anywhere, anytime. Information is at our fingertips, immediately accessible when we need it, organized, synthesized and analyzed. It allows us to be effective and efficient in our daily lives, our increasingly hectic days are more manageable.

But as a society technology is diminishing the necessity of direct human interaction for survival. I believe this is having a detrimental effect on us as individuals, as well as the communities in which we live. When we do have the opportunity to socialize, we are often distracted by an email or text. Ironically, despite this “enhanced” communication, many feel more isolated and alone. We are all aware of the problem with texting and driving or “distracted driving”, a very dangerous practice with frequently fatal consequences.

I feel an equally important issue is “distracted patient care.” The requirements placed on physicians to complete an office visit are daunting, particularly in a 15-20 minute time slot. The more complex the patient, the more hoops (distractions) to jump through. Complex patients often require us to search for, and extract data, scanning for useful morsels of information in templated haystacks of patient notes. We have to order tests and justify it to the computer. Only after you have done this are you allowed to advance through the game of coding and billing... making sure you don’t accidentally select “weightlessness” instead of “weight loss” for your diagnosis. All a maze of button clicking: taking your attention away from the other human being in the room. I have been on the other side of that keyboard and it doesn’t feel very good. It’s no wonder patients feel their doctors don’t care as they did before, and physicians in record numbers are getting burned out.

These peripheral tasks all are part of a system of health care that has forgotten its primary purpose and have left physicians feeling like data entry specialists. It feels as if the train has left the station and we have no control.

I have a solution. Take that one patient each day, the one who is complicated, frustrated, their diagnosis or best treatment remains elusive. Turn away from the computer, take the time, be in the moment. Listen, really listen. Perform that thorough physical exam you were taught in medical school and discuss your differential diagnosis, treatment options, and come up with a treatment plan together. You may not diagnose or cure their problem, but a working physician/patient relationship will have developed to create a path forward. You will both feel as if you have accomplished something. Your job will be more rewarding.

Think back to when you were a medical student. When interviewing the patient you often were trying to think of your next question before you actually heard the answer from the last one. When we grow as physicians, the process becomes more natural. Trying to determine whether that chest pain is cardiac or not requires a nuanced line of questioning, listening to the answer, and formulating the next question based on that answer. When looking at a screen you miss nonverbal cues. With this approach, the patient knows they have been heard, a relationship of trust has been developed and you as a physician have done what you went in to medicine to do.

Yes, we do need to function in todays world of health care, but in the end, its about caring for the patient first and foremost, and in turn, ourselves. We need to treat our patients, with our full attention, undistracted by technology or business. I have found on days that I am able to do this I feel better. I feel like a doctor.
The New USS South Dakota (SSN-790)

By Keith Hansen, MD

“Subter Mare Dominamur”
“Under the sea we rule”

“Under the sea we rule” is the motto of the U.S. Navy vessel christened the USS South Dakota. On Feb. 2, the newest Virginia-class attack submarine, the USS South Dakota (SSN-790), was commissioned at Groton, Connecticut. SSN-790 will be the third U.S. Navy vessel with the name USS South Dakota.

In 1904, the first USS South Dakota (ACR-9) launched and was assigned to the Armored Cruiser Squadron of the Pacific Fleet. Initially the South Dakota was located off the West Coast of the U.S. During WWI it was involved with escorting troop convey to the mid-Atlantic, where British vessels took over the duty and escorted the troop carriers the rest of the voyage. Following WWI the South Dakota was sent back to the Pacific to serve as the flagship of the Asiatic fleet and renamed the USS Huron (after Huron, South Dakota) to release the name South Dakota, so it could be used for the next U.S. Navy vessel.

Initially the battleship BB-49 was to bear the name, South Dakota. BB-49 was a new type of battleship and the South Dakota was to be the lead of a six-ship class of post-WWI battleships. However, the plans were discontinued for these battleships after the Washington Accord, an agreement to limit the size of warships to hopefully reduce the risk of another World War.

In June of 1941, the second USS South Dakota (BB-57) was launched and christened by Mrs. Harlan J. Bushfield, wife of the governor of South Dakota at the New York Shipbuilding yards in Camden, New Jersey. The South Dakota was christened twice which is unusual for a Naval vessel. The reason for the double-christening is Mrs. Genevieve Trask, of Pierre, and friend of the governor, asked if a special bottle of champagne she had received from her husband at their wedding could be used for the christening. The governor’s office, South Dakota’s congressional delegation, and the Navy approved the use of Mrs. Trask’s champagne, but the other bottle of champagne also had to be used. At the time of the christening, Mrs. Bushfield broke Mrs. Trask’s bottle of champagne across the bow of the ship, and at the same time an employee of the shipyards broke the official bottle. Following launch and after crossing to the Pacific, in mid-October 1942, the USS South Dakota was assigned to Task Force 16, part of the USS Enterprise battle group. During WWII, the American public knew the USS South Dakota as “Old Nameless” or “Battleship X.” During WWII, the USS South Dakota served in both the Pacific and Atlantic oceans. She was involved in almost every major naval engagement in the Pacific and was the first to fire on the Japanese mainland. At the Japanese surrender in 1941, the USS South Dakota was anchored in Tokyo Bay. The USS South Dakota served with distinction and was awarded 13 battle stars. In January 1946, the USS South Dakota (BB-57) was placed in a reserve status and scrapped soon after. Some parts of the USS South Dakota were preserved and sent to Sioux Falls as part of the South Dakota Memorial Foundation Museum.

The newest vessel named South Dakota (SSN-790) is a nuclear powered, Virginia-class attack submarine, 17th in the fleet. The South Dakota is 377 feet long with a beam (greatest width at waterline) of 34 feet. She will be able to dive to greater than 800 feet and have a submerged speed of greater than 25 knots (greater than 28.8 miles per hour). The South Dakota will not have to refuel during its expected 30-year lifespan. As a Virginia-class submarine, the South Dakota is a multi-mission platform to carry out the missions of the submarine force. The boat’s crest pays tribute to the state of South Dakota and to the previous vessels that carried the name. First, the iconic Mount Rushmore sits in the upper middle portion of the crest. Mount Rushmore is surrounded by three coyotes, with our state bird’s (pheasant) feathers along the side. Also hanging from the side is a rattlesnake tail, the one venomous snake in South Dakota that represents the deadly stealth of the USS South Dakota. The crest also has 13 stars, which commemorate the 13 battle stars of BB-47 from WWII. The crest also has two dolphins representing the outstanding crew, a silver one representing the enlisted crew and gold the officers. The USS South Dakota (SSN-790) joins a proud and distinguished group of vessels named for the 40th state and is sure to carry on the proud traditions of the submarine force and the U.S. Navy.

RESOURCES

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“Every locality has a different perspective on what is most important for providing excellent health care. Everyone must be represented when policy is being crafted.”

Mary Carpenter, MD
AMA MEMBER SINCE: 1982
South Dakota
AMA House of Delegates

“The AMA and SDSMA help me “filter out” all the noise in my practice, which allows me to focus on the issues that are most important to me—my patients, the practice of medicine and my fellow physicians.”

Robert Allison, MD
AMA MEMBER SINCE: 1991
South Dakota
AMA House of Delegates
Annular Pancreas in Adults: Case Report and Literature Review

By Jeffrey Edman; Hassan Turaihi, MD; Bhavesh Patel, MD; Gary Timmerman, MD; and Thavam Thambi-Pillai, MD;

Abstract
Annular pancreas is an uncommon congenital cause of gastric outlet obstruction. The incidence is usually referenced at between five and 15 per 100,000 based on autopsy series. When present, this rare condition surfaces with symptoms in the pediatric population during the first few months of life. An adult presenting with symptoms of gastric outlet obstruction due to annular pancreas is an unusual incident. This case describes gastric outlet obstruction due to a partial annular pancreatic band in an otherwise healthy 32-year-old male. Given the scarcity of this pathological process in adults; no specific guidelines exist about the management of this condition. Continued reporting of this pathology is essential for development of such guidelines. Literature review, embryology and treatment options will be discussed.

Introduction
Annular pancreas incidence is usually referenced at five to 15 per 100,000 based on autopsy series, but due to the often asymptomatic nature of the abnormality concrete numbers are difficult to generate. Embryologically, the pancreas is formed during the fourth to eighth weeks of life by ventral pancreatic bud fusion with the dorsal pancreatic bud during rotation of the foregut. Failure of the fusion in time with elongation of the ventral bud towards the dorsal bud can lead to pancreatic tissue partially or completely encircling the descending duodenum. In this case we look at an adult male with nonspecific gastrointestinal symptomatology that ended up having a pancreatic tissue partially encircling the duodenum causing stenosis. This case is unique due to the age of presentation and unequivocal early imaging studies due to only partial encircling of the duodenum by pancreatic tissue.

Case Report
A 32-year-old male with no pertinent past medical history presented to his primary care physician with worsening symptoms of abdominal pain, fullness after meals, dysphasia, and eructation. The patient endorsed he had been suffering from these symptoms since the age of 29 and has lost around 30 pounds during this timeframe. These symptoms were the worst after meals and when lying down. He denied loss of appetite or change in bowel habits. Family history was negative for inflammatory bowel disease and hereditary colorectal cancer syndromes.

Initially, the patient had tried non-steroidal anti-inflammatory drugs (NSAIDs) for short term relief with little improvement of his discomfort. The patient was referred by his primary physician to the gastroenterology (GI) department with recommendation for esophagogastroduodenoscopy (EGD).

At EGD, findings showed severe stenosis involving the second portion of the duodenum and distal esophageal mucosal changes suggestive of eosinophilic esophagitis (EoE). Gastric and duodenal biopsies showed no evidence to suggest eosinophilic gastroenteritis. Biopsies obtained from the stenotic area showed nonspecific inflammation. Further specific testing for celiac disease, H. Pylori and Crohn’s disease were all negative. To further evaluate the duodenal anatomy computed tomography (CT) enterography was obtained, which confirmed the nonspecific
narrowing of the second portion of the duodenum (Figure 1). Following a month of oral proton pump inhibitor (PPI) use, the patient reported only minimal improvement in his symptoms and so endoscopic ultrasound (EUS) was recommended. During this evaluation, severe duodenal bulb dilation was noted as a result of the previously mentioned stricture involving the second portion of the duodenum. On EUS there was no evidence of mass or malignancy causing these pathological findings, but annular pancreas was suspected. This prompted a magnetic resonance imaging (MRI) enterography, which supported the EUS findings suggestive of annular pancreas. An upper GI contrast swallow study with small bowel follow through was done as well with findings consistent with focal duodenal stenosis in the proximal second portion of duodenum (Figure 2). With all the imaging studies suggesting annular pancreas the patient was referred to the surgery clinic.

At the pancreatic-biliary surgery clinic treatment options were discussed. The main option discussed was surgical intervention to bypass the stenotic region using a duodenoduodenostomy or duodeno-jejunostomy dependent on intraoperative findings. This is the gold standard currently and hopefully would provide a permanent fix of the problem. The patient agreed with this plan and after discussing the risks and benefits of surgery the patient was scheduled to undergo an open duodeno-duodenostomy.

After obtaining informed consent, the patient was taken to the operating room for exploratory laparotomy. Once inside the abdominal cavity, a thorough inspection demonstrated a tight band of pancreatic tissue encircling the second portion of the duodenum correlating with the preoperative workup (Figure 3). To provide better exposure of the duodenum a generous kocherization of the duodenum was performed. This technique allowed mobilization of the duodenum and the head of the pancreas from their retroperitoneal attachments. Once freed we were able to fold the proximal dilated segment of the duodenum over the distal decompressed segment (Figure 4). To bypass this area of constriction, enterotomies were created and a hand sewn functional anastomosis was created (Figure 5). A nasogastric tube was positioned across the anastomosis to provide decompression of the duodenum until healing occurred. A surgical drain was positioned in the surgical field to provide drainage of any fluid in the event of anastomotic leak. The patient tolerated the procedure well with an estimated blood loss of 30 mL.
The postoperative course was uneventful with the nasogastric tube removed on post-operative day three and diet advanced to a soft diet. The patient was discharged home on postoperative day four, with instruction to resume regular diet after 10 days following his discharge. The patient reported complete resolution of his symptoms during his routine one-month postoperative follow up visit.

**Discussion**

Annular pancreas incidence is usually referenced between five and 15 per 100,000 based on autopsy series, due to the sometimes asymptomatic nature of the abnormality concrete numbers are difficult to generate. Embryologically the pancreas is formed during the fourth to eighth weeks of life by ventral pancreatic bud fusion with the dorsal pancreatic bud during rotation of the bowel. Failure to do so properly with elongation of the ventral bud towards the dorsal bud can lead to pancreatic tissue partially or completely encircling the second portion of the duodenum. A symptomatic annular pancreas normally presents during the neonatal period, but can present at any age. Infancy and the fourth decade of life are the two most common times. Neonates with this pathology are commonly diagnosed early, usually they will have more severe disease leading to early symptoms. Adult manifestation comes from less severe disease and results in vague symptoms involving multiple systems. Annular pancreas in adults can present with elevated liver function tests and right upper quadrant pain following fatty foods mimicking liver or biliary tree origin. Other presentations may include small bowel obstruction with nausea/vomiting and stomach distension. Rarely annular pancreas may present as severe epigastric pain radiating to the back associated with acute pancreatitis from damage to the pancreatic tissue leading to inflammation.

Once annular pancreas is suspected, diagnosis is typically made with imaging studies. CT is the most helpful diagnostic tool although ultrasound (US) and MRI are helpful as well.

EGD, upper gastrointestinal (GI) series or EUS can be helpful in confirming the diagnosis as well. Lab values that suggest annular pancreas are elevated lipase, amylase, and LFT’s, although these are nonspecific and imaging is necessary as a result.

Treatment involves bypass of the annular pancreas by either a duodeno-duodenostomy or duodeno-jejunosotomy. These are the preferred options over resection of the
annular pancreas due to decreased risk of pancreatic damage leading to recurrent pancreatitis and the increased efficacy of bypass.\(^5\) Decision between the duodeno-duodenostomy and duodeno-jejunostomy is dependent on the case and how much bowel is available to be wrapped up towards the first section of the duodenum. The second through fourth portions of the duodenum lie retroperitoneal which sometimes makes mobilization difficult and necessitates the usage of the jejunum for anastomosis. Ideally, we would look to use the minimal amount of bowel required to create a functional anastomosis and circumvent the stenosis.

Prognosis is generally good for these patients with a normal life span provided the diagnosis is made before duodenal perforation. Vague symptoms can plague adults for years before annular pancreas is even suspected, but a duodeno-duodenostomy can alleviate symptoms with minimal adverse effects or risks besides those from a typical bowel surgery.\(^5\)

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J.L. is a pleasant 66-year-old gentleman, known to have liver cirrhosis secondary to hepatitis C, complicated by ascites. He was seen by the hepatology clinic for hepatitis C treatment. During one of his clinic visits, he complained of abdominal pain and bloating, along with postprandial fullness. He also had some nausea but no vomiting. The ascites was controlled with diuretic therapy. A physical exam revealed the stigmata of liver disease, including spider angiom as and palmar erythema. His abdomen was soft and distended with no tenderness or flank dullness. The liver edge was palpable upon inspiration. He also had bilateral edema in his lower extremities. The patient's lab work was unremarkable, including complete blood counts and the basic metabolic panel. His alkaline phosphatase was 194, and his HbA1c was 5.4. Subsequently, J.L. received an abdominal ultrasound, which showed a normal-sized spleen with no significant ascites. Additionally, his gallbladder was partially distended. The common bile duct measured 5.1 mm.

Eventually, the patient underwent an esophagogastroduodenoscopy for further evaluation. He was found to have a large amount of food in his gastric body, though he hadn’t eaten for more than 12 hours prior to the procedure. There was no evidence of varices or portal gastropathy. Given these findings, the patient was referred for a gastric-emptying study. After he was asked to stop proton pump inhibitors for 24 hours and stop pain medications and antiemetics for three days, he underwent a nuclear gastric-emptying study using a Tc-99m sulfur colloid, which revealed a severe delay in gastric-emptying (13 percent at 60 minutes, 14 percent at two hours [normal is 40 percent or greater], and 57 percent at four hours [normal is 90 percent or greater]). The patient was diagnosed as having gastroparesis, thought to be related to liver cirrhosis, given his symptoms and the absence of other explanations. He was started on dietary modifications and prokinetic medications, resulting in some improvement in his symptoms.

Gastroparesis is defined as delayed gastric-emptying without evidence of mechanical obstruction of the stomach. The age-adjusted prevalence of definite gastroparesis is estimated to be 24.2 per 100,000 for both genders. However, it is unclear how often patients with gastroparesis are referred to gastroenterologists or if they even seek health care for their symptoms, and therefore the true prevalence of gastroparesis is not known and tends to be underestimated. This is alarming, as the disease is associated with substantial morbidity and mortality rates.

In terms of the etiology of gastroparesis, diabetes mellitus is the most recognized systemic illness associated with it. It is reported in 29 percent of the patients in tertiary referral settings. The other potential etiologies are mainly postsurgical, idiopathic, and iatrogenic. Interestingly,
most gastroenterology societies ignore the fact that liver cirrhosis is a major cause of gastroparesis.

Liver cirrhosis has significant effects on the gastrointestinal (GI) tract, as it associated with malnutrition and structural changes such as esophageal varices and portal hypertensive gastropathy. And the GI effects go even beyond that, to sensorimotor abnormalities. Early studies pointed specifically to delayed gastric-emptying in liver cirrhosis. However, there was conflicting data from other studies reporting normal or even accelerated gastric-emptying. Many factors contributed to this controversy, including selection bias, small study populations, and the use of different measurement methods. With that in mind, it’s worth noting that the prevalence of gastroparesis in patients with liver cirrhosis ranges from 24 percent up to 95 percent. These numbers indicate that delayed gastric-emptying in patients with liver cirrhosis is more common than we previously thought and may contribute to their GI symptoms, poor oral intake, impaired oral drug absorption, and impaired physical and mental health-related quality of life.

The exact mechanism of delayed gastric-emptying in liver cirrhosis is poorly understood. In the past, GI symptoms were mostly attributed to the compression of ascites or enlarged spleen on the stomach and small intestine. More recently, many studies have showed several factors that may contribute to gastroparesis in cirrhotic patients even in the absence of ascites or splenomegaly. One of the postulated theories relates to GI hormone abnormalities — namely, the hormones motilin, cholecystokinin, glucagon, insulin, ghrelin, and secretin. Autonomic dysfunction has also been considered a contributing factor to gastric dysmotility in cirrhotic patients. In fact, autonomic neuropathy is recognized in up to 68-80 percent of patients with liver cirrhosis. On the other hand, gastric antral vascular ectasia and portal hypertension have also been suggested as possible causes of abnormal gastric motility in cirrhosis. The effect of portal hypertension goes beyond the stomach, as it causes delayed small intestine transit time. Dimitrascu et al. (1997) implicated the type of meal eaten as a major determinant of gastric-emptying in cirrhotic patients, which may account for the large divergence in results published on this topic.

The symptoms of gastroparesis are mostly the same despite the different etiologies, including liver cirrhosis. In general, the most commonly reported symptom is nausea (92 percent) followed by vomiting (84 percent), abdominal pain (75 percent), and early satiety (60 percent). In liver cirrhosis, other reasons for the above-mentioned symptoms should be excluded as GI symptoms reported in up to 80 percent of patients. In addition, cirrhotic patients have a higher prevalence of peptic ulcers and Helicobacter pylori infections. All of these conditions could potentially produce similar symptoms as those of gastroparesis, which creates a significant overlap between these conditions and makes the diagnosis of gastroparesis even more difficult.

In terms of management, gastroparesis in liver cirrhosis is not different from any other etiologies. Dietary modifications and the use of prokinetic and antiemetic agents are the cornerstone of treatment. Early reports have shown Cisapride to be effective in cirrhotic patients specifically because it can shorten the gastric-emptying time, but its use has been limited because of its side effects. In addition, it’s worth noting that 63 percent of patients with cirrhosis and autonomic dysfunction showed improvement after liver transplantation.

In conclusion, gastroparesis in liver cirrhosis is a unique entity and proven to be present despite the controversies around it. It could potentially explain many of the GI symptoms in cirrhotic patients and affect their mental and physical health. Unfortunately, this entity is not recognized by major gastroenterology societies, for many reasons. Future studies with a large number of patients are needed to address this topic more extensively.

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The earliest pacemakers were invented in the 1920s simultaneously by two physicians in Australia and the U.S. Like most medical advances, the earliest renditions of the machines were not well received and were initially rejected by the medical community. The technology continued to improve and the medical community did readily adopt external pacemakers in the early 1950s and by 1959 the first right ventricular endocardial catheter was introduced through the jugular vein. The advent of transistor technology made it possible for fully implantable pacemaker to become available in the 1960s. However, these devices were bulky and only had a life span of about three years. These implanted devices typically consisted of trans-venous leads attached to a generator which was housed inside of a subcutaneous pocket. From the 1970s to the 2000s, there were significant advances in the technology of implantable pacemakers including improvements in battery life, improved materials, improved algorithms for pacing, and improvements in the targets for pacing such as bi-ventricular pacing.

The next big shift in paradigm came in 2012 when the first fully implantable leadless cardiac pacemakers were introduced. Two major types of leadless pacing devices have been designed: single component and multicomponent. Single component devices have all the sub components (like battery, pulse generator, electrode) encased within a single unit whereas multicomponent devices have two separate components, subcutaneous battery unit transmitting energy wirelessly to separate endocardial pacing unit. There are currently two single component devices with fully contained leadless systems: Micra Transcatheter Pacing System (TPS) and Nanostim Leadless Cardiac Pacemaker (LPS). At this point, Micra has received FDA approval and it is commercially available in the U.S. All multisystem devices are investigational at this point.

Since the leadless cardiac pacemaker is a single chamber pacing device, the FDA has approved it for a select group of patients: those that have symptomatic AV block in the presence of atrial fibrillation (AF). Other situations in which leadless Micra pacemaker may be indicated arise when dual chamber lead placement is difficult or high risk in those with symptomatic AV block in the absence of AF or symptomatic bradycardia-tachycardia syndrome.

Micra measures 25.9 x 6.7 mm and uses four self-expanding nitinol tines hooking into the myocardium. Nanostim measures 42 x 5.9 mm and uses an active screw in helix with maximum penetration of 1.3 mm with three angled nitinol lines running perpendicular to helix for secondary fixation of the helix. Both devices are MRI safe. From the available short-term data, it is estimated that the lifespan of Micra system is 12.5 years compared to 15 years of the Nanostim system.

Similar to standard pacemakers, leadless cardiac pacemakers should be implanted by an experienced cardiac electrophysiologist or a non-electrophysiologist cardiologist with special training in implantation. Leadless cardiac pacers are implanted through a percutaneous, minimally invasive approach. The overall procedure for both devices is similar except for use of different size equipment and different deployment process.

Since the Micra system is the only FDA approved device available in the US for clinical use, we discuss the technique for its implantation: The femoral vein is accessed using an introducer needle. Once a long guidewire is advanced to the inferior vena cava, the introducer needle is removed and the femoral vein is dilated. Once the dilator is removed, an introducer sheath is inserted which ends in the mid atrial location. The Micra delivery system is advanced through the introducer sheath and retracted toward the inferior vena cava prior to insertion into the right ventricle. The Micra system is
positioned at the apical septal position using intravenous contrast. Then pressure is added along the system and the Micra is deployed. Two out of four tines need to be inserted in the tissue to confirm adequate deployment. Electrical measurements are then taken by a programming device on the patient’s chest. Once measurements are confirmed, the system is removed and the vein access site is closed. There is no trans-venous lead and no subcutaneous pocket. Patients are typically monitored on hospital telemetry for 24 hours post op prior to discharge in uncomplicated cases.

The major benefit of leadless cardiac pacing is that two potential sources of complications are eliminated which include the pacemaker leads and the generator pocket. Examples include lesser or no rates of pneumo/hemothorax, hemorrhage or hematoma, pacemaker infection, wound/local infection, lead related re-intervention, pacing threshold elevation requiring re-intervention, pocket revision.6

The complication rates when compared to conventional pacers should be considered when using leadless pacemakers. Early data reveals a higher overall short-term complication rate of 4.8 percent with leadless pacers compared to 4.1 percent with conventional pacers. There is also an increased risk of cardiac perforation, 1.5 percent with leadless pacers vs. 0.1 percent with conventional pacers. However, the learning curve for novel leadless devices and the possibility of underreported complications from earlier trials for conventional pacers must be kept in mind as a possible explanation for these results.6 Femoral access site complications, are unique to leadless pacers due to their size and large sheath insertion (0.9 percent vs. 0 percent).6

There are some other limitations that one must consider given the type of device. Currently only single chamber pacing devices are available for clinical use. However, with the dual chamber fully implanted devices in development, there is a possibility that this will not be a limitation soon. Currently, there is no available option of leadless pacing device combined with defibrillator. However, there is a possibility of combining the leadless pacemaker with a subcutaneous pacemaker to minimize implanted leads.

While in the initial stages of evolution and development, leadless pacing devices have shown tremendous potential. The higher complication rate documented in the available studies may improve with increasing experience among
implanters of leadless devices. The shorter studies so far have been promising but longer prospective studies will be required to investigate long term safety and efficacy.

**REFERENCES**


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Introduction
Community-acquired pneumonia (CAP) is a common problem in the U.S., resulting in approximately 600,000 hospital admissions and $10.6 billion in healthcare costs annually.1 Traditionally, confirmation of a specific etiologic agent has relied on sputum and blood cultures, serology, and/or urinary antigen testing. Despite these testing methods, a specific pathogen has been identified in less than 40 percent of CAP cases.2 Thus, treatment recommendations have been focused on empiric antibacterial therapy.3 This leads to potential antibiotic misuse or overuse with the attendant risks of antimicrobial resistance, adverse effects including C. difficile infection, and excess cost. Rapid molecular diagnostic testing has been proposed as one technique to improve antibiotic stewardship and specifically, this may improve antibiotic utilization in CAP.4 Multiple studies have shown that multiplex polymerase chain reaction (mPCR) assays for respiratory pathogens substantially increase pathogen identification and do so in less time.5-8 In one specific study, pathogens were detected in 87 percent of patients with CAP compared to only 39 percent with culture-based traditional methods.9 The mPCR testing also claims increased sensitivity/specificity. The Biofire FilmArray manufacturer quotes a sensitivity range from 87.4 to 100 percent, and specificity from 89.1 to 100 percent for viral and bacterial samples.10 Other studies have also concluded that the positive predictive value for bacterial atypical CAP is higher because these causes are not seasonal and present with nonspecific symptoms.11 With the recent availability of rapid turnaround molecular pathogen detection platforms in clinical microbiology laboratories (results in approximately one hour if the test is run immediately), clinicians have the opportunity to improve viral detection which may lead to improved antibiotic utilization.10 However, if the result is positive for a virus, the next step occurs at the discretion of the physician, and antibiotics are not stopped automatically. Studies evaluating antibiotic utilization have shown inconsistent results.4 The goal of our study was to assess differences in antibiotic utilization between patients with and without mPCR testing in patients who were discharged with a primary diagnosis of pneumonia.

Methods
This single-center retrospective cohort study included patients aged 18 years and older who were discharged from a 550-bed tertiary care hospital with a diagnosis of...
pneumonia (DRG 193-195: simple pneumonia and pleurisy) between Oct. 1, 2015 and Dec. 31, 2016. Data was extracted from the Sanford One Chart electronic medical record system (EPIC Systems, Verona, WI) by a single author of the study. Data was recorded on table of variables form on a secured electronic database. Accuracy of data transfer was improved upon by having a single person reviewing all of the records and by limiting data to objective findings: antibiotic history, discharge orders, and laboratory testing available via Epic electronic health records. The primary outcome of interest was antibiotic use. Other data collected included demographic data and diagnostic testing for respiratory pathogens. mPCR testing was performed on nasopharyngeal samples using the FilmArray 2.0 Respiratory Panel (BioFire Diagnostics LLC, Salt Lake City, UT). Respiratory pathogens tested in the panel were adenovirus, coronavirus, human metapneumovirus, human rhinovirus/enterovirus, influenza A, influenza B, parainfluenza, respiratory syncytial virus, Mycoplasma pneumoniae, Chlamydia pneumoniae, and Bordetella pertussis. The study protocol was approved by the Sanford Health Institutional Review Board. Continuous variables were summarized as means and were analyzed using one-way analysis of variance (ANOVA). Discrete variables were summarized as counts and percentages.

Results

A total of 233 discharges met criteria for inclusion and were reviewed. Of these patients, 70 patients underwent mPCR testing, including 42 men (60 percent) and 28 women (40 percent). The average age of the tested population was 69 years. mPCR testing was negative in 53 patients (mPCR- 76 percent) and positive in 17 patients (mPCR+ 24 percent). Among the mPCR+ tests, 16 (94 percent) were positive for a viral pathogen and one (6 percent) was positive for a bacterial pathogen (Table 1). In the mPCR tested subset of 70 patients, 22 (31 percent) underwent simultaneous testing for influenza via other methodologies, all of which were negative. Sputum cultures were performed in 59 patients (84 percent) who also received mPCR testing (Table 2). Of the patients with sputum cultures 10 were positive. Four of the sputum cultures grew respiratory pathogens, including Streptococcus pneumoniae, Staphylococcus aureus, and Haemophilus influenzae, while the remaining six grew mixed flora, Candida albicans, and Enterococcus faecalis. Lastly, blood cultures were drawn from 63 of the 70 patients that received mPCR testing (90 percent) and two (3 percent) returned positive for Staphylococcus aureus and Pseudomonas aeruginosa.

In examining antibiotic utilization, 66 patients (94 percent) were treated with intravenous antibiotics while hospitalized. Thirty patients (46 percent) continued antibiotics after discharge, all of which were orally administered. The average total duration of antibiotics for the mPCR- group was 8.3 days (range 0-15) compared to 4.9 days (range 0-12) in the mPCR+ group with a viral pathogen ($p = 0.0004$).

Discussion

In this cohort, the majority of patients admitted for pneumonia (70 percent) did not undergo mPCR. The reasons for this are unknown and a potential point for future exploration. Among the subset of patients who underwent mPCR testing, 22 (31 percent) had a pathogen identified, including 17 (24 percent) patients with a positive mPCR and five patients (7 percent) with positive sputum or blood cultures. One mPCR+ patient also had a concomitant positive sputum culture, which grew S. pneumoniae. This finding is similar to other published studies in which the majority of patients hospitalized with pneumonia do not have a pathogen identified. In studies focused on identifying pathogens to fill this gap, viral

<table>
<thead>
<tr>
<th>Table 1. mPCR findings</th>
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<tbody>
<tr>
<td>Pathogen</td>
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<tr>
<td>Influenza A and B</td>
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<tr>
<td>Respiratory syncytial virus</td>
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<td>Human metapneumovirus</td>
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<td>Rhinovirus/enterovirus</td>
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<tr>
<td>Parainfluenza 1-3</td>
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<tr>
<td>Coronavirus HKU1, NL63, 229E, OC43</td>
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<td>Mycoplasma pneumoniae</td>
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<table>
<thead>
<tr>
<th>Table 2. Additional testing for respiratory pathogens*</th>
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<tbody>
<tr>
<td>Diagnostic test</td>
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<tr>
<td>Rapid influenza diagnostic test (RIDT)</td>
</tr>
<tr>
<td>Respiratory virus direct fluorescent antibody</td>
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<tr>
<td>RIDT and PCR</td>
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<tr>
<td>Influenza PCR only</td>
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<tr>
<td>Sputum culture</td>
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* No viral pathogens were identified through these diagnostic tests.
Nearly 3 out of 10 mothers smoked prior to pregnancy.*

South Dakota PRAMS-like survey data indicates that 28% of all South Dakota mothers smoked sometime in the 2 years before pregnancy, and almost half (13% of all mothers) smoked during the last 3 months of pregnancy.†

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   - Up to 4 additional relapse prevention calls with a QuitLine coach
   - Gift card incentives if eligible
   - For more on how to refer your patients: SDQuitLine.com/providers/referral-options

2. Smokefree Mom
   SmokefreeMOM is a mobile text messaging service designed for pregnant women across the United States to help them quit smoking.
   - For program information, visit women.smokefree.gov/smokefreemom.aspx

†For full report: dsh.sd.gov/documents/statistics/2016-SD-PRAMS.pdf. Data from 2016 South Dakota PRAMS-like survey were weighted to provide statewide estimates.

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pathogens have been detected at varying rates from 30-87 percent. These differences have been attributed to variations in test characteristics between mPCR platforms, populations studied, and specimen type (upper versus lower respiratory tract). We showed a viral detection rate of 23 percent, a rate which potentially limits the utility of mPCR in the sampled population.

While diagnostic testing for viruses is frequently employed, the impact of the testing on antibiotic utilization is variable. Three studies showed no difference in the duration of the antibiotic use in hospitalized patients. However, other studies have demonstrated a decrease in total duration of antibiotics, especially when combined with antibiotic stewardship interventions. In our study, we found a statistically significant reduction in the total duration of antibiotics in patients who had a viral pathogen detected by mPCR. This suggests that mPCR testing may be an effective antibiotic stewardship intervention. However, there are multiple limitations to be considered. Most notably, in the group of mPCR-positive patients, the average duration of antibiotics was eight days. While clinical trial data are limited, current recommendations are to treat CAP for five days or until afebrile for 48-72 hours and no more than one sign of clinical instability. Recent studies have shown that five days is not inferior to longer courses of antibiotics. Regardless of improved diagnostic testing and pathogen identification, we would likely decrease antibiotic utilization by simply improving clinician adherence to current data and guidelines.

Another confounding issue is interpretation of mPCR results. In the Epidemiology of Pneumonia in the Community Study, among 238 asymptomatic control patients, 2 percent were positive for any respiratory pathogen and 1 percent were positive for rhinovirus. More recent work by Shaman, et al. showed depending on the definition of symptomatology, 58-93 percent of persons with positive mPCR testing were asymptomatic. Similarly, Gadsby et al. found 82 percent of the positive mPCR tests positive for viral pathogens on sputum were also positive for bacterial pathogens. These findings highlight the problem that mPCR may confirm the presence of a virus; however, it does not confirm that as the cause of acute disease. Investigators are actively investigating the potential for quantification of pathogens as a marker for acute pathogenicity.

In light of the outlined limitations, mPCR is a potentially powerful tool in clinical microbiology. The exact role of mPCR in current clinical practice remains unclear and further work is needed to find the most clinically useful and cost effective applications. However, the potential for mPCR to improve antibiotic stewardship is significant as antibiotic misuse or overuse is associated with significant costs including: drug resistance, nosocomial infections, and increased length of stay. mPCR testing may also decrease the need for additional ancillary testing for CAP such as urinary antigens. Based on the data from our cohort, interventions to support test stewardship, including avoidance of duplicate testing, and enhanced guideline adherence remain the cornerstone of antibiotic stewardship.

Conclusion

mPCR for detection of respiratory pathogens is a now widely available tool in microbiology laboratories, which has increased the capability of identifying viral pathogens in particular. At this point, best practices surrounding use of mPCR remain unclear.

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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Screening for Impaired Glucose Tolerance (Prediabetes) and Prevention of Type 2 Diabetes

By Mohamed A. Abdallah, MBBS; Khalid Mohamed Ahmed, MBBS; Dennis Stevens, MS, MD; and Marcio Griebeler, MD

Abstract
The enormous implications caused by type 2 diabetes on patients, families and health systems in the U.S. require health care providers to apply measures that reduce its burden. Scientific evidence clearly shows that proper screening of populations at risk, implementation of interventions proven beneficial in preventing type 2 diabetes and the use of modern technology in educating patients to adopt a healthier lifestyle are paramount in decreasing the incidence of type 2 diabetes. In this article, we try to answer some of the questions raised by both patients and health care providers about how lifestyle modifications can play a key role in ameliorating and reversing impaired glucose tolerance and type 2 diabetes.

Introduction
Type 2 diabetes is a leading cause of morbidity and mortality in the U.S. It was the seventh leading cause of death accounting for more than 250,000 deaths in 2015. More than 30.3 million individuals or 9.4 percent of the population are estimated to have diabetes in 2015. Of those, 1.25 million had type 1 diabetes. Of the 30.3 million adults, 7.2 million were undiagnosed. While 1.5 million Americans are diagnosed with diabetes every year, this number continues to rise. Diabetes imposes a huge financial burden on the health system in the U.S. It also increases indirect costs related to employment and diminishes productivity. The total costs of diabetes in the U.S. were $245 billion in 2012. It accounted for 14 percent of the U.S. health care expenditures, one-half of which are related to complications including: myocardial infarction, stroke, end-stage renal disease, retinopathy and foot ulcers. The objective of this article is to discuss the rationale of screening and prevention of type 2 diabetes.

Rationale
Many of the criteria necessary for a screening test are appropriate for diabetes including: (1) the disorder should be an important public health problem; (2) an early asymptomatic stage should exist; (3) an acceptable screening test should be available; and, (4) an acceptable treatment during the asymptomatic stage should improve long term outcomes. The high incidence and prevalence of diabetes and its significant burden on the health of the general population shows that it meets the first criteria as a significant health problem. The evidence is less clear whether screening can decrease the mortality of diabetes. The long-term complications of diabetes would be expected to require more than a decade to develop; thus, long-term trials would be necessary to demonstrate improvements in morbidity or mortality. Early identification of diabetes would allow the use of interventions to prevent its complications such as cardiovascular disease through the use of statins at lower lipid thresholds and the use of angiotensin-converting-enzyme inhibitors (ACE inhibitors) or angiotensin receptor blockers (ARB) for lower targets for blood pressure control than for non-diabetic patients. Patients might be more motivated to adopt healthier lifestyles if they know that they also suffer from glucose intolerance.

Recommendations for Screening
Two possible approaches for screening are to screen the entire population or to screen groups identified to be at high risk. Cost effectiveness and the reduction of mortality with such interventions will guide decisions regarding the optimal approach. There are different opinions among scientists, clinicians and policy makers regarding screening for type 2 diabetes. The strength of the evidence, social,
financial and many other factors explain such variation in opinions. The following is a summary of the recommendations of some expert groups regarding screening for diabetes.

U.S. Preventive Services Task Force
The US Preventive Services Task Force (USPSTF) recommended screening for abnormal glucose as part of cardiovascular risk assessment in overweight and obese adults aged 40 to 70 years. They urged clinicians to refer patients with abnormal blood glucose to intensive behavioral counseling interventions to promote healthful diet and physical activity with repeat screening every three years. These recommendations are based on limited evidence.

American Diabetes Association
The American Diabetes Association (ADA) recommends screening for the general population and any population at risk (Table 1). 10

<table>
<thead>
<tr>
<th>Table 1. Criteria for screening for diabetes or pre-diabetes in asymptomatic adults. 10</th>
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<tbody>
<tr>
<td><strong>At age 45 years for all patients</strong></td>
</tr>
<tr>
<td><strong>High risk adults:</strong> overweight or obese patients with body mass index (BMI) 25 or greater (23 or greater if Asian American) who have one or more of the following risk factors regardless of age:</td>
</tr>
<tr>
<td>• Hypertension (140/90 or greater or therapy for hypertension)</td>
</tr>
<tr>
<td>• Physical inactivity</td>
</tr>
<tr>
<td>• History of cardiovascular disease</td>
</tr>
<tr>
<td>• HDL less than 35 and/or triglyceride level greater than 250 mg/dl</td>
</tr>
<tr>
<td>• A1C 5.7% or greater, IFG, IGT in previous testing</td>
</tr>
<tr>
<td>• Women diagnosed with gestational diabetes mellitus, polycystic ovarian syndrome</td>
</tr>
<tr>
<td>• First degree relative diagnosed with diabetes</td>
</tr>
<tr>
<td>• High risk ethnicity (African Americans, Pacific islanders, Latinos, Native Americans)</td>
</tr>
</tbody>
</table>

Abbreviations: HDL: high density lipoprotein, A1C: hemoglobin A1C, IFG: impaired fasting glucose, IGT: impaired glucose tolerance, OGTT: oral glucose tolerance test

Centers for Disease Control and Prevention
The Centers for Disease Control and Prevention (CDC) uses a risk tool assessment. Patients with high risk for diabetes should contact their primary care provider (physician) for further testing. They suggested testing by either fasting plasma glucose (FPG), oral glucose tolerance testing (OGTT), or Hemoglobin (A1C) testing for individuals 45 years or older, those who are overweight, first-degree relative of an individual with diabetes, those from a high-risk ethnic group, those with a history of gestational diabetes and those with a sedentary lifestyle. 12,11

The Canadian Task Force on Preventive Health Care:
The Canadian Task Force on Preventive Health Care (CTFPHC) recommends using a validated risk calculator to identify people at high risk. 14 Patients with high risk need to be screened with Hemoglobin A1C every 3-5 years and annually for those at very high risk. They did not recommend routine screening of low to moderate risk individuals.

Population at Risk
Almost half of patients with type 2 diabetes and prediabetes are not aware of their disease. 9 Identification of those patients is crucial in terms of early diagnosis, treatment, and prevention but screening the whole population for diabetes would be inconvenient, impractical and expensive. 9 Screening should be offered for those who are above 45 years regardless of their risk factors. Those with a BMI of 25 or more (or BMI of 23 or more if they are Asian Americans) should be screened if they have at least one of the following risk factors: first degree relative with diabetes, high risk race/ethnicity, history of gestational diabetes, polycystic ovary syndrome, history of cardiovascular disease, hypertension, low HDL or high triglycerides; and patients who have prior abnormal fasting plasma glucose (FPG), oral glucose tolerance test (OGTT) or hemoglobin A1C (A1C). 11

Screening Tests
FPG, A1C and OGTT can be used for screening. 11,15 Risk scores and urine glucose may also be used. Each one has its own advantages and disadvantages and their use may be limited by availability and cost.

FPG
The specificity of a fasting plasma glucose greater than or equal to 126 mg/dL (7.0 mmol/L) is greater than 95 percent, making it the most specific test for diagnosis of diabetes. It can be used for confirming the diagnosis of diabetes in patients with a high likelihood of diabetes. It is not a suitable test for screening as it is only 50 percent sensitive. In addition to that, the specificity and sensitivity of FPG are lower for people over the age of 65 years. 16 Therefore, interpreting a normal FPG should take into consideration the likelihood of having diabetes (from the patient’s risk factors profile). Re-testing with two hour-GTT or A1C should be considered in patients with high likelihood of having type 2 diabetes.

A1C
Recently, many physicians are using this test to screen and
Primers in Medicine

diagnose diabetes. The specificity and sensitivity of an A1C greater than or equal to 6.5 percent are 79 and 44 percent, respectively.17 This test is more convenient because it does not require fasting and it has less day-to-day perturbations during illness and stress. The presence of diabetic retinopathy correlated better with A1C greater than or equal to 6.5 percent than with fasting plasma glucose or OGTT criteria and might even support an argument that A1C is a better reference standard.18,19 On the other hand, there are many disadvantages of AIC. Many factors unrelated to hyperglycemia can affect its level, including age, ethnicity, pregnancy, anemia and hemoglobinopathies, hemodialysis, recent blood loss or transfusion or erythropoietin therapy.11

OGTT
The two-hour GTT (glucose tolerance test) with 75 gm glucose intake is the test with the highest sensitivity. It is used to diagnose more patients with type 2 diabetes than the AIC and or FPG.11 The efficacy of interventions for primary prevention of type 2 diabetes has primarily been demonstrated among individuals with impaired glucose tolerance (IGT), not for individuals with isolated impaired fasting glucose (IFG) or for those with impaired glucose tolerance defined by A1C.11,20 This test is inconvenient and time consuming and has been limited for screening in pregnant women.11

Risk Scores
Risk factor assessment has been investigated as a strategy for screening, but most have not been validated in diverse populations and are not in widespread use. These are usually simple questionnaires about important diabetes risk factors (e.g., age, weight, family history of diabetes, personal history of hypertension, physical activity) to predict the presence of impaired glucose tolerance in patients with high pretest probability. Many expert groups, medical societies and organizations use risk scores to guide screening for patients with high risk. The ADA risk test is recommended to guide providers on whether performing a diagnostic test is appropriate.11

Urine Glucose Measurement of urine glucose is not recommended for screening due to its insensitivity in detecting type 2 diabetes.11 Additionally, glucosuria can result from defects in renal tubular function (e.g., proximal renal tubular acidosis or in familial renal glycosuria.22 However, if glucosuria is detected in urine analysis performed for other indications and there is high suspicion for diabetes, then blood testing should be completed (A1C, fasting plasma glucose, or two-hour OGTT).

Confirming Diagnosis
Regardless of the test used, any abnormal test result should be repeated in a subsequent day without delay using the same test on new blood samples. A second abnormal test result will confirm the diagnosis. If two different test results were abnormal, the diagnosis is confirmed. If two different test results showed discordance than the test result that is above the diagnostic cut point should be repeated. Normal repeated tests warrant a short-term follow up in three to six months.31

Interval of Screening
Evidence is less clear regarding the interval of screening; however, many medical societies recommend testing patients in intervals of three to five years. The ADA recommends that in patients with normal testing a minimum of three-year intervals is reasonable. Patients with borderline results (FPG of 100-125 mg/dl or A1C of 5.7-6.4 percent) should be followed up every one to two years.11

Prevention of Type 2 Diabetes in Patients with Impaired Glucose Tolerance
The ultimate goal of screening for diabetes is to identify patients who are at risk for diabetes and to implement interventions that can delay micro-vascular and macro-vascular complications. Several large-scale studies have shown that type 2 diabetes can be prevented through exercise, diet, pharmacological interventions or bariatric surgery.15 Most medical societies recommend combinations of those interventions to reduce the risk for diabetes.23-26

Lifestyle Interventions
Lifestyle interventions comprise combinations of physical activity, dietary therapy and behavioral modifications. Evidence shows that lifestyle interventions are the most effective way to prevent type 2 diabetes in patients at risk.23,15 Type 2 diabetes was reduced by 58 percent over three years in the Diabetes Prevention Program (DPP), by 43 percent at 20 years in the Da Qing study, by 43 percent at 7 years in the Finnish Diabetes Prevention Study (DPS), and by 34 percent at 10 years in the U.S. Diabetes Prevention Program Outcomes Study (DPPOS).20,27-29

The ADA used the Diabetes Prevention Program (DPP) as guidance to recommend physical activity and dietary changes.15 The DPP set weight loss and physical activity
goals for all participants in an individual-based model rather than a group-based one. This flexibility allows people to choose methods that fit them in order to reach those goals (the type of exercise and how many times per week for example). The DPP intervention was administered as a structured 16-session core curriculum completed within 24 weeks followed by a more flexible maintenance program of individual sessions, group classes, motivational campaigns, and restart opportunities. The core curriculum included sections on lowering calories, increasing physical activity, self-monitoring, maintaining healthy lifestyle behaviors, and psychological, social, and motivational challenges.

Physical Activity and Weight Loss
Maintaining a minimum of 7 percent weight loss and 150 minutes of physical activity per week similar in intensity to brisk walking was consistently beneficial in many studies for preventing diabetes. More physical activity is associated with more benefit and less risk of developing diabetes.

The goal for physical activity was selected to approximate at least 700 kcal per week of expenditure from physical activity. This goal was described as at least 150 minutes of moderate intensity, aerobic physical activity per week similar to brisk walking. Participants were encouraged to distribute their activity throughout the week with a minimum frequency of three times per week with at least 10 minutes per session. A maximum of 75 min of strength training of the 150 minutes/week of physical activity was recommended.

In addition to aerobic activity, a diabetes prevention program may include resistance training. Breaking up prolonged sedentary time is encouraged and is associated with moderately lower postprandial glucose levels. The preventative effects of exercise apply to the prevention of gestational diabetes (GDM).

In the DPP, regarding weight loss, the recommended pace of weight loss was 1-2 lbs. per week to achieve 7 percent weight loss during the first six months of the intervention. Caloric and fat intake goals were used to achieve weight loss. Calorie goals were calculated by estimating the daily calories needed to maintain the participant’s initial weight and subtracting 500-1,000 calories per day (depending on initial body weight). Dietary fat intake goals were given in grams of fat per day, based on 25 percent of calories from fat. Fat and calorie goals were used as a means to achieve the weight loss goal rather than as a goal in and of itself.

Diet and Nutrition
Multiple studies have shown that correct decisions regarding diet are associated with lower risk for developing type 2 diabetes. Rather than recommending a specific type of diet, it is wise to outline a general theme of nutrients. This approach allows greater flexibility and personal preferences, which may improve long-term adherence. Data suggest that whole grains, nuts, berries, yogurt, coffee and tea are associated with reduced risk of diabetes. Conversely, red meats and sugar-sweetened beverages are associated with an increased risk.

In the Dietary Approaches to Stop Hypertension (DASH), or a Mediterranean-style diet, fruits, vegetables, whole grains, beans, nuts, and seeds, olive oil and low to moderate wine consumption are permitted. These diets have been studied extensively and in one trial appeared to reduce the incidence of diabetes independent of weight loss. The trial included 7,447 men and women and examined the effects of two different Mediterranean diets. One included supplementation with extra virgin olive oil and the other mixed nuts compared with a low fat control diet. Cardiovascular outcomes in men and women at high risk for cardiovascular disease (CVD) were compared at a median time of four years. The incidence of diabetes was significantly reduced in the groups assigned to the Mediterranean diets. Although the results support the use of the Mediterranean diet, there were concerns about the specific methods used in this trial. It is not clear which components of the diet were protective or if the benefit resulted from multiple factors. Recent evidence suggests that the subtype of the fat might be an important factor in the development of type 2 diabetes. Randomized trials of Mediterranean diets with diabetes as a primary endpoint are necessary before recommending them for prevention of diabetes.

High carbohydrate diets have been associated with type 2 diabetes and coronary heart disease. Food is classified according to its impact on blood glucose after meals using glycemic index (GI) and glycemic load (GL). Many studies and meta-analyses demonstrated the association between high GI and risk of type 2 diabetes, coronary heart disease (CHD) and breast cancer. There was a significant dose-response between dietary GL and the risk of type 2 diabetes especially when the GL was greater than
About **HALF** of the women who quit smoking during pregnancy relapse within 6 months of delivery.

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95 g/2000 kcal. The website www.glycemicindex.com can be used to provide information for counseling patients.

Low (60 to 130 grams of carbohydrates) and very-low (0 to less than 60 grams) carbohydrate diets have many benefits in terms of promoting weight loss and preventing type 2 diabetes, cardiovascular mortality and mortality. They are safe and successful in achieving short-term weight loss (at six months) compared to low fat diet; however, long term success has not be demonstrated at one year. In a prospective cohort of more than 85,000 women and 44,000 men followed up for more than 20 years, low carbohydrate diet based on vegetable sources were associated with lower all cause and cardiovascular mortality compared to low carbohydrate diet from animal sources.

Nonnutritive sweeteners have the potential to reduce overall calorie and carbohydrate intake. The U.S. Food and Drug Administration has reviewed several types of hypo-caloric sweeteners for safety and approved them for consumption by the general public, including people with diabetes.

Sugar-sweetened beverages have been associated with an increased risk of diabetes. In the US, two prospective studies examined more than 150,000 women. Those who consumed one or more drinks per day were at increased risk of weight gain and type 2 diabetes. A meta-analysis provided evidence that sugar-sweetened beverages consumption promotes weight gain in children and adults. This was reproduced in Europe in a cohort of more than 30,000 and in Asia in more than 43,000 participants.

**Sustainability and Cost Effectiveness of Lifestyle Interventions**

**Sustainability**

In a follow-up observational study, the Diabetes Prevention Program Outcomes (DPPOS), the benefit of the lifestyle intervention persisted for more than 10 years. Participants who reverted to a normal glucose tolerance at least once during the DPP had a lower risk of developing diabetes than those who consistently had values suggesting pre-diabetes. Reversion to normal glucose tolerance, even if brief, was associated with a lasting reduction in the risk of developing diabetes. Another study showed continued benefit seen 15 years after in those who reverted by to normal glucose tolerance (even transiently) with either lifestyle intervention or with metformin treatment.

**Cost Effectiveness**

One cost-effectiveness study demonstrated that the lifestyle intervention used in the DPP for three years and the DPPOS for seven years were cost-effective compared with a no-intervention placebo group. DPP content in community settings has the potential to reduce overall program costs while still producing weight loss and diabetes risk reduction. In response to investigations such as these, the Centers for Medicare and Medicaid Services (CMS) proposed expanded Medicare reimbursement for DPP programs. This makes DPP the first ever preventive service model certified for expansion from the CMS Innovation Center.

**Factors Associated with the Efficacy of Lifestyle Interventions**

A recent study showed that meeting either lifestyle or weight loss goals at 6 months had the strongest association with achieving these goals at 12 and 18 months. Early engagement, regular attendance, self-monitoring, and self-weighing are of a paramount importance for satisfactory results. Alternative approaches should be implemented for those not meeting goals by 6 months to enhance long-term success.

**Technology Assistance to Deliver Lifestyle Interventions**

Technology now plays a fundamental role in patient care. Many studies demonstrated its efficacy in helping patients implement lifestyle interventions. This can take the form of digital video-based content delivery, Internet based social networks and mobile applications to guide exercise and weight loss. A recent study showed that an all-mobile approach to administering DPP content can be an effective tool over the short term in overweight and obese individuals. This has driven many medical societies to encourage their use.

**Pharmacologic Interventions**

Metformin has been shown to decrease the incidence of type 2 diabetes. The 2018 guidelines by the ADA recommend metformin therapy for prevention of diabetes in patients with pre-diabetes, especially for those with body mass index 35 kg/m2 or greater, age less than 60 years, women with prior gestational diabetes, and/or those with rising A1C despite lifestyle intervention. It is as effective as lifestyle intervention in those patients.

The current use of metformin is suboptimal despite its low
cost, excellent safety profile and effectiveness. One study published in 2017 using the National Health and Nutrition Examination Survey (NHANES) 2005-2012 demonstrated that less than 1 percent of 7,652 patients with pre-diabetes used metformin. Surprisingly, metformin use was low even among those with BMI greater than 35 kg/m², a group for whom metformin use is recommended. Metformin use did not vary by race, income, or education. Many other studies have showed similar results with a prescription rate varying from 3.7 percent-8 percent.  

Prolonged use of metformin has been shown to be cost effective over a 10-year period. Further, its use is safe. It is recommended starting a low dose to avoid some of the gastrointestinal side effects. Lactic acidosis is rarely seen and primarily occurs in patients with renal impairment or hepatic failure. Hypoglycemia is rare to nonexistent with metformin monotherapy. Reductions in hemoglobin and hematocrit levels have occurred during the first year of Metformin treatment with reports of Vitamin B12 deficiency in some patients.

**Other Medications**

In addition to metformin, thiazolidinediones, alpha-glucosidase inhibitors, orlistat, and liraglutide have demonstrated efficacy. Although these drugs delay the onset of diabetes and reduce the length of exposure of hyperglycemia, the benefit and harms of the intervention must be considered. Thiazolidinediones can cause fluid retention, weight gain, heart failure, myocardial infarction MI (rosiglitazone) and possibly bladder cancer (pioglitazone). Alpha-glucosidase inhibitors can cause gastrointestinal side effects and have poor long-term compliance. Nateglinide, an angiotensin-converting enzyme (ACE) inhibitor, angiotensin II receptor blockers (ARBs), estrogen, insulin and Vitamin D are not effective for preventative therapy and are not recommended for the prevention of type 2 diabetes in patients with impaired glucose tolerance.

**Diabetes Self-Management, Education and Support**

Diabetes self-management education and support programs may be appropriate venues for people with pre-diabetes to develop and maintain behaviors that can prevent or delay the development of diabetes. There are significant barriers to the provision of education and support to those with pre-diabetes. Although reimbursement remains a barrier, studies show that providers of diabetes self-management education and support are particularly well equipped to assist people with pre-diabetes in developing and maintaining behaviors that can prevent or delay the development of diabetes.

**Conclusion**

Type 2 diabetes places a significant burden on patients and the health care system in the U.S. Identification of populations at risk through screening for impaired glucose tolerance and diabetes allows the implementation of interventions that can prevent and treat type 2 diabetes. Research data consistently have shown that dietary therapy, increasing physical activity and managing obesity together with the use of pharmacological interventions are effective in achieving such goals. Education, partnership between health care providers and patients and the use of modern technology are beneficial in helping patients adopt and sustain a healthy lifestyle.
“Don’t put that in your mouth.” A Discussion of Button Battery Ingestions in the Pediatric Population

By Megan Sampson, DO; and Tonya Adamiak, MD

Abstract
Ingestions are a problem that plagues the pediatric population and can at times be life threatening. Signs, symptoms, and interventions for button battery ingestion will be summarized for the reader. A case presentation from practice will be given to help enhance readers experience and learning.

Introduction
The pediatric population is highly susceptible to gastrointestinal foreign body ingestions, with the peak incidence occurring between 6 months and 6 years of age. Small objects, such as coins, toys, and crayons, typically lodge in the anatomically narrow proximal esophagus.1 Of objects with potential for being ingested button battery ingestions have the potential to cause serious complications. Esophageal impaction of a button battery can quickly lead to significant mucosal ulceration, perforation, and even gastrointestinal hemorrhage and death from aortoesophageal fistula. It is important to be able to recognize potential presenting symptoms of a gastrointestinal foreign body, be able to differentiate a button battery from a coin on X-ray, and understand that esophageal button batteries are an emergency. Herein a discussion of a patient presentation and provider management recommendations.

Case Report
Presentation
A 13-month-old boy presented from outside hospital with history of vomiting, fever, and decreased activity for one week. He was seen by his primary care provider four days prior to presentation and diagnosed with clinical pneumonia. He was treated with a course of azithromycin and steroids, but had ongoing symptoms, and additionally started drooling and refusing to eat solids.

Physical Exam
The patient was afebrile with stable vital signs. He was awake and alert, fussy but consolable. No signs of trauma. Lungs with coarse breath sounds right lower lobe, but good air exchange and no respiratory distress. Occasional cough. Remaining physical exam was unremarkable.

Evaluation
Chest X-ray was completed (Figure 1 and 2). There was a round radiopaque foreign body in the proximal esophagus. PA view showed double rim or halo sign and lateral view showed step off sign.

Plan
The patient was taken to the operating room for upper endoscopy. A button battery was identified in the proximal esophagus and was removed using rat-toothed forceps without complication. Thermal injury was seen where the button battery had been lodged (Figure 3).

Hospital Course
The patient was admitted to the hospital following upper endoscopy. Pediatric ENT and gastroenterology were involved in the patient’s care. The patient was started on a proton pump inhibitor to help protect the esophagus from further injury related to any refluxed gastric acid. CT of the chest with contrast on hospital day 1 showed an irregular appearance and mild dilation of the upper thoracic esophagus near the thoracic inlet, correlating to the site of battery impaction on prior X-ray. The patient then had an esophagram which showed narrowing and irregularities consistent with inflammation in the proximal esophagus at the site where the button battery had been.
lodged. Esophagram did not show any evidence of contrast extravasation or concern for perforation. The patient was initially NPO and on hospital day 2 he was started on a liquid diet. MRI of the neck and soft tissue on hospital day 3 showed mild enhancement of soft tissues around the esophagus in the lower cervical and upper thoracic region, presumed secondary to inflammation from the battery. Clinically he did well in the hospital and was discharged home on hospital day 9. He was discharged home on a liquid diet and continued on the proton pump inhibitor.

Follow Up
The patient was on a liquid diet for several weeks, and then was gradually advanced to a regular diet, which he tolerated well. He was followed closely in pediatric GI clinic, given potential concern for esophageal stricture.
that would require future endoscopic dilations. Esophagrams were repeated at one month post-ingestion and eight months post-ingestion. The one-month post-ingestion esophagram showed that the irregularity in the proximal esophagus had improved. There was no significant stricture identified. The eight-month post-ingestion esophagram was normal. There were no abnormalities or strictures identified. The patient did not require any endoscopic dilations.

**Discussion**

Button batteries are small, round, flat batteries frequently found in watches, hearing aids, small electronic devices and toys. Lithium has become the preferred button battery cell type because it has longer shelf life and battery life, lighter weight and thinner form, higher energy density, and resistance to cold. Button batteries cause mucosal injury by the generation of hydroxide radicals resulting in caustic injury from high pH.

Incidence: Button battery ingestions have a peak incidence between 1-2 years of age. According to the National Poison Data System, 3,383 button battery ingestions occurred in 2016. Greater than 3 percent of these were classified as moderate, severe, or death. The total number of button battery ingestions throughout the most recent years has not shown any clear incidence trend; however, the percent resulting in severe complications or death has increased, presumably due to the increased diameter size of the button battery and the change to lithium cells. The larger diameter results in increased likelihood of esophageal impaction and the lithium composition results in increased voltage delivery. In one series that described esophageal button battery ingestion cases that resulted in a significant or fatal outcome, approximately 94 percent of button batteries were greater than 20 mm in diameter (20 mm being between the size of a penny and nickel). Battery diameter of 20-25 mm was the most important predictor of a clinically significant outcome, followed by age younger than 4 years old, and ingestion of more than one battery.

**Clinical Signs and Symptoms**

Consider the possibility of a battery ingestion in every patient with acute airway obstruction; wheezing or other noisy breathing; drooling; vomiting; chest pain or discomfort; abdominal pain; difficulty swallowing; decreased appetite or refusal to eat; or coughing, choking or gagging with eating or drinking. If a battery has already caused an esophageal perforation, symptoms can include fever, hemorrhagic shock, tension pneumothorax, and subcutaneous emphysema. One foreign body series reported that about half of pediatric ingestions are not witnessed by a parent or caregiver, stressing the importance of being able to recognize these symptoms as a potential presenting symptoms of a foreign body ingestion. It should also be noted that the patient may be asymptomatic, and brought in only after the ingestion was witnessed by a caretaker.

**Imaging**

Radiographs should be obtained to confirm the location, size, shape, and number of ingested foreign bodies. It is critical to be able to differentiate between a button battery and coin on X-ray, as the urgency of management is different and the potential complications of an esophageal button battery can be very serious or even fatal. A button battery will have a “halo-like appearance or “double ring” on PA view. Lateral X-ray can show a “step off” appearance of a button battery.

**Treatment/Intervention**

A battery lodged in the esophagus is an emergency. Potential complications of esophageal button battery ingestions include aorto-esophageal fistula, tracheoesophageal fistula, esophageal stricture, esophageal perforation, and vocal cord paralysis from recurrent laryngeal nerve injury, among others. Severe complications can occur when a battery has been lodged in the esophagus for as little as two hours, and complications can be delayed in presentation, with strictures maybe not becoming apparent for weeks to months. Aorta-esophageal fistula secondary to esophageal button battery impaction is one of the most feared complications, with fatality rate nearing 100 percent. For this reason, if there is active bleeding in a patient with an esophageal button battery, or the patient is unstable, it is best to have cardiovascular surgery and/or pediatric surgery present at the time of endoscopic removal.

Following removal of an esophageal button battery, if there is any evidence of esophageal injury, the patient should be admitted to the hospital for observation. The patient should initially be NPO to monitor for signs of injury. Esophagram should be done to exclude perforation before advancing diet. If the endoscopy had shown signs of esophageal injury, it is recommended to do CT angiography or MRI chest to evaluate for aortic injury and determine the proximity of injury to the aorta. Following removal of esophageal button battery, patients need to be closely
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Figure 6. National Capital Poison Center Treatment and Triage Algorithm

**Suspect a battery ingestion in these situations**

- “Coin” ingested. Carefully check AP x-ray for battery’s double- or halo-effect and lateral view for step off. Use magnification.

**Battery ingestion known or suspected**

- Give honey 10 mL every 10 mins if child ≥ 12 months, lithium coin cell possibly ingested, and ingestion within prior 12 hours. (See text guideline below for detail, #2.) Do not delay going to ER to give honey. Otherwise, NPO until esophageal position ruled out.¹
- Take up to 5 minutes to determine imprint code (or diameter) of companion or replacement battery.
- Consult National Battery Ingestion Hotline at 800-498-8666 for assistance with battery identification and treatment.

**TIPS, PITFALLS & CAVEATS**

- 3 “N”s: Negative – Narrow – Necrotic. The negative battery pole, identified as the narrowest side on lateral x-ray, causes the most severe, necrotic injury. The negative battery pole is the side opposite the “+” and without the imprint.
- 20 mm lithium coin cell is most frequently involved in esophageal injuries. Smaller cells lodge less frequently but also cause serious injury or death.
- Definitive determination of the battery diameter prior to passage is unlikely at least 40% of ingestions.
- Assume hearing aid batteries are <12 mm.
- Manage ingestion of a hearing aid containing a battery as an ingestion of a small (≤12 mm) battery.
- Do not induce vomiting or give cathartics. Both are ineffective.
- Assays of blood or urine for mercury or other battery ingredients are unnecessary.

**Symptomatic patient, no ingestion history. Consider battery ingestion if:**

- Airway obstruction or wheezing
- Drooling
- Vomiting
- Chest discomfort
- Difficulty swallowing, decreased appetite, refusal to eat
- Coughing, choking or gagging with eating or drinking

**Immediate removal batteries lodged in the esophagus.**

- Consider sucralfate suspension or honey if ≤12 h post ingestion (see text guideline below for detail, #11).
- Do not delay removal if patient has eaten.
- Prefer endoscopic removal (instead of retrieval by balloon catheter or magnet affixed to tube) for direct visualization of tissue injury. Inspect mucosa for extent, depth and location of damage. Note position of battery and direction negative pole faces.
- If no endoscopic evidence of perforation, irrigate injured areas of esophagus with 50-150 mL 0.25% sterile acetic acid to neutralize residual alkali (see text guideline below, #13a).

**After removal, if mucosal injury was present, observe for and anticipate delayed complications:**

- tracheoesophageal fistula, esophageal perforation, mediastinitis, vocal cord paralysis, tracheal stenosis or tracheomalacia, aspiration pneumonia, empyema, lung abscesses, pneumothorax, spondylodiscitis, or excavation from perforation into a large vessel.

**NOTES:**

1 NPO except for honey or sucralfate suspension.

2 X-ray abdomen, esophagus and neck. Batteries above the range of the x-ray have been missed. If battery in esophagus, obtain AP and lateral to determine orientation of negative pole. If ingestion suspected and no battery visualized on x-rays, check ears and nose.

3 If battery diameter is unknown, estimate it from the x-ray, factoring out magnification (which overestimates diameter).
followed. And it should be noted that serious complications have been reported as long as 18 days after battery removal. Patients with a history of esophageal button battery ingestion need to be monitored for any signs of GI bleeding, as many times this is the initial sign of a fatal hemorrhage from aorto-esophageal fistula, and if this “sentinel bleed” is recognized in a stable patient, a time window exists in which surgical intervention can be accomplished and the patient’s life could possibly be saved.2

Prevention
Caregivers should check and secure the battery compartment of all household products, store batteries out of a child’s reach and sight, and never leave loose batteries sitting out. Even old “dead” batteries are dangerous, as they still have enough residual charge to cause problems. It is also important to make sure hearing aids have a child resistant battery compartment. The National Battery Ingestion Hotline provides 24/7 telephone treatment guidance for the public and healthcare professionals (202-625-3333). Information on safety tips for button batteries can be found at www.poison.org/battery/tips.asp.

Conclusion
It is important for providers to have a high index of suspicion if a pediatric patient presents with respiratory symptoms, chest pain, vomiting, drooling, or feeding refusal, as these can be presenting symptoms of a gastrointestinal foreign body. X-ray should be done to determine if a foreign body is present and the X-ray should be closely examined to differentiate a coin from a button battery. Button batteries that are greater than 20 mm in diameter are of significant concern, due to the larger size increasing the risk of esophageal impaction. A button battery lodged in the esophagus is an emergency because serious complications can occur in as little as two hours. Prevention is the first step in decreasing the risk of serious complications related to swallowed foreign bodies.

REFERENCES

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Introduction

Management of arrhythmias in the past has been limited to medications. New procedures (surgical and percutaneous) are now options in the management of arrhythmias and have reduced the role of antiarrhythmic drugs (AAD) today, to a more complimentary role rather than as primary management. The understanding of the mechanism and underlying proarrhythmic properties is imperative in understanding the benefits and limitations of each commonly used AAD. The commonly used AAD will be reviewed in a case-based format, as it relates to the primary care practitioners during outpatient and inpatient management. Their major associated side effects and drug interactions of each class of AAD will be a main focus.

First Case Presentation

A 35-year-old male who is fairly active, presents to you for intermittent palpitations that occur sporadically, associated with extreme shortness of breath, each episode lasting 20-30 minutes, and complains that his heart is “beating out of his chest”. This occurs once or twice per week, and the last episode lasted over 1 hour which prompted him go to the emergency department to be evaluated. He was noted to have atrial fibrillation (AF) at rates around 160-170 beats per minute (bpm). The patient’s rhythm converted back to sinus rhythm spontaneously, and he is in today for follow up as to how to best further manage his AF. He does not take any medications. His vital sign’s today are as follows: blood pressure 125/80 mmHg, pulse 85 bpm, respiratory rate of 23 per minute, and he appears in good health. Physical exam was unremarkable. Electrocardiogram shows normal sinus rhythm without evidence of pre-excitation and without evidence of left ventricular hypertrophy (LVH). Echocardiography (ECHO) did not show any significant valvular abnormalities and preserved left ventricular (LV) function and normal left atrial size. What is the best course of management at this time?

This is a young patient that has paroxysmal atrial fibrillation (PAF) that is very symptomatic. PAF is defined as each AF episode that lasts less than 7 days. Table 1 describes the different classification of AF. He continues to be active and does not have evidence of structural heart
disease (as defined by LVH greater than or equal to 1.5 cm, reduced LV function, significant valvular abnormalities or history of coronary artery disease [CAD]). He is an ideal candidate for a class IC AAD such as flecainide or propafenone, which are the two clinically relevant class IC agents in use today. In the 1980s, it was recognized that AAD, which initially were designed to prevent arrhythmias can themselves be pro-arrhythmic as evidenced by the Cardiac Arrhythmia Suppression (CAST) trial, and thus careful selection of an AAD is important. Class IC AAD are approved mainly for atrial arrhythmias; (i.e., AF, atrial tachycardia, atrial flutter, supraventricular tachycardia [SVT]), however, can be considered in patients with ventricular tachycardia (VT) without structural heart disease. Flecainide is not commonly used in the setting of refractory unstable VT as these patients in the clinical setting likely have structural heart disease (i.e., prior myocardial infarction, reduced cardiac function, etc). Patients presenting with VT without structural heart disease (i.e., outflow tract VT, fascicular VT) are most often younger patients with presentation that are generally not life-threatening arrhythmias and are very responsive to medical therapy such as AAD, and catheter ablation. Thus, in a setting of a patient who is in refractory VT and hypotensive, flecainide is rarely used. However, patients with a history of catecholaminergic VT (CPVT), flecainide can be considered. The primary abnormality in these patients are abnormal calcium handling within the myocardial cells and often presents with classical bi-directional VT (which is similar to digoxin toxicity), especially during exercise. Flecainide has a role in CPVT, as it functions to stabilize the calcium membrane channels.

Class IC agents have a high affinity for activated sodium channel but slow binding kinetics. As such, they have the biggest effect on phase 0 of the ventricular action potential, thus prolonging the QRS duration. They have stronger effects at faster heart rates, and thus the term “use dependence.” This is generally a positive attribute in that the effect of the drug is more prominent at faster heart rates, thus increasing the chance of converting an atrial arrhythmia to sinus rhythm. They are the first line medication for patients with structurally normal hearts and are contraindicated in patients with structural heart disease. There should be close monitoring of the QRS duration while on a class IC AAD as an increase of 25 percent or greater in QRS duration from baseline should warrant discontinuing the drug or reducing the dose. Propafenone has small beta blocker activity due in part to its metabolite, 5-hydroxy propafenone. However, both class IC agents ideally should be started in combination with a beta blocker. Class IC agents are particularly effective in patients who have infrequent, but very symptomatic bouts of PAF. An oral load of propafenone (600 mg if patient weight is 70 kg or greater, 450 mg if patient weight is less than 70) or flecainide (300 mg if patient weight is 70 kg or greater, 200 mg if patient weight is less than 70) has been shown to reduce emergency department visits and inpatient admissions. This method is known as “pill in the pocket” approach; however, care should be exercised when prescribing this loading dose, and ideally should be deferred to a cardiologist. The most common side effects of flecainide are paresthesia, chest pain, and diplopia, whereas propafenone have similar side effects as flecainide, but with higher incidence of central nervous system (CNS) side effects. If the patient continues to have episodes of PAF despite being on at least one AAD, either class I or III, or intolerance to AAD, the patient should be referred for catheter ablation.

**Second Case Presentation**

A 60-year-old patient presents to his primary care with complaints of palpitations, shortness of breath and symptomatology consistent with his history of AF. He has a past medical history of persistent AF, CAD with stents placed in the past, most recent ECHO showing normal LV thickness, mildly reduced LV function (ejection fraction 45 percent), and a left atrial size that is moderately enlarged. The most recent Holter monitor revealed that his rates have been relatively well controlled, with average rates around 100 bpm, but the patient is still symptomatic. He states that he feels like he has less energy and gets short of breath easily. However, when he is at his baseline and not in AF, he is relatively active, can walk 18 holes of golf without symptoms of chest pain or shortness of breath.

### Table 1. Classification of atrial fibrillation (AF)

<table>
<thead>
<tr>
<th>Classification</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paroxysmal AF</td>
<td>AF episode(s) that persist less than seven days</td>
</tr>
<tr>
<td>Persistent AF</td>
<td>AF episode(s) that persist for seven or greater days</td>
</tr>
<tr>
<td>Long-standing persistent AF</td>
<td>Persistent AF that persists for longer than a year</td>
</tr>
<tr>
<td>Permanent AF</td>
<td>A term used when joint decision by patient and physician is made to stop further attempts to restore and/or maintain sinus rhythm</td>
</tr>
<tr>
<td>Non-valvular AF</td>
<td>AF in the absence of rheumatic mitral stenosis, mechanical or bioprosthetic heart valve, or mitral valve repair</td>
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breath. His medications include metoprolol succinate 100 mg daily, aspirin 81 mg daily, clopidogrel 75 mg daily and hydrochlorothiazide 25 mg daily. He has normal renal function and states compliance with his medications. His electrocardiogram (EKG) today reveals AF at a rate of 95 bpm, with a QT corrected (QTc) of 440 msec (prolonged QTc for males is 450 msec or greater, for females 470 msec or greater). Vital signs are stable. He inquires what additional options are available for him at this time.

This patient differs from the first case presentation in that his AF is well controlled but continues to be symptomatic. This is not an uncommon presentation in clinical practice. While a patient is in AF, they lose the atrial contraction, which could contribute up to 20-30 percent of cardiac output. This patient has been labeled as persistent AF, which is defined as AF episodes lasting greater than seven days, whereas PAF is defined as lasting less than seven days (table 1). There is a subset of AF, termed longstanding persistent AF, which is defined by AF that lasts longer than one year. Permanent AF is defined as joint decision by the patient and physician to forego attempts at restoring sinus rhythm.

The ideal agent for this patient would be a class III agent such as sotalol, dronedarone or dofetilide. Their predominant effect on the ventricular action potential is by prolonging phase 3 of the action potential, thus prolonging the QT interval. Similar to our first case presentation, referral for catheter ablation would be appropriate if the patient continues to be symptomatic despite being on one AAD or intolerance. In addition to starting AAD, cardioversion should be considered. The class I agents that were discussed in the first case are not an option for this patient given his history of CAD and reduced cardiac function (structural heart disease).

Class III agents prolong the QT interval from baseline and increase the chance of early afterdepolarizations (EAD), and thus Torsades de Pointes (TdP), a life threatening polymorphic VT. It is imperative to monitor the QT interval while on these medications. If the QT corrected (QTc) is greater than 500 msec (or if greater than 550 with bundle branch block), discontinuation or reduction of the dose should be considered. Class III AAD has higher affinity for inactivated sodium channels, and a stronger effect during slower heart rates, thus the term “reverse use dependence.” This translates higher increase in QT prolongation during bradycardia. This is generally a negative attribute to this class of medication. Care should be undertaken when starting class III agents as bradycardia will prolong QT intervals and increase the risk of TdP. Sotalol ideally should be started in an inpatient setting to monitor QT intervals during initiation.

Sotalol use is approved for maintenance of sinus rhythm in patients with AF or atrial flutter and VT. It primarily works by two major mechanisms. It blocks the potassium channel regulating phase III of the action potential, providing class III AAD properties. It also has beta receptor blocking properties as seen with class II agents. It is usually well tolerated but similar to other AAD, it has pro-arrhythmic properties (risk of TdP) which limit its use, such as patients with baseline prolonged QT intervals, or in congenital long QT syndromes. Sotalol should not be started if the patient has a history of heart failure or LVH. This patient has CAD and mildly reduced LV function, but has not had any symptoms of heart failure at this time, thus sotalol is still permissible. Sotalol’s main side effects are relatively mild and include nonspecific nausea, gastrointestinal (GI) symptoms and fatigue.

Dronaderone is an analogue of amiodarone without the iodine component. Thus, dronedarone lacks the extra-cardiac iodine related side effects such as thyroid dysfunction but is considered inferior to amiodarone. It is only approved for AF, not for ventricular arrhythmias. It is contraindicated in patients with permanent AF and decompensated heart failure due to increased risk of death, stroke and heart failure in this subset of patients.

Dofetilide’s indication is similar to sotalol and can be used for atrial arrhythmias such as AF or atrial flutter. It can be considered in a patient with heart failure and structural heart disease as long as there is not any significant LVH (greater than 1.5cm). Dofetilide can be considered for use in VT as off label use, especially in the setting of refractory VT despite amiodarone therapy or to decrease incidence of ICD shocks in patient with a defibrillator. It is a highly regulated medication, and can only be started by certified prescribers in a hospital setting to reduce the incidence of TdP. Amiodarone is also another class III AAD, and will be discussed on the third case presentation.

Third Case Presentation

A 70-year-old female, presents with recurrent implantable cardioverter defibrillator (ICD) shocks. Upon interrogation of the device, sustained VT was revealed, and thus the ICD shocks were appropriate. She has a past medical history of diabetes mellitus, chronic kidney disease (CKD), myocardial
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infarction, CAD with multiple percutaneous coronary interventions, congestive heart failure, with a known LV ejection fraction of 20%. During these episodes, she would feel very lightheaded, and near syncope. She has known chronic total occlusion of her right coronary artery, and diffuse left sided coronary artery stenosis not amendable to percutaneous intervention. Her baseline function is very limited, and she lives in a nursing home. She is dependent on her activities of daily living such as toileting, feeding, and is mostly bedbound. Her home medications include amiodarone 400 mg daily, metoprolol succinate 150 mg daily, aspirin 81 mg daily and insulin. What additional option is available for her?

This patient is in end stage heart failure, in addition to likely having ischemic VT that cannot be revascularized due to her frailty and complex coronary anatomy not amendable to intervention. She is already on amiodarone, and is still having breakthrough VT. She is an ideal candidate for amiodarone therapy, as she is elderly, and unlikely to suffer the long-term side effects of amiodarone, but unfortunately has not been successful thus far due to recurrent VT. Sotalol would not be a good option for her even though it is approved for treatment of VT because of her heart failure history and renal dysfunction (as sotalol is mainly renally excreted). Class IC agents would not be an option given her history of structural heart disease (CAD, reduced cardiac function). Dronedarone is not an option as it is only approved for atrial arrhythmias. The next best option would be adding a class IB agent in addition to amiodarone therapy.

Class IB agents include lidocaine and mexiletine. Lidocaine is only available in the intravenous route whereas mexiletine is the oral analogue of lidocaine. They have rapid kinetics and have minimal effect on the ventricular action potential, and thus minimal effect on the QRS morphology on EKG. They are approved for ventricular arrhythmias only, and are considered inferior to amiodarone for management of VT. However, they can be used in addition to amiodarone in the setting of refractory VT. Lidocaine can be used for the treatment of TdP as it does not prolong the QT interval. Lidocaine and mexiletine are both predominantly metabolized by the liver (greater than 90 percent). Patients with decompensated heart failure (liver congestion leading to liver failure), and history of liver dysfunction should avoid class IB agents as it can predispose to toxicity. The most common side effects of lidocaine are mostly neurologic including seizures, tremors and hallucinations. Mexiletine’s main side effect is mainly GI symptoms (nausea, stomach cramps, diarrhea, constipation, etc) while neurological dysfunction are less common.

Dofetilide could be considered an option for this patient in place of amiodarone as an off-label use to decrease incidence of VT. Dofetilide can be safely used in patients with heart failure similar to amiodarone. In this patient however, starting a second agent (lidocaine) with a different mechanism of action would probably be the better option as lidocaine is approved for VT.

Amiodarone is an effective anti-arrhythmic for various supraventricular and ventricular arrhythmias. It has been approved for ventricular arrhythmias but is also very useful in supra-ventricular arrhythmias. It is relatively safe in patients with structural heart disease. It can be used for AF rate control in heart failure, maintenance of sinus rhythm after cardioversion, prevention of post-operative AF or atrial flutter after cardiac surgery, prevention of SVT including atrioventricular nodal tachycardia (AVNT), atrioventricular nodal reentrant tachycardia (AVNRT), focal atrial tachycardia, and stable to life threatening VT or ventricular fibrillation (VF). It has multiple complex electrophysiological effects. It inhibits the phase III potassium efflux and is thus classified as a class III agent. However, it also shares properties with class I agents by inhibiting the sodium channel, class II agents by antagonizing beta adrenergic receptors and class IV agents by inhibiting calcium channels. Due to multiple mechanisms of action, it has anti-chronotropic effect leading to slowing of heart rate, anti-dromotropic effect via atrioventricular (AV) nodal blockade leading to increased PR interval, slow ventricular conduction leading to widening of the QRS, and slower repolarization leading to increased QT interval. It has been known to be associated with multiple adverse effects and thus it requires judicious monitoring for possible side effects. The most notable side effects include the following:

- Effects related to its electrophysiological mechanism of action like hypotension, AV blockade, QT prolongation;
- Iodine related endocrine dysfunction including both hyper or hypo-thyroidism, pulmonary toxicity, corneal deposits;
- Jepatotoxicity, nausea, vomiting, diarrhea, constipation, neurological tremor, abnormal gait, peripheral neuropathy, skin photosensitivity, blue-gray pigmentation.
Figure 1. Antiarrhythmic drug selection for patients.

1) Can consider catheter ablation in experienced center, and patient’s preference
2) Avoid if severe left ventricular hypertrophy (wall thickness > 1.5cm)
3) Should be used with AV nodal blocking agents

Table 2. Commonly used class I and III anti-arrhythmic drugs, mechanisms of action, indications and common side effects

<table>
<thead>
<tr>
<th>Class</th>
<th>Mechanism of action</th>
<th>Drug</th>
<th>Indications</th>
<th>Common side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>IA</td>
<td>Sodium channel blockade -slows conduction velocity (less than IC) and prolongs action potential duration</td>
<td>Disopyramide, Procaainamide, Quinidine</td>
<td>AF, atrial flutter, VT, VF</td>
<td>Xerostomia, constipation, urinary hesitancy, AV blockade, dizziness, fatigue</td>
</tr>
<tr>
<td>IB</td>
<td>Sodium channel blockade -no effect on conduction velocity, may shorten action potential duration</td>
<td>Lidocaine, Mexitilene</td>
<td>VF, VT</td>
<td>Headache, seizure, tremor (loss with Mexitilene), bradycardia, nausea, vomiting (more with Mexitilene)</td>
</tr>
<tr>
<td>IC</td>
<td>Sodium channel blockade -slows conduction and prolongs action potential duration</td>
<td>Flecainide, Propafenone</td>
<td>SVT, AF, atrial flutter, VT</td>
<td>Dizziness, headache, fatigue, tremor,</td>
</tr>
<tr>
<td>III</td>
<td>Potassium channel blockade</td>
<td>Amiodarone and Dronedereone (Have additional alpha- and beta-adrenergic blockade, sodium channel blockade, calcium channel blockade)</td>
<td>AF (with or without structural heart disease), atrial flutter, SVT (AVRT or AVNRT), monomorphic or polymorphic VT (stable or unstable), VF</td>
<td>Mechanism of action related: hypotension, AV blockade, QT prolongation. Iodine related (amiodarone): hyper- or hypo-thyroidism, pulmonary toxicity, corneal deposits. GI: hepatotoxicity, vomiting, diarrhea, constipation. Neurological: tremor, abnormal gait, peripheral neuropathy. Skin: blue-gray pigmentation, photosensitivity. *Dronedereone: Does not have iodine related extra-cardiac effects</td>
</tr>
<tr>
<td></td>
<td>Sotalol (Has additional beta-blocker properties)</td>
<td>SVT, AF, atrial flutter, ventricular arrhythmia such as monomorphic VT.</td>
<td>Bradycardia, fatigue, weakness, dyspnea, edema, hypotension</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ibutilide</td>
<td>AF, atrial flutter, facilitation of electrical cardioversion</td>
<td>Monomorphic or polymorphic VT such a TdP, VPC</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dofetilide</td>
<td>SVT, AF, atrial flutter</td>
<td>TdP, headache, dizziness</td>
<td></td>
</tr>
</tbody>
</table>

Atrial fibrillation (AF), ventricular tachycardia (VT), ventricular fibrillation (VF), atrio-ventricular (AV), supraventricular tachycardia (SVT), atrio-ventricular reentrant tachycardia (AVRT), atrio-ventricular nodal reentrant tachycardia (AVNRT), gastrointestinal (GI), torsades de pointes (TdP), ventricular premature complexes (VPC)
Many of amiodarone’s side effects are chronic and can occur years after initiation of the drug, thus the lowest chronic dose is advised if possible (100-200 mg daily). While amiodarone commonly increases the QT interval (i.e., QTc greater than 500 msec), it is not a drug that is well known to cause TdP.

**Antiarrhythmic Drug Selection**

The decision to start a patient on an antiarrhythmic is predicated on the risks and benefits for the individual. Younger patients who are relatively active will likely benefit more from maintaining sinus rhythm, as the atrial contraction can contribute large amount of cardiac filling during diastole. However, in elderly patients who have severe diastolic dysfunction, the contribution of atrial kick may be minimal due to noncompliant left ventricle. In addition, if the left atrial size is enlarged (suggestive of chronic remodeling), the probability of maintaining sinus rhythm will be diminished. Figure 1 is a guideline on what to start for a patient if AAD are considered. At this current time, there is no consensus recommendation regarding testing for pharmacogenomics prior to prescribing AAD, but this may change in the future.

Class IC AAD are contraindicated in patients with structural heart disease. They are considered the first line medications for patients without structural heart disease in the management of AF. For patients with known structural heart disease, the class III AAD can be used with preference to avoid amiodarone if possible and can be considered when other AAD have failed or are contraindicated. Amiodarone is one of the safest AAD in short term use, but the long-term side effects limits its use, especially in the younger population. If there is LVH, only dronedarone and amiodarone should be considered. Table 2 shows a list of the most clinically relevant class I and III AAD with mechanism of action, common indications and side effects. The class Ia agents are not clinically relevant and is rarely used today for management of arrhythmias. Ibutilide is a class III agent used for acute chemical cardioversion in an emergency room setting and should be reserved for use by specialists. Both class IB agents and ibutilide were not discussed on this review article but were included in Table 2 for completion.

**Common drug-drug interactions of antiarrhythmics**

There are many common drug-drug interactions of AAD
that one should be aware of when prescribing as shown in Table 3. Inhibitor effects on the substrate will increase drug concentration, whereas inducer effects on the substrate will decrease drug concentration. A frequently encountered combination in clinical practice is that of amiodarone and warfarin. Warfarin is metabolized by CYP (cytochrome P450) 2C9, and its drug concentration is increased with amiodarone (since amiodarone inhibits CYP 2C9). For example, if amiodarone is initiated in a patient on chronic warfarin therapy, there should be close monitoring of the international normalized ratio (INR) in the short term until the INR has been deemed stable. When prescribing class III agents, close evaluation of additional QT prolonging medications (i.e., antidepressants, certain antibiotics, etc.) the patient maybe concurrently taking should be carefully assessed and avoided if possible.

Amiodarone, dronedarone are inhibitors for the P-glycoprotein transport system, and can commonly increase digoxin levels, and thus increase incidence of digoxin toxicity. Dronedarone commonly artifactually increases serum creatinine levels, but it has no effect on renal function. The effect on serum creatinine is modest at around 10-15 percent, and is to be expected, as it blocks the tubular reabsorption of creatinine. No adjustment of dronedarone due to increased creatinine is recommended, however, it could confound the diagnosis of CKD in a patient with borderline abnormal renal function.

With the introduction of the direct oral anticoagulant (DOAC), (i.e. rivaroxaban, apixaban, edoxaban, dabigatran), drug interactions can be of concern. The factor Xa inhibitors (rivaroxaban, apixaban, and edoxaban) are a substrate of CYP 3A4, whereas dabigatran (a direct thrombin inhibitor) is a substrate of P-glycoprotein (Table 2). Drug interactions with DOAC and AAD are also confounded due to the levels of DOAC are not as easily measurable as coumadin. Care should be implemented when there are drug interactions with AAD and DOAC and should be avoided if possible. However, at this time, there are no clear consensus regarding dose adjustments while a patient is on an AAD and case by case basis should be implemented.14

Conclusions
The decision to start a patient on an AAD can be a complex one and is based on the clinical scenario. There is some generalizability regarding starting AAD in certain population as described in this manuscript, common side effects, what to monitor, and common drug interactions. This manuscript introduces the basic concepts of the most commonly used AAD used today. In the clinical setting, most AAD are co-managed with Cardiologist and primary practitioners, and general knowledge of commonly used AAD is beneficial for improved patient care.

References

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The opioid epidemic has become a national health crisis. Since 2012, suspected opioid overdoses have been steadily increasing. National data collected from 2011 to 2016 found the 10 most frequently mentioned drugs in overdose deaths included six opioids (fentanyl, heroin, hydrocodone, methadone, morphine and oxycodone), two benzodiazepines (diazepam and alprazolam), cocaine, and methamphetamine. In South Dakota, a similar pattern was noted with fentanyl, heroin, oxycodone, and hydrocodone accounting for four of the top five medications involved in drug-related overdose deaths in 2017. Treatment with opioids can help manage pain however, research indicates some patients will misuse them. A recent study found that patients with symptoms of acute or chronic, non-cancerous pain, seeking care at a physician’s office, received an opioid prescription roughly 20 percent of the time. Vowles and associates conducted an analysis of 38 studies, 35 conducted in the U.S., from 2000 to 2013 and found that approximately 21-29 percent of patients with chronic pain prescribed opioids will misuse them, which was defined as “use not in accordance with prescribed directions, regardless of the presence or absence of harm resulting from use.” The Henry J Kaiser Family Foundation reported the death rate from opioid overdoses in South Dakota, when age-adjusted, was 5 per 100,000 population in 2016, which ranked fifth lowest in the country.

Due to national trends, the Centers for Disease Control (CDC) has published a guideline on the use of opioids in chronic pain not related to end-of-life care, palliative care, or the treatment of active cancer. The CDC recommends before starting chronic pain therapy realistic treatment goals and outcomes of therapy should be established. It is also important to determine the underlying cause of the pain to guide pharmacologic and nonpharmacologic treatment options. When determining which agent to initiate for chronic pain, the CDC recommends nonpharmacologic treatments and/or nonopioid agents such as acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), and select antidepressants or anticonvulsants. Treatment should provide the patient with the greatest benefit in pain reduction with minimal risk of harm. If opioid therapy is appropriate, they recommend starting with the lowest effective dose of an immediate-release formulation in conjunction with other nonopioid agents as appropriate. Extended-release/long-acting opioids have been noted to have a higher risk of overdose when used as the initial agent and thus are not recommended. Re-evaluation of therapy is suggested at a minimum of every three months for patients on chronic therapy and within one to four weeks of initiation or dose escalation. When possible, a dose reduction of roughly 10 percent per month is encouraged. Opioids should be discontinued if the patient is not receiving any clinically meaningful pain relief.

Careful reassessment of the patient’s risks and benefits should occur if increasing to a dose 50 or greater morphine milligram equivalents (MME) per day and a dose 90 or greater MME per day is recommended to be avoided without justification. To calculate a patient’s MME per day, total the daily dose of each opioid medication taken and use the conversion factor to adjust for medication potencies; this sum value is the patient’s total MME per day. A table of MME can be found in the CDC guidelines. Examples of a prescription which contains roughly 50 MME per day include: 50 mg of hydrocodone (10 tablets of hydrocodone/acetaminophen 5/325 mg), approximately 33 mg of oxycodone (6 tablets of oxycodone/acetaminophen 5/325 mg or 2 tablets of oxycodone ER 15 mg), or 12.5 mg of hydromorphone (3 tablets of hydromorphone 4 mg).

Along with routine monitoring recommendations, additional actions are recommended to assess and mitigate risk factors and the potential for harm. The CDC suggests use of a urine drug screening prior to initiating opioid therapy, and at least annually, to ensure compliance with opioid therapy and to determine if the patient is taking
additional controlled substances or illicit drugs. The guidelines suggest avoiding the prescribing of benzodiazepines concurrently with opioids. Park and associates\(^7\) research found that over half of patient overdose deaths occurred with concomitant benzodiazepine and opioid therapy compared to opioid monotherapy. The CDC also suggests the prescription drug monitoring programs (PDMP) should be checked every three months to assess for dangerous combinations and other prescriptions. Naloxone is also a possible strategy to reduce the risk of harm. Naloxone, available as an auto-injection or nasal spray, is an opioid antagonist which can reverse the respiratory depression that occurs during an opioid overdose. Naloxone therapy should be considered for patients on opioids at high-risk for overdose, such as those with a history of substance abuse or overdose, an opioid dose of 50 or greater MME per day, or concomitant prescribing of a benzodiazepine.\(^1,8\)

Acute pain treatment can lead to long-term opioid use. Shah and associates\(^9\) studied commercially insured, opioid naïve, adult patients excluding those with a cancer diagnosis or substance abuse disorder from 2006-2015. They found an increased risk for chronic opioid use at 1 and 3 years began as early as the third day of therapy and increased with each additional day of therapy. The sharpest increases were observed in those patients who were on therapy more than 5 or 31 days, had an initial prescription for a 10 or 30 day supply, had a second prescription or refill, or a cumulative dose of 700 or greater MME. Based on their findings they suggested that prescribing less than 7-day supply, ideally 3 or fewer days, could reduce unintended chronic opioid use. The CDC guidelines also recommend that when used for acute pain, opioids should be at the lowest effective dose effective and only for the expected length of the pain severe enough to require opioids.\(^1\)

Many states have begun to implement changes aimed at limiting initial opioid prescription quantities. In May 2018, South Dakota Medicaid released a statement on changes to the South Dakota Medicaid prescription drug benefit regarding opioid prescriptions to align with the CDC guidelines.\(^1,10\) Medicaid patients who have a 30-day prescription for a controlled substance are now eligible for a refill after 26 days.\(^10\) Prior authorization is now required for a patient who requires more than one long acting and one short acting opioid agent to control their pain. Opioid naïve, defined as those who have not filled an opioid within the previous 60 days, are limited to an initial fill of a 7 day supply and maximum of 60 morphine equivalent dose. The final change began in October 2018 requiring prior authorization for prescriptions exceeding a 300 morphine equivalent dose. Each month the morphine equivalent dose required for prior authorization is decreased until reaching a 90 morphine equivalent dose on Oct. 1, 2019. This scheduled dose reduction was designed to mimic approximately a 10 percent dose reduction each month, which will hopefully minimize the impact to the patient and allow sufficient time to successfully implement changes safely. Patients with a terminal diagnosis are exempt from the changes except the early refill date.

During this opioid epidemic, guidelines and recommendations will continue to be developed for chronic and acute pain treatment with the goal of preventing medication misuse. By implementing changes in acute pain treatment requiring opioids, such as limiting initial fill quantities and day supply and avoiding long acting opioids, patients are less likely to continue therapy past acute treatment needs. For patients on chronic opioid therapy utilization of the lowest effective dose, establishing goals of therapy, regularly monitoring therapy and utilization of drug testing and PDMP data to ensure appropriate use as well as consideration of naloxone are all important strategies to maximize benefit while reducing the risk of harm. Implementation and adoption of these strategies will hopefully improve safety of pain therapy and patient outcomes.

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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Mr. D, a diabetic patient of mine, came into the clinic with exercise induced leg pain. Initially the pain would go away if he stopped exercising, but now it was coming on after walking less than a block. He said, “the pain is meaner and lasts longer after I stop.”

The diagnosis of Mr. D’s condition is claudication, a condition where arterial flow to the legs is blocked. I immediately called the vascular specialist for an urgent appointment. Two weeks later the patient returned to the office after having had his blocked arteries dilated with a balloon followed by the placement of stents to hold them open. Happily, he told me he could once again walk for miles without pain.

His vascular doctor had started artery dilating meds, lipid meds, and daily baby aspirin. I renewed my encouragement for Mr. D to eat fewer calories, exercise daily, keep his blood sugars controlled, and the most important advice, “Stay away from sitting long on that darn couch. It’s like smoking.”

The narrowing process, called atherosclerosis, can be the result of genetic tendencies, diabetes, smoking, high blood pressure, high blood lipids, and even normal aging. Arteries usually narrow gradually, but atherosclerosis can sometimes trigger a clot and a sudden complete blockage resulting in a sudden, and devastating event. It can occur in coronary arteries of the heart, cerebral arteries of the brain, renal arteries of the kidneys, and peripheral arteries of the legs. And, in general, when it’s happening in one, it’s happening in all.

Narrowing of blood flow can deprive muscles, skin, ligaments, and nerves downstream of precious blood flow and oxygen, resulting in an aching and agonizing hurt that would bring anyone to their knees. When it happens in the legs, like what happened with Mr. D, the pain symptom is called claudication and the condition is called peripheral artery disease or PAD.

PAD affects five percent of all people over 50, but more than 30 percent of all diabetics in the same age group. There can be a curve ball with diabetes or with aging, as these people sometimes lose feeling in their legs and feet due to nerve destruction, setting them up for PAD without pain. The presenting sign then would be painless sores on the feet which won’t heal. This is especially treacherous because, without good blood flow, the healing is very slow and the patient, having no pain, is not as motivated to do the work required to heal ulcers.

Leg pain that goes away with rest may not sound so bad, but peripheral arterial disease is one of the most formidable challenges in modern medicine.
Since 1963, February has been known as American Heart Month. Designed to encourage Americans to battle against heart disease, the health observation pays tribute to efforts by providers, public health officials, researchers, patients and volunteers. Because this disease can potentially be delayed or prevented, efforts are made to raise awareness and promote change.

Historically thought of as a disease prominent in middle-aged or older men, data indicates younger men and women are also at risk and need to be appropriately assessed. Heart disease is the leading cause of death for both men and women. The condition is responsible for over 600,000 deaths a year, which equates to approximately one in every four deaths in the United States.

Awareness via social media and public service announcements has stressed the concept that this disease is widespread and lifestyle changes are extremely important in combating and lowering adverse events. Obesity is a contributing factor that continues to worsen in all ages in our society, especially in sedentary youth. Diabetes impacts thirty million patients and serves as a significant risk. Other risk factors include hypertension, elevated cholesterol, and smoking. It is estimated that 50 percent of Americans have at least one of these three factors.

The Centers for Disease Control and Prevention (CDC) estimated that 50 percent of men and 64 percent of women who die suddenly from heart disease have no previous symptoms. Becoming aware of warning signs and risk factors is vital to help prevent sudden death.

The Department of Health and Human Services is engaged in the Million Hearts Initiative, which began in 2011 and is now known as Million Hearts 2.0. Emphasis for the initiative is on engaging patients in healthy behaviors and promoting extended use of cardiac rehabilitation, which support the National Prevention Strategy. In addition, health disparities are addressed through efforts aimed at racial and ethnic minorities.

The Great Plains Quality Innovation Network partners with home health providers, physician offices, patients and families to improve cardiac care. Technical assistance and best practices for prevention and cardiac care include efforts related to the ABCS measures (aspirin therapy, blood pressure control, cholesterol control and smoking cessation) and home health agency participation in a HHQI cardiovascular data registry. Assisting physician clinics with the Quality Payment Program improvement measures involving cardiovascular risk factors and treatment has the dual benefit of improving cardiac health and addressing payment reform and value-based purchasing.

Clinical guideline transformation, accurate diagnosis and increased individual management of hypertension and hyperlipidemia continue to evolve. Encouraging patients and their families to become engaged in this ongoing battle, from promoting medication compliance to healthy diet and exercise, is crucial to all aspects of care and will require innovation and teamwork. Healthcare professionals in all healthcare settings can help foster healthy hearts in February and throughout the entire year.

More information on cardiac health can be found on the Great Plains QIN web site (https://greatplainsqin.org/initiatives/cardiac-care) or by contacting Stephan Schroeder, MD, CMD, CMQ (Stephan.Schroeder@area-a.hcqis.org) or Holly Arends, CHSP, CMQP (Holly.Arends@area-a.hcqis.org).
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Nominations Now Open for 2019 SDSMA Awards

Nominations for the 2019 SDSMA Awards are now being accepted – visit sdsm.org to nominate a physician or supporter today!

Each year the SDSMA recognizes physician members and supporters for their work to improve the practice of medicine in South Dakota by presenting five distinguished awards.

Please consider nominating a colleague or supporter who is deserving of recognition for his or her work. They may work right alongside of you, or serve on a committee with you, or volunteer in your organization or community, or maybe they are your mentor. Through these awards, the SDSMA strives to encourage and recognize the highest standards of service, and give recognition to the accomplishments and dedication of our members and supporters to the medical profession and citizens of South Dakota.

The SDSMA is seeking nominations for the following:

- Distinguished Service Award
- Community Service Award
- Young at Heart Award
- Outstanding Young Physician Award
- Richard P. Holm, MD, Media Award

Visit sdsm.org to review each award and complete a nomination form. Those with questions may contact Elizabeth Reiss at ereiss@sdsm.org.

Nominate someone today and help your colleagues and supporters get the recognition they deserve!

Memorial Scholarship Established in Honor of Former SDSMA CEOs

A memorial scholarship has been started in honor of former SDSMA CEOs who have passed away. Contributions will be used for medical school scholarships.

Please mail contributions to SDSMA Foundation, 2600 W 49th St Ste 200, Sioux Falls, SD 57105 with CEO Memorial Scholarship in the memo line.

Donors may contribute in honor of a specific former CEO by including their name in the memo line:

- Richard C. “Dick” Erickson, SDSMA CEO from 1964-1972, passed away Nov. 30, 2018
- Paul Jensen, SDSMA CEO from 1999-2006, passed away Dec. 5, 2018
- Robert D. "Bob" Johnson, SDSMA CEO from 1972-1999, passed away Nov. 27, 2017

SDSMA Membership Services works hard to ensure that you have the programs and services you want and need, as well as marketing the association to potential new members. We want to hear from you if you have questions, concerns or ideas on how we can serve you better, or if you know of a potential new member. It’s your association and we’ll work with you to make it the best it can be.

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

February 2019
SSDM Leadership Meet with Gov. Noem

SSDM President Christopher Dietrich, MD, and CEO Barb Smith met with Gov. Kristi Noem to discuss issues of concern to the SSDMA. In that meeting, the SSDMA visited with Gov. Noem about prescription drug abuse and diversion, and the need to focus on prevention and treatment – both of which the newly sworn governor indicated as priorities in her 2019 State of the State address.

While meeting with the governor, SSDMA also spoke on the issue of graduate medical education. South Dakota is facing a critical shortage of physicians as more than 1 in 4 South Dakota residents live in what has been classified as a “primary care shortage area.” Workforce experts predict that the U.S. will face a shortage of 130,000 physicians across all specialties by 2025. The SSDMA asked Gov. Noem for her assistance to increase Medicare support and federal funding for GME, and to lift the cap on GME positions to address the physician shortages in undersupplied specialties and underserved areas.

The SSDMA also shared with the governor the Association’s concerns regarding the current and ongoing closure of long-term care facilities across South Dakota. Small, rural communities have been hit especially hard. When these facilities close, it impacts residents, their families as well as members of the community. When rural nursing facilities close their doors, it results in an immediate loss of jobs for those employed by the facility. That loss extends to those who support the facility as well as leading to fewer patients to care for in the clinic and hospital settings.

Mobridge and Madison are currently dealing with these losses as a result of the foreclosure of their nursing facilities.

SSDM Doctor of the Day Program in Full Swing as 2019 Legislative Session Begins

The SSDMA Doctor of the Day Program at the State Capitol during the legislative session highlights the importance of medical care to our representatives.

Physicians representing the SSDMA as Doctor of the Day provide medical assistance should any emergency arise at the Capitol. The Doctor of the Day is also able to attend committee meetings, watch sessions of the House and Senate, and visit with South Dakota’s representatives to offer views on healthcare.

Thank you to our volunteers! You can see a full listing of volunteers and learn more about the program at sdsma.org. 2019 dates have been filled; however, those interested in volunteering next year, please contact the SSDMA office.

Pictured is Greg Gerrish, MD, of Watertown, who was Doctor of the Day on Jan. 16, along with Gov. Kristi Noem and Policy Analyst Kennedy Noem.
A physician attending a person diagnosed with or suspected of having a reportable disease or condition must report the disease or condition to the South Dakota Department of Health (SDDOH).

In many cases, state law dictates how and when such a report must be made. In most cases, the physician remains obligated to make the report despite designation of another person to make such report. Reports mandated by state law are allowable under the HIPPA mandated federal medical privacy requirements.

Category I diseases and conditions are reportable immediately by telephone. Category II diseases and conditions are reportable within three days after recognition or strong suspicion of disease.

In addition to communicable diseases, physicians are required to report information regarding the performance of abortion, auditory or visual impairment, cancer, child abuse, elder abuse, fetal alcohol syndrome, incidents of fetal death, gunshot wounds, certain metabolic disorders, ophthalmia neonatorum, patient drug diversion, syphilis, tuberculosis and certain venereal diseases.

For more, view the South Dakota Department of Health list of reportable diseases at https://doh.sd.gov/diseases/infectious/reporting.aspx. And, download the SDSMA legal brief Reportable Diseases and Events at sdsm.org. Through the SDSMA Center for Physician Resources, the SDSMA has developed more than 50 legal briefs that are available to members. In addition, the Center develops and delivers programs for members in the areas of practice management, leadership and health and wellness.

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Read InSesion for Legislative News

The SDSMA’s weekly email newsletter InSession keeps you informed about the SDSMA’s legislative activities and key health-related bills and action alerts. In addition to being emailed to members, InSession is also posted at sdsm.org. InSession is published once per week during the legislative session, providing a timely look at the actions of the legislature.

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- South Dakota State Medical Association
- Utah Medical Association

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