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A Time of Changes for Medicine

By Tom Hermann, MD
SDSMA President

As I assume the role of SDSMA president for the coming year, let me share with you why I think participation in organized medicine is so important to us all. In doing so, I will not only ask you to prepare yourself professionally for the changes that lie ahead but will encourage you to become involved and to be active in helping shape the future of medicine.

As physicians we know the key to identifying and helping to solve problems is knowledge of the subject; obtaining a good medical history and examination; and then based on the assessment, a good plan of action. We have reached a point in time where we must now apply that same process to ensure the very survival of how we practice medicine.

A monumental change in the delivery and reimbursement for medical care lies ahead as the Centers for Medicare and Medicaid (CMS) seeks to reform how the government pays for medical services. The recently passed Medicare Access and CHIP Reconciliation Act (or MACRA) will transition health care providers from a reimbursement model of compensation based on fee for services to one in which providers are reimbursed based on quality, performance, and meaningful use. Other alternative payment models will also be considered. MACRA will profoundly affect payment to all providers, clinics, hospitals and the health systems, and our teaching institutions – to include funding and reimbursement for our medical students, mid-level providers, and residents.

In March it was my privilege to attend, along with Drs. Mary Carpenter and Tim Ridgway, the American Medical Association (AMA) National Advocacy Conference in Washington, D.C. We were favorably impressed by our AMA’s leadership, knowledge of the issues, and communication and input to CMS. Of note, Andy Slavitt, acting administer of CMS, acknowledged how helpful and important the AMA has been and will continue to be with respect to federal policy issues. It is the AMA’s intent to provide guidance to CMS during this transition in such a way that the final outcome will include revised administrative and billing processes that are less burdensome, and to ensure greater rewards for the delivery of high quality health care. Congressional members and CMS staff are hearing the voices of our AMA in conjunction with other leaders of organized medicine. To help you keep up to date on these and other federal activities, the AMA Wire is an online resource for AMA members that includes summaries on the happenings in Washington D.C., and I strongly recommend it to you.

Changes in medicine are not only happening at the federal level but at home as well. In late 2016, the SDSMA voted to change our bylaws, and over the next 12 months, we will transition to a smaller Policy Council and Board of Directors form of government. The Policy Council will meet twice a year to discuss, develop and adopt policy for the association, while the Board will handle much of the day-to-day activities and operations.

The SDSMA continues to work hard to advocate on behalf of our members and our profession. Our advocacy efforts, led by our dedicated SDSMA staff and skilled lobbyists, brought forth legislation in 2016 that will allow physicians to prescribe opioid antagonists to family members and those closest to individuals at risk of an overdose. Of note, I would like to express my gratitude to the thoughtful participation of those who supported the SDSMA Doctor of the Day program this legislative session. The Doctor of the Day program not only provides a valuable service to our legislators in Pierre, it provides a positive image of physicians and organized medicine. Last fall, the SDSMA worked closely with the South Dakota Board of Medical and Osteopathic Examiners to develop and adopt guidelines on the white paper Prescribing of Opiate Analgesics for Chronic Non-Cancer Pain – the guidelines were developed in favor of having restrictions on prescribing placed in rule. And the SDSMA continues to work with Gov. Daugaard’s South Dakota Health Care Solutions Coalition in hopes of developing a meaningful plan and funding mechanism that will receive legislative budget approval, and lead to the expansion of Medicaid.

Other association-related activities include DAKOTACARE, which despite undergoing a change in ownership, remains a statewide insurance plan with SDSMA endorsement, and the SDSMA Foundation which awarded several scholarships for our medical students. I would also like to mention the ongoing development of the SDSMA Center for Physician Resources which will launch its first foundational leadership course cohort later this year.

Your SDSMA leadership, guided by Past President Dr. Tim Ridgway, the Council of Physicians, and the district medical societies represents you well. Physician leadership is important and I urge you to be a voice for medicine and your patients. As things change, we must work together to advocate for, and to protect and promote the ideals of our profession.
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In this month’s issue of South Dakota Medicine, we have a number of interesting, thought provoking manuscripts. Our new President, H. Thomas Hermann, Jr., MD delivers his first in a monthly series of editorials. This is a challenging time in organized medicine with local, regional and national pressures exerted on the practice of medicine. I look forward to reading Dr. Hermann’s editorials to update us on what is happening in the South Dakota State Medical Association and American Medical Association.

Dr. Petereit and his group provide data in the article “A Multi-faceted Approach to Improving Breast Cancer Outcomes in a Rural Population, and the Potential Impact of Patient Navigation.” Dr. Petereit has contributed substantially to the literature on how to provide up-to-date, evidence-based medicine to patients with cancer in rural and frontier communities. In this publication, they discuss how breast conservation treatments have increased in South Dakota to mirror those of national rates. The reason for this increase in breast conservation in the treatment of breast cancer appears to by multi-factorial with a number of potential causes. Breast cancer patient navigation was not significantly associated with increased breast conservation; however, it was an important component of caring for the entire patient. I applaud Dr. Petereit and his group for the wonderful work they are doing in improving cancer care for our patients.

This issue also has a Primers article, “Peripheral Neuropathy Update.” In this manuscript the authors give up-to-date recommendations on the evaluation and management of patients with peripheral neuropathy, an oftentimes debilitating condition.

There are also three interesting case reports in this issue. Two of these case reports deal with patients suffering from cardiovascular disease; the first a discussion of a patient with acute papillary muscle rupture after myocardial infarction and the other a case of an acute stent fracture. Another case report described an unusual presentation for celiac disease in an elderly patient. In this case the elderly woman presented with a burning tongue. This case report reminds us of the importance of a detailed history and physical examination with the subsequent obtaining of laboratory tests dependent on these findings.

The developmental origins of adult disease (Barker’s hypothesis) was first hypothesized by Dr. David Barker in the 1990s. This hypothesis posited that adult diseases such as hypertension, diabetes, cardiovascular disease and others could be predisposed to by stresses that happen in the womb. The classic studies demonstrated that individuals who had intrauterine growth restriction were at higher risk for developing diabetes mellitus as an adult. Recent studies suggest that changes within the mitochondria (power house of the cell) may play an important role in Barker’s hypothesis. Mitochondria are maternally inherited. Within the developing follicle the oocyte rapidly expands its number of mitochondria, to the point where there are more mitochondria within a mature egg than within any somatic cell, even those with rapid metabolic rate. At the time of ovulation the egg is cut-off from the maternal system (gap junctions between the egg and granulosa cells break down at the time of the LH surge) and mitochondria stop replicating. Following fertilization the embryo undergoes progressive cell division which dilutes the number of mitochondria present within each cell. The mitochondria do not start replicating again until after blastulation. One interesting issue is that activation of the egg, fertilization and cell divisions require ATP, which is provided by the slowly shrinking pool of mitochondria. One can see that alterations within the mitochondria which could interfere with oxidative phosphorylation (the process to make ATP) could have a profound impact upon the developing embryo.

Mitochondrial DNA is exposed to low levels of reactive oxygen species (which can damage the DNA) from their own oxidative phosphorylation from the time of formation of primordial follicles within the embryo until
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ovulation (which can be decades). As a woman grows older these mutations increase and reduce the metabolic efficiency of mitochondria. To compensate for this reduction in efficiency oocytes from women of advanced age have an increased number of mitochondria. Because ATP is necessary in cell division especially in formation of the spindle apparatus it is hypothesized that these changes may result in increased risk of aneuploidy in the resultant embryo.

If the embryo survives then it will have these mitochondria with reduced efficiency as well as normal mitochondria inherited from the egg. Previous studies suggested that the normal mitochondria would replicate better and replace the inefficient ones (those with mutations), resulting in an individual with normal mitochondria. Recent studies suggest that the mitochondria with mutated DNA and accompanying inefficiencies have the same chance of replicating and persisting as the normal mitochondria. This suggests that a mutated mitochondrial DNA can be passed to offspring through the maternal cell line very rapidly. These abnormal mitochondria with altered metabolism may increase the risk of insulin resistance, the metabolic syndrome and diabetes mellitus thus supporting Barker’s hypothesis. Further studies on how mitochondria relate to Barker’s hypothesis are needed, as well as potential substances/conditions that can cause mutations or interfere with mitochondria and potentially be inherited by offspring.
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A Case of Post Myocardial Infarction Papillary Muscle Rupture

By Amornpol Anuwatworn, MD; Christopher Milnes, MS IV; Vishesh Kumar, MD; Amol Raizada, MD; Verlyn Nykamp, MD; and Adam Stys, MD, FACC, FASA, FSCAI, FACP

Abstract
Papillary muscle rupture is a rare, life-threatening post myocardial infarction mechanical complication. Without surgical intervention, prognosis is very poor. Clinicians need to recognize this complication early, as prompt therapy is crucial. We present a case of inferior ST elevation myocardial infarction complicated by posteromedial papillary muscle rupture resulting in severe acute mitral regurgitation (flail anterior mitral leaflet), acute pulmonary edema and cardiogenic shock. In our patient, a new mitral regurgitation murmur suggested this mechanical complication. Complete disruption of papillary muscle was visualized by transesophageal echocardiography. This case illustrates the importance of good physical examination for early diagnosis of papillary muscle rupture, so that life-saving treatment can be administered without delay.

Introduction
In the era of reperfusion therapy using primary percutaneous coronary intervention, papillary muscle rupture (PMR) is a very rare (0.26 percent), but often catastrophic complication.1,2 The survival rate of patients with post myocardial infarction mechanical complications at 30 and 90 days is 44 percent.1

Case Presentation
A 70-year-old man, otherwise healthy, presented to a referring hospital with angina pectoris at rest for 10 days. He developed severe dyspnea at rest on the day of presentation. Physical exam revealed blood pressure of 107/59 mmHg, heart rate of 125 beats per minute, respirations of 32 breaths per minute and oxygen saturation of 92 percent on face mask with positive airway pressure. He appeared critically ill, pale, diaphoretic, and was using accessory respiratory muscles. A holosystolic 3/6 murmur loudest at the apex was heard along with diffuse rales over both lungs. Peripheral pulses were diminished. ECG showed ST elevation with Q waves and T wave inversion in leads II, III, and aVF. An urgent transthoracic echocardiogram (TTE) showed left ventricular ejection fraction of 45 percent with inferior wall hypokinesis and severe mitral regurgitation (MR). Initial troponin was 5.08 ng/mL (normal range of 0-0.03 ng/mL). Creatinine was 2.00 mg/dL. Chest X-ray showed pulmonary edema. The patient was treated with aspirin, subcutaneous enoxaparin and intravenous furosemide. He required intubation before the transfer to our hospital.

As a new holosystolic murmur loudest at the apex in a patient with acute coronary syndrome suggests acute mitral regurgitation secondary to rupture of papillary muscle or chordae tendineae; emergent transesophageal echocardiogram (TEE) was performed on arrival, which showed posteromedial papillary muscle rupture (Figure 1) with flail anterior mitral leaflet and severe eccentric mitral regurgitation (Figure 2). Coronary angiogram revealed 100 percent occlusion of the distal right coronary artery (Figure 3) with non-obstructive disease in the left coronary system. An intra-aortic balloon pump was inserted and emergent mitral valve replacement with coronary artery bypass grafting was performed. A 29 mm St. Jude Epic bioprosthesis was implanted. Pathology confirmed papillary muscle head (Figure 4). Troponin peaked at 19.12 ng/mL. The patient was discharged in stable condition after nine days in the hospital and was doing well on eight weeks ambulatory follow-up.
Discussion

PMR is commonly reported in inferior wall ST elevation myocardial infarction (STEMI) as the rupture of posteromedial papillary muscle is more common due to a single blood supply by the posterior descending coronary artery. Anterolateral papillary muscle is supplied by both left anterior descending and left circumflex coronary arteries. As illustrated in our case, the patient developed rupture of the posteromedial papillary muscle due to total occlusion of the distal right coronary artery. In addition, PMR usually occurs between two to seven days after STEMI. Complete rupture of papillary muscle results in acute mitral regurgitation, acute pulmonary edema and cardiogenic shock. This complication is life-threatening; therefore, early diagnosis of PMR is vital. Acute MR secondary to PMR should be suspected if patients have a new holosystolic murmur. Traditionally, this murmur is heard loudest at the apex and radiates to axilla. This is in contrast to ventricular septal rupture murmur which is a harsh holosystolic murmur heard loudest at the left sternal border. TTE is an initial diagnostic tool with a sensitivity of 65 to 85 percent. Although prolapse or flail of the mitral leaflet and regurgitant jet can be easily seen on TTE, ruptured papillary muscle may not be identified.
TEE improves diagnostic yield of PMR.\textsuperscript{4}

Without emergent surgical intervention, the mortality of acute post infarction MR is 70 percent to 80 percent.\textsuperscript{1} One-third of patients die immediately, 50 percent pass away within one day and only 6 percent survive longer than two months. With early diagnosis and prompt surgical intervention, the mortality is less than 30 percent.\textsuperscript{5} Furthermore, hemodynamic stabilization with inotropic support, afterload reduction and use of intra-aortic balloon pump is recommended until surgery can be performed. Intra-aortic balloon counter pulsation helps to reduce the afterload/regurgitant volume and improve stroke volume in acute MR.\textsuperscript{2,5} In addition, mitral valve surgery with concomitant coronary bypass graft surgery has been shown to improve long-term survival.\textsuperscript{2}

Conclusion

Good history and physical examination is important for early recognition of acute MR secondary to PMR. A new MR murmur, acute pulmonary edema and cardiogenic shock following an episode of acute STEMI may indicate this mechanical complication. TEE is a useful imaging modality to confirm diagnosis of PMR. Although the rate of muscle rupture is declining, it is important to stay vigilant when evaluating these patients.

REFERENCES


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Burning Tongue as Initial Presentation of Celiac Disease in an Elderly Woman: A Case Report

By Andrea Sherman, MS III; and Alla Zamulko, MD, PhD

Abstract
There are few reports in the literature where celiac disease presents with tongue manifestations, although atypical presentations of celiac disease are not uncommon. This case report highlights an atypical presentation of celiac disease in an elderly female. Our patient presented to clinic with complaints of a burning tongue for the past two years as well as occasional loose stools and fatigue. Work-up revealed iron deficiency anemia, zinc deficiency and an abnormal celiac panel. Complete symptom improvement was noted by 10 weeks into the initiation of a gluten free diet. Celiac disease can present at any age and should be considered as a differential in findings of malabsorption and gastrointestinal symptoms.

Introduction
Oral manifestations in celiac disease (CD) are fairly common, including increased incidence of dental enamel defects, oral soft tissue lesions, and atrophic glossitis. While one study showed that 29.7 percent of patients with CD experienced tongue burning and soreness, there are few case reports found in the literature where the presenting symptom of CD was a burning tongue.1 In Italy, a 33-year-old male presented with an atrophic, burning tongue of four-month duration and was eventually diagnosed with CD. His glossitis resolved within one month of a gluten free diet.2 Another case was that of a 17-year-old Sardinian woman whose dentist identified atrophic glossitis and tongue ulcerations and work-up revealed nutritional deficiencies and CD. In discussion with the patient and parents, it was determined that the patient had had these tongue symptoms intermittently since the age of 5.3 A third case was a 72-year-old female from Italy whose only symptom was burning at the dorsum of her tongue, a diagnosis of CD and the start of a gluten free diet led to symptom resolution one month later.4

Case
Our patient is an 81-year-old female with a past medical history of gastroesophageal reflux disease and duodenal strictures who presented with complaints of a burning tongue. Her tongue had been burning daily for the past two years causing pain and discomfort with no associated mouth sores. Other symptoms included occasional loose stools and fatigue. She denied weight loss, dysphagia, symptoms of heartburn, or melena. The patient was seen by her dentist every six months and never had this addressed with her dentist nor had abnormalities mentioned. Examination of the tongue showed a 3 to 4 mm, elevated, lesion on the right side of the tongue, prominent taste buds, but no ulcerations or other lesions present (Figure 1). Differential of a burning tongue are listed in Table 1. As part of the work-up for a burning tongue, it was discovered that the patient had significantly low IgA and an elevated gliadin peptide IgG and IgA greater than seven times the normal range. Her anti tissue transglutaminase (anti-tTG) and endomysium antibody (EMA) levels were normal. Further lab work-up revealed severe iron deficient microcytic anemia and zinc deficiency. Comprehensive Metabolic Panel (CMP), magnesium, folate and vitamin B12 levels came back within normal limits. A test for Candida was also done and came back negative. An upper endoscopy was not done, as she had one performed two years prior which showed ulcerative duodenitis with no evidence of malignancy or Crohn’s and immunohistochemical staining for H. pylori and Cytomegalovirus (CMV) were negative. A colonoscopy at that time was...
normal. Ear nose throat (ENT) was also consulted initially to rule out any malignancy or nerve damage.

After the initial visit, the patient was given a dexamethasone mouthwash suspension, which she took for two weeks. After the results of the celiac panel, the patient was advised to start a trial gluten free diet and to take iron and zinc supplements. We saw the patient in follow-up two and four weeks after the initial visit and initiation of the gluten free diet. She noticed gradual symptom improvement with a decrease in oral pain, improvement in bowel function, and her tongue showed less inflammation on exam than the initial visit (Figure 2). Complete resolution of the burning tongue was seen 10 weeks after the initiation of the gluten free diet.

**Discussion**

It was suspected that this patient has seronegative celiac disease. Although elevations in anti-tTG IgA are up to 98 percent sensitive and 95 percent specific and positive EMA IgA is greater than 90 percent sensitive and 95 percent specific for CD, they are unreliable lab markers in patients who have IgA deficiency. Her IgA levels were below 3 mg/dL, normal being 61 to 356 mg/dL. Having IgA deficiency also places her at risk for CD, as IgA deficiency is associated with a 10 to 20 greater risk than the general population for developing CD. Although anti-gliadin IgG and IgA is less sensitive and specific than anti-tTG, it is still up to 90 percent sensitive and 85 to 95 percent specific. In addition, she presented with a malabsorptive scenario with severe iron deficiency anemia and zinc deficiency with other causes of iron deficiency ruled out via a normal colonoscopy. In cases of suspected

<table>
<thead>
<tr>
<th>Table 1. Differential Diagnoses for Burning Tongue[^1-6]</th>
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<tr>
<td>Candidiasis</td>
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<td>Trauma</td>
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<td>Nerve Damage</td>
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<td>Burning Mouth Syndrome</td>
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<td>Celiac Disease</td>
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<td>Vitamin Deficiency</td>
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<td>Zinc</td>
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<td>Diabetic Neuropathy</td>
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<tr>
<td>Drug induced Xerostomia</td>
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<tr>
<td>Depression</td>
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[^1-6]: The table lists various conditions that could cause burning tongue symptoms.
CD in concurrence with normal anti-tTG, negative EMA, and low IgA, a trial gluten free diet can aid in diagnosis. In our case, our patient had noticed improvement in her bowel function and tongue inflammation and pain upon initiating a gluten free diet.

The purpose of sharing this case is to bring awareness of one of the ways that celiac disease can present. Celiac disease is an autoimmune disease with antibodies against the gluten protein, gliadin. CD primarily affects the mucosa of the small intestine, resulting in villous atrophy. Although the typical presentation of CD is gastrointestinal upset, such as chronic diarrhea and abdominal pain, CD may actually present more commonly with atypical symptoms or malabsorption. In addition, while most CD diagnoses occur in young adults, it can start at any age. Presentation of CD in the elderly may be reflective of its malabsorptive effects such as cachexia, iron deficiency anemia, neurological disorders and even severe osteoporosis.

While a diagnosis of CD can be straightforward with identification of elevated anti-tTG, positive EMA, and small intestine biopsy indicating villous atrophy, crypt hyperplasia, and lymphocytic infiltration, there may be times where the diagnosis is not that clear and obtaining IgA levels, anti-gliadin IgG levels, and a gluten free trial can aid in diagnosis of CD. Treatment for CD should include a gluten free diet as well as vitamin supplementation of any determined deficiencies.

REFERENCES


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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.
Introduction

Coronary artery stent fracture (SF) is a recognized complication that happens fairly infrequently. SF is associated with in-stent restenosis and possibly in-stent thrombosis. Few case reports of SF are found after initial implantation of days, months to years with presentations of acute coronary syndrome or progressive angina. We present a rare case of acute SF during post dilation of an everolimus eluting stent at a critical stenosis junction of a saphenous vein graft to the first diagonal of the left anterior descending artery. A shorter oversized drug eluting stent was placed to cover the stent fracture with good angiographic results. To our knowledge, this is the first incidence in literature of an acute stent fracture in a saphenous vein graft.

Case Report

A 75-year-old female presented to our facility with symptoms of unstable angina (chest tightness with exertion that has been accelerating in the past two to three weeks). Chest tightness was similar to previous angina when she required percutaneous coronary intervention (PCI) in 2008.

Past medical history was significant for coronary artery bypass (CABG) in 1994, and multiple PCI – the last one in 2008. She also has history of peripheral vascular disease and chronic kidney disease.

Cardiovascular physical exam was unremarkable except for high blood pressure (178/73) and decreased lower extremity pulses without significant edema. Electrocardiogram showed left ventricular hypertrophy (LVH) with repolarization abnormalities. Chest X-ray revealed mild linear scarring or atelectasis, but was otherwise normal. Initial troponin level was 0.17 ng/mL, with the second and third troponin six hours apart being 0.04 ng/mL. Echocardiography revealed a preserved left ventricular systolic function and moderate to severe concentric LVH with no significant valvular heart disease.

Review of her angiogram in 2008 revealed that she had PCI of the native right coronary artery (RCA) with complete stenting of the RCA with good angiographic results. PCI of the saphenous vein graft (SVG) to D1 was also done at the time, which resulted in complete stenting of the SVG (Figure 1).

Angiography on this admission revealed severe three-vessel coronary artery disease (CAD) with patent internal mammary graft to LAD and patent SVG to RCA. SVG to obtuse marginal artery was chronically occluded. SVG to D1 had distal anastomosis stenosis of 95 to 99 percent at touchdown with patent stent in D1 distal to anastomosis (Figure 1).

SVG to D1 anastomosis was initially dilated with a 2 mm x 12 mm balloon inflated to 22 atmospheric pressure...
(atm). Promus Premier (Boston Scientific) 2.5 x 16 mm drug eluting stent (DES) was deployed at 22 atm (Figure 2). Post dilation with noncompliant balloon (3.0 mm x 12 mm) inflated at 24 atm was performed for 20 seconds. After balloon deflation, the acute SF was noted. The decision was to place another shorter oversized stent to cover the SF, thus a Promus Premier 3 mm x 8 mm DES was deployed across the fractured segment at 20 atm. Final angiographic results was satisfactory (Figure 3). The patient did well after the procedure and was discharged the next day. Review of her chart showed that she was re-hospitalized within one month for acute cholecystitis which required a cholecystectomy tube, but no complications from the complex PCI.

**Discussion**

SF has been well documented in lower extremity stenting (femoral, femoropopliteal arteries) and is thought to be a result of external forces (movement) compressing the vessel causing undue stress on the stents. Coronary SF is a relative newly recognized complication of PCI, dating back to early 2000. These SF are thought to result from the prolonged rhythmic contraction of the heart that causes constant cyclical stress on the stent struts which eventually may lead to metal fatigue and fracture at critical hinge points. Risk factors for SF include stent length, RCA implantation (possibly due to motion of RCA in atrioventricular groove), overlapping of stents, vessel tortuosity, vein grafts, high pressure balloon inflations. Many of the cases of coronary SF were in the sirolimus eluting stent (SES) which was a first generation DES. In a metaanalysis by Chakravarty et al., most of the SF were in the SES, which were designed with thicker struts, allowing it to be more easily diagnosed on angiogram because it is more radiopaque. A possible explanation was that the SES was a closed cell design and comparatively thicker struts than the newer DES on the market now which are open cell design, which helps increase its longitudinal strength, and thus its lower incidence of SF.

SF correlate with adverse outcomes ranging from in-stent restenosis, in-stent thrombosis, coronary aneurysm to intracoronary perforation that can clearly have serious consequences. In one study, the incidence of SF was as high as 1.9 percent, but has been reported to range from 0.84 to 8.4 percent in first generation DES. Autopsy studies have shown SF to be as high as 29 percent suggesting many are previously undiagnosed SF. With the advent of DES, neo-intimal growth has dramatically reduced, resulting in lower in-stent restenosis rates compared to bare metal stents (BMS). At sites of SF, local delivery of the drug is impaired, and the free metal strut would be in direct contact with coronary blood flow.
which can cause focal re-stenosis. According to studies, target lesion revascularization (TLR) rate can range from 50 to 70 percent at sites of SF.\textsuperscript{13,14} Surprisingly, in the era of BMS, there were not many SF reported, likely because of the expected intimal hyperplasia as a protective mechanism in preventing the free metal strut from being in direct contact with the coronary blood flow.\textsuperscript{11} Treatment of SF are generally geared towards treating the lesion as an unstable plaque, and re-stenting the area of the SF with another DES as the new stent will allow for local drug delivery to prevent intimal hyperplasia.\textsuperscript{16} Mitomo et al. evaluated plain old balloon angioplasty (POBA) in the treatment of SF versus DES implantation retrospectively, and found that POBA group had higher TLR and higher

**Figure 2.** Complex angioplasty of SVG to D1 insertion site. (A) Initial balloon inflation (compliant 2 mm x 12 mm balloon). (B) Stent deployment of 2.5 x 16 mm DES. (C) Balloon inflation with noncompliant 3 mm x 12 mm balloon. (D) White arrow pointing at site of acute stent fracture of DES. SVG, D1 (first diagonal branch of the left anterior descending), DES.
incidence of major adverse cardiac events (MACE). However, due to incomplete understanding of the mechanism of focal stenosis in SF, and because not all SF are clinically relevant, there is no uniform approach to SF at this time. SF may be visualized with angiography; however, often it may be a subtle finding. Focal stenosis of a DES can be suspicious for a SF, and for confirmation, intravascular ultrasound can be used. In the experimental setting, computed tomography (CT) or optical coherence tomography (OCT) have been used as well.

Review of literature did show a case of an immediate SF after a zotarolimus eluting stent implantation in the RCA that required a complete stenting of the RCA. Immediate SF after deployment is an extremely rare event that is not well described. This is the first case to our knowledge of an acute SF in an SVG in the literature. The mechanical forces on the stent struts in our case were likely pronounced due to fibrosis, calcification, high pressure inflation, and acute angle at the insertion site of SVG. The distal to anastomosis previously implanted stent in the D1 branch could have contributed to the complication by stiffening the vessel system. The post dilation was performed with an oversized noncompliant balloon that could have contributed to acute fracture risk (Figure 2). The fractured stent parts were bridged with an oversized shorter DES with good angiographic and clinical results.

Conclusion
We present an extremely rare case of acute SF in SVG distal anastomotic site. The high pressure balloon post inflation of the stent in this clinical setting was challenging due to that patient’s coronary anatomy and could have contributed to her complication of acute SF.

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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Introduction

Peripheral neuropathy is a common diagnosis, affecting 2.4 percent of the general population, 8 percent of persons over 55 years of age, and 26.4 percent of patients with type 2 diabetes mellitus.1 As such, a thorough understanding of the clinical presentation and evaluation is relevant to practitioners of any specialty who are all likely to encounter patients with neuropathy. This article provides a framework for evaluating the patient with symptoms of peripheral nerve disease. Such evaluation includes identifying common causes based on specific symptom patterns; recognizing symptoms suggesting reversible causes or diseases requiring urgent treatment; and understanding initial laboratory and diagnostic screening based on the individual patient and the reliability of such testing.

Causes of Peripheral Neuropathy

A number of systemic diseases, disorders of metabolism, toxic exposures, medications, infections and hereditary disorders cause peripheral neuropathy. A brief overview of these causes is outlined below, and the following sections will address their characteristic clinical presentations with guidelines for recognizing unique patterns. The most common treatable causes are diabetes, hypothyroidism and nutritional deficiencies,1 all of which represent causes of acquired peripheral neuropathies. Most cases of peripheral neuropathy with a known cause are acquired (Table 1).

Hereditary causes of neuropathy include diseases in which the neuropathy is the sole or primary feature of the disease, and those in whom neuropathy is part of a more widespread neurological or systemic disorder. Charcot-Marie-Tooth disease, also referred to as hereditary motor and sensory neuropathy, encompasses a clinically and genetically diverse group of inherited neuropathies and represents the most common hereditary neuropathy.2

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Table 1. Causes of Acquired Neuropathies

<table>
<thead>
<tr>
<th>Systemic Disease</th>
<th>Medications</th>
<th>Toxins</th>
<th>Autoimmune</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes mellitus</td>
<td>Chemotherapy</td>
<td>Alcohol excess</td>
<td>Guillain-Barre syndrome</td>
</tr>
<tr>
<td>Thyroid disease</td>
<td>- Cisplatin</td>
<td>Heavy metals</td>
<td>(Acute inflammatory demyelinating Polyradiculoneuropathy - AIDP)</td>
</tr>
<tr>
<td>- Hyperthyroidism</td>
<td>- Vincristine</td>
<td>- Arsenic</td>
<td>Chronic inflammatory demyelinating Polyradiculoneuropathy (CIDP)</td>
</tr>
<tr>
<td>- Hyperthyroidism</td>
<td>Isoniazid</td>
<td>- Lead</td>
<td>Vasculitis</td>
</tr>
<tr>
<td>Cancer</td>
<td>Vitamin B6 excess</td>
<td>- Mercury</td>
<td></td>
</tr>
<tr>
<td>- Paraneoplastic syndrome</td>
<td>Amiodarone</td>
<td>- Gold</td>
<td></td>
</tr>
<tr>
<td>- Lymphoma</td>
<td>Digoxin</td>
<td>Organophosphates</td>
<td></td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>Lithium</td>
<td>Diphtheria toxin</td>
<td></td>
</tr>
<tr>
<td>Chronic liver disease</td>
<td>Statins</td>
<td>Tetanus toxin</td>
<td></td>
</tr>
<tr>
<td>Monoclonal gammopathy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Amyloidosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Multiple myeloma</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- MGUS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- IgM, IgG or IgA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sarcoïdosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Critical illness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uremia</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Nutrition</th>
<th>Infection</th>
<th>Compression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin deficiency</td>
<td>HIV/AIDS</td>
<td>Mononeuropathy</td>
</tr>
<tr>
<td>- B12</td>
<td>Lyme disease</td>
<td>- carpal tunnel syndrome</td>
</tr>
<tr>
<td>- B6</td>
<td>Leprosy</td>
<td>Radiculopathy</td>
</tr>
<tr>
<td>Malabsorption</td>
<td>Syphilis</td>
<td></td>
</tr>
<tr>
<td>- Celiac disease</td>
<td>Herpes zoster</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- postherpetic neuralgia</td>
<td></td>
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<td></td>
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<td></td>
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</tr>
</tbody>
</table>

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These and other less commonly encountered hereditary causes are outlined in Table 2.

Despite clinical and laboratory evaluation, as many as one-third of patients with chronic neuropathy will not have an identifiable cause for their symptoms. These idiopathic cases may be referred to as cryptogenic sensory polyneuropathy, which is a diagnosis of exclusion.

Clinical Presentation and Pattern Recognition

The evaluation of a patient presenting with symptoms of peripheral neuropathy should attempt to first localize the symptoms to the peripheral nerves, then identify the affected fibers and characterize the nature and distribution of symptoms in order to narrow the differential diagnosis. Once narrowed, further testing will define the underlying pathology and direct disease management. This section briefly outlines the initial questions that should be investigated in a patient presenting with symptoms of peripheral neuropathy.

Is there evidence of central nervous system involvement?

When a patient presents with distal numbness, tingling, pain or weakness, the first step is to rule out involvement of the central nervous system before categorizing the patient’s symptoms as peripheral neuropathy. The presence of central nervous system (CNS) symptoms, such as cognitive symptoms, difficulty speaking, double vision or visual field deficits, cranial nerve involvement or impaired bowel or bladder function suggests that peripheral nerve disease cannot be the only diagnosis, although it may be one component of the patient’s disease. CNS involvement may be indicated by the presence of these symptoms or with upper motor neuron signs on exam, including brisk deep tendon reflexes, a positive Babinski sign and muscle spasticity. The presence of these signs or symptoms would suggest a primary CNS lesion, the coexistence of neuropathy and an unrelated upper motor neuron problem or, in conjunction with symmetric sensory symptoms, a vitamin B12 or copper deficiency.

Does the patient have sensory, motor, autonomic or mixed symptoms?

The peripheral nervous system has autonomic and somatic components, and although either may be affected in peripheral neuropathy, most signs, symptoms and diagnostic testing reflect somatic nerve dysfunction. Somatic nerve
dysfunction may cause sensory (afferent) or motor (efferent) symptoms. The first step in evaluating peripheral neuropathy involves characterizing the patient’s symptoms as motor, sensory, autonomic or mixed to help identify which nerve fibers are involved in the patient’s disease. It is also necessary to rule out any upper motor neuron involvement.

Positive sensory symptoms are the most easily recognized by patients and frequently represent the patient’s chief complaint. These may include tingling, burning, pain and dysesthesias. The patient may also complain of sensory loss or numbness, usually beginning in the toes and feet, which may lead to imbalance, difficulty walking or a widening gait. The disease may progress to involve the fingers and hands, causing difficulty with fine motor movements and gripping objects. Positive motor symptoms, such as fasciculations, are less commonly recognized by the patient and should be specifically elicited in history-taking. Weakness will often not be present, especially early in the disease course, because collateral re-innervation may preserve motor function. These common sensory and motor symptoms are outlined in terms of positive and negative symptoms in Table 3. Autonomic

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Distribution</th>
<th>Differential Diagnosis</th>
<th>UMN Signs</th>
<th>Time Course</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory loss without weakness</td>
<td>Symmetric</td>
<td>Cryptogenic sensory polyneuropathy</td>
<td>Absent</td>
<td>Chronic, slowly progressive</td>
</tr>
<tr>
<td>+/- pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensory loss without weakness</td>
<td>Symmetric</td>
<td>Diabetes mellitus</td>
<td>Absent</td>
<td>Chronic, slowly progressive</td>
</tr>
<tr>
<td>+/- autonomic symptoms</td>
<td>Length-Dependent</td>
<td>HIV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensory loss without weakness</td>
<td>Symmetric</td>
<td>Vitamin B12 deficiency</td>
<td>UMN signs</td>
<td>Acute, Subacute</td>
</tr>
<tr>
<td>Sensory ataxia</td>
<td>Asymmetric</td>
<td>Copper deficiency</td>
<td>present</td>
<td></td>
</tr>
<tr>
<td>+/- autonomic symptoms</td>
<td></td>
<td>Friedrich’s ataxia</td>
<td></td>
<td>Chronic</td>
</tr>
<tr>
<td>Weakness</td>
<td>Symmetric</td>
<td>Sensory neuronopathy</td>
<td>Absent</td>
<td>Acute, Subacute or Chronic</td>
</tr>
<tr>
<td>Sensory loss</td>
<td>Proximal and distal</td>
<td>(ganglionopathy)</td>
<td></td>
<td>Autonomic</td>
</tr>
<tr>
<td>+/- pain</td>
<td></td>
<td>Sjogren’s syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cancer (paraneoplastic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weakness</td>
<td>Symmetric</td>
<td>Acute inflammatory demyelinating polyneuropathy (GBS)</td>
<td>Absent</td>
<td>Subacute or Chronic</td>
</tr>
<tr>
<td>Sensory loss</td>
<td>Distal</td>
<td>Chronic inflammatory demyelinating polyneuropathy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>+/- autonomic symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weakness</td>
<td>Symmetric</td>
<td>Hereditary motor and sensory neuropathies (Charcot-Marie-Tooth)</td>
<td>Absent</td>
<td>Chronic, begins in 1st two decades of life</td>
</tr>
<tr>
<td>Sensory loss</td>
<td>Distal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+/- pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weakness</td>
<td>Asymmetric</td>
<td>Vasculitis (mononeuritis multiplex)</td>
<td>Absent</td>
<td>Subacute, Painful</td>
</tr>
<tr>
<td>Sensory loss</td>
<td>Distal</td>
<td>HNPP</td>
<td></td>
<td>Chronic, Painless</td>
</tr>
<tr>
<td>+/- pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weakness</td>
<td>Asymmetric</td>
<td>Amyotrophic lateral sclerosis (ALS)</td>
<td>UMN signs</td>
<td>Chronic, weeks to months</td>
</tr>
<tr>
<td>without sensory loss</td>
<td>Distal</td>
<td>Multifocal motor neuropathy</td>
<td>present</td>
<td></td>
</tr>
<tr>
<td>Autonomic loss</td>
<td>Generalized</td>
<td>Amyloidosis</td>
<td>Absent</td>
<td>Chronic, progressive</td>
</tr>
<tr>
<td>+/- pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+/- weakness, sensory loss</td>
<td></td>
<td></td>
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</tbody>
</table>

UMN = upper motor neuron, EMG = electromyography, NCS = nerve conduction studies, GBS = Guillain-Barre syndrome, HNPP = hereditary neuropathy with liability to pressure palsies.
symptoms are often predominant in amyloidosis. These symptoms include orthostatic dizziness, dry eyes and dry mouth, gastrointestinal symptoms, secretomotor symptoms, and erectile dysfunction.\(^\text{10}\)

What is the distribution of symptoms?
The next step is to describe the distribution of the patient’s symptoms as symmetric or asymmetric and focal or generalized. Many patients with peripheral neuropathy present with symmetric, distal sensory loss in a “stocking and glove” distribution, but patients may have predominantly proximal symptoms, or a distribution that involves both proximal and distal limbs. Physical exam should include evaluation for a distal-to-proximal gradient by asking if light touch is perceived less strongly at a distal point compared with a proximal point\(^\text{6}\) as well as by comparing distal and proximal deep tendon reflexes. In many cases, patients may be able to identify a specific point below which their symptoms begin and above which they have no complaints, which may be confirmed on physical exam. The physical exam findings should also determine whether the patient’s symptoms occur in a dermatomal pattern or demonstrate radicular features.

What is the time course of the disease?
Next, describe the rate of progression of the patient’s symptoms. Acute neuropathies progress over days or weeks, and often have a recognized time of onset. Chronic neuropathies develop over months or years, and often have an insidious course, with the patient unable to identify the time of onset.\(^\text{6}\) The symptom course should also be described as progressive or relapsing.\(^\text{6}\) Hereditary neuropathies often have a long, slowly progressive course.\(^\text{2}\) A longstanding neuropathy should prompt evaluation for a hereditary cause. Pertinent exam findings include high arched feet or flat feet, curled or hammer toes, claw hands, muscle wasting and difficulty walking on heels or toes.\(^\text{6}\) Common patterns of involvement\(^\text{9}\) with sample differential diagnoses are summarized in Table 4.

What is the Underlying Pathology?
The symptom characterization detailed above provides a narrowed differential diagnosis. Identifying the underlying pathology of a patient’s peripheral neuropathy symptoms often involves laboratory and other diagnostic testing, as outlined in Figure 1, which provides an algorithm for the diagnostic workup of peripheral neuropathy. However, it is

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**Figure 1. Diagnostic Approach to Peripheral Neuropathy**

[Diagram showing diagnostic approach to peripheral neuropathy, including suspected peripheral neuropathy, order basic labs (CBC, CMP, FBG, TSH), refer to neurology, EMG/NCS, and differential diagnosis for axonal, demyelinating, and mixed neuropathies.]
important to start with some pertinent history: does the patient have any co-existing medical conditions, especially those listed in Table 1? Does the patient have any family history indicating risk for diseases causing neuropathy or evidence of hereditary neuropathies? Does the patient take any medications that might cause neuropathic symptoms or, although rare, have any recent toxin exposure?

When history, physical exam and initial laboratory evaluation are insufficient for diagnosis, the patient should be referred to a neurologist. The neurologist will likely perform electrodiagnostic testing (EMG = electromyography, NCS = nerve conduction studies) and these results will determine the need for additional laboratory or diagnostic evaluation as detailed in the diagram below.

**Laboratory Tests**
Laboratory tests may be helpful in evaluating a patient for diabetes, autoimmune disease, infection, nutritional deficiencies, toxins, hereditary conditions, and certain cancers. Testing should be targeted, focusing on ruling out specific causes that are suggested by the patient’s history and physical exam findings. According to a recent American Academy of Neurology practice parameter, the labs with the highest yields in peripheral neuropathy are blood glucose, serum B12 level with metabolites (methylmalonic acid with or without homocysteine), and serum protein immunofixation electrophoresis. Testing for impaired glucose tolerance may be considered if the suspicion for diabetic neuropathy is high but fasting blood glucose testing yields insufficient evidence of diabetes mellitus. Other labs that are commonly ordered based on clinical suspicion include complete blood count (CBC), comprehensive metabolic panel (CMP), thyroid stimulating hormone (TSH), anti-nuclear antibody (ANA), erythrocyte sedimentation rate (ESR) and paraneoplastic panel. HIV and hepatitis C testing should be obtained in high-risk patients. Lumbar puncture may also assist in identifying patients with inflammatory demyelinating polyneuropathies, characterized by a markedly elevated spinal fluid protein with minimal elevation in leukocytes, known as albuminocytologic dissociation.

**Electrodiagnostic Tests**
Electrodiagnostic tests include nerve conduction studies (NCS) and needle electromyography (EMG) as well as the less commonly performed repetitive nerve stimulation, blink reflex testing and late response testing. NCS and EMG evaluate large nerve fiber function to aid in classifying the underlying pathology as axonal damage, demyelination, conduction block or mixed processes. This testing may confirm peripheral nerve involvement, confirm the suspected diagnosis or suggest other possible etiologies. Small sensory and autonomic fibers are not assessed with routine NCS and EMG.

**Skin and Nerve Biopsy**
Small nerve fiber dysfunction can cause sensory or autonomic symptoms. A skin punch biopsy may be done in the presence of predominant pain or autonomic symptoms to measure the density of the epidermal small nerve fibers and suggest small nerve fiber involvement. A nerve biopsy may show vasculitis, abnormal deposits of substances such as amyloid, demyelination not evident on electrodiagnostic studies (atypical CIDP), or axonal degeneration and should be considered when there is clinical suspicion for these types of neuropathies.

**Autonomic Tests**
Orthostatic vitals and the tilt table test measure the change in blood pressure and pulse when the patient changes position from lying supine to standing vertically. Orthostatic hypotension is defined as a decrease in systolic blood pressure by 20 mmHg or in diastolic by 10 mmHg within three minutes of standing. These tests are positive when the patient experiences symptoms of dizziness or syncope in association with a drop in blood pressure.

The Quantitative Sudomotor Axon Reflex Test (QSART) measures the patient’s sweating in response to the release of acetylcholine from sympathetic postganglionic fibers. The thermoregulatory sweat test (TST) is a measure of regional sweat production in response to an elevation in body temperature under controlled conditions. If both the QSART and TST show absent sweating, a postganglionic lesion is present. If QSART is normal but TST shows absent sweating, there is a preganglionic lesion.

**Treatment**
Extensive evaluation of a patient’s peripheral neuropathy should aim to identify or rule out a treatable underlying cause. Common causes that are amenable to treatment include diabetes mellitus, metabolic or hormone abnormalities and vitamin deficiencies. Due to the limited regenerative ability of injured nerves, controlling or correcting these underlying imbalances will only stop or slow symptom progression and generally does not reverse nerve damage that is already present. Immune-mediated...
neuropathies, such as AIDP and CIDP, are usually very responsive to treatment with intravenous immunoglobulin, corticosteroids, plasmapheresis or monoclonal antibodies.\(^1\) Surgical intervention is indicated for patients with nerve damage from injury or compression. Dietary supplementation with alpha-lipoic acid may reduce symptoms and implanted neuromodulator devices (commonly in the spinal cord) may stimulate nerve function for some patients.

Other treatment options are directed toward alleviating symptoms. Physical therapy and mobility aids, such as a cane, walker or wheelchair, help preserve patient mobility and safety. Pharmacologic therapy commonly used for peripheral neuropathy usually does not modify the disease process, slow the progression of the illness or completely eradicate the patient’s pain.\(^7\) For patients with painful neuropathy, medications may improve quality of life and daytime functioning. Medications approved for treating neuropathic pain include anticonvulsants (gabapentin and pregabalin are first line); nonsteroidal anti-inflammatory drugs (NSAIDs) and tramadol for patients with sufficient renal function, and tricyclic antidepressants (TCAs) or duloxetine. Topical products including 5 percent lidocaine patch and capsaicin may be effective for focal pain.

**Conclusion**

After evaluation and characterization of the symptoms and physical exam findings in a patient with suspected peripheral neuropathy, a specific pattern should emerge that suggests a relatively narrow list of differential diagnoses. The pattern recognition approach allows the provider to target laboratory and diagnostic testing toward the most likely underlying causes for the patient’s symptoms in an effort to more efficiently diagnose reversible causes or diseases requiring urgent treatment. For as many as one-third of patients, a thorough evaluation will not reveal a definitive cause. Treatment should focus on correcting the underlying disease when possible to prevent progression of nerve damage. Other treatment should aim to alleviate pain and ensure patient safety.

**References**

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By Daniel Peterit, MD, FASTRO; Adam Omidpanah; Amy Boylan, RN; Patricia Kussman, LPN; Denise Baldwin; Deborah Banik, RN, PhD; Mary Minton, RN, PhD; Eric Eastmo, MD; Paul Clemments, MS; and B. Ashleigh Guadagnolo, MD, MPH

Abstract
The mastectomy rate in rural areas of the Northern Plains of the U.S. was 64 percent from 2000 through 2005. We implemented a breast cancer patient navigation (BPN) program in May 2007 to increase breast conservation (BC) rates.

Methods: We analyzed mastectomy and BC rates among our 1,466 patients with either ductal carcinoma in situ (DCIS) or stage I/II invasive breast cancer treated from 2000 through 2012. We used interrupted time series (ITS) to compare rates in treatment following implementation of BPN. In addition, breast conservation rates were compared to population data from the Surveillance, Epidemiology, and End Results (SEER) database.

Results: The BC rates were 56 percent for navigated patients versus 37 percent for non-navigated patients (95 percent CI for difference: 14.8 to 25.6 percent). There was a consistent annual increase in treatment with BC versus a mastectomy (+2.9 percent/year, p-trend < 0.001). The BC rate of 60 percent in 2012 now mirrors those observed in the SEER database. The ITS did not find that the change in BC rates over time was significantly attributable to implementation of the BPN. Other secular trends may have contributed to the change in BC rates over time.

Conclusions: A number of factors may have contributed to an increase of BC rates over time, including physician and patient education, more radiation therapy options, and possibly a dedicated breast cancer PN program. This analysis demonstrates that overall breast cancer care among this rural and medically-underserved population is improving in our region and now parallels other regions of the country.

Introduction
Randomized trials comparing mastectomy to breast conservation (BC) — lumpectomy and radiation therapy — have established the equivalency of both treatment options for appropriately selected, early-stage breast cancer patients. Over two decades ago, the National Institutes of Health (NIH) issued a consensus statement recommending BC as the preferred treatment approach for women presenting with early-stage breast cancer. Despite these endorsements, many women who are clinically eligible for BC treatment for breast cancer continue to undergo a mastectomy for a variety of reasons. The inverse relationship between BC rates and the geographic distance from the nearest cancer center is supported by the observation of lower rates of BC in rural areas. A 1992-93 Medicare survey reported only 1.4 percent of Medicare patients in our region underwent BC in western South Dakota. In addition, a recent analysis from our state, South Dakota, documented a mastectomy rate of 43.5 percent overall, with small rural areas reporting a 50 percent rate, and urban areas 43 percent.
Patient navigation (PN), an innovative care delivery concept for vulnerable populations, was created by Freeman and colleagues to mitigate observed high rates of late-stage presentations of breast cancer among the poor and minority populations of Harlem, New York. Once PN was implemented among these vulnerable populations, the five-year overall survival for breast cancer patients increased from 39 percent to 79 percent. While many PN programs have been developed to improve cancer screening rates, and to decrease the interval from cancer detection/cancer diagnosis and subsequent treatment, only recently have PN programs been developed for cancer patients undergoing treatment. Three randomized trials for navigated cancer patients recently reported improved cancer care experience with a nurse patient navigator.1-3

In 2002, we implemented a PN program, funded by the National Cancer Institute (NCI), as part of a cancer disparities outcomes improvement project to address the high cancer mortality rates experienced among Northern Plains American Indians (Al).14,15 Recent data from the Centers for Disease Control and Prevention (CDC) reported that breast cancer is the most frequently diagnosed cancer and leading cause of cancer mortality among American Indian/Alaska Native (AI/AN) women in the Northern Plains.16 Our PN program has identified a number of barriers to early cancer detection including trust, education, access to screening, and distance from the cancer center.17-20 In 2007, we implemented a dedicated breast PN program for all breast cancer patients, not just our American Indian patients, as we identified that all patients in this rural and medically-underserved community were lacking both access to education of breast cancer treatment options as well as support during cancer treatment.

Methods
Setting
Rapid City Regional Hospital (RCRH) is a community-based hospital which serves a geographic area which includes approximately 350,000 residents. This regional tertiary hospital provides health care services to three of the largest Indian reservations in the U.S. where an estimated 70,000 Al reside: Cheyenne River, Rosebud, and Pine Ridge reservations. Additionally, according to the Index of Medical Undersevice, western South Dakota, where our center is located, is designated medically underserved.21

In 1999, recruitment of new surgeons and radiation oncology staff, and infrastructure to this rural cancer center brought brachytherapy services and national cooperative group breast cancer clinical trial options to this region. This allowed availability of evidence-based brachytherapy services for breast cancer care. Brachytherapy services are a breast conservation treatment option that could potentially occur over days rather than the usual six weeks of radiation therapy needed for external beam radiation therapy regimens for breast cancer. This option altered the logistical and financial considerations for many patients in this region who often live geographically remote from the cancer center.

Intervention
RCRH developed a comprehensive breast cancer treatment patient navigation program in 2007. Beginning in May 2007, nearly all newly-diagnosed breast cancer patients presenting to our cancer center were referred to the breast patient navigator (BPN) after a positive biopsy for breast cancer or ductal carcinoma in situ (DCIS). The BPN arranged immediate consultations with surgery and radiation oncology, as well as medical oncology when appropriate. Patients were given educational materials and information on local therapy treatment options (e.g., surgery, adjuvant radiation therapy) to aid their decision-making process. The BPN continued to assist patients throughout treatment, and follow-up via telephone consultation or in-person assistance during a patient’s visit to the cancer center. The BPN helped coordinate appointments, contacted medical providers, and answered patient questions both during treatment and in follow-up.

Measures Collected
We divided our sample into two time eras: before and after the implementation of BPN in May 2007. We only considered patients with DCIS or Stage I/II cancers for analysis. Patient demographics were measured using a combination of medical chart abstraction and self-report. Geographic remoteness was measured using translated Rural Urban Commuting Area (RUCA) scores to urban, large rural, small rural and isolated classifications based on ZIP code of the patient’s primary residence at time of diagnosis. Patient race was self-reported. RUCA codes are a marker for rurality based on census tract information and are effective at representing access to care.22

Patients who received lumpectomy and/or radiotherapy were coded as having received BC therapy, and patients who underwent a mastectomy were counted as non-BC.
This treatment indicator was taken to be the outcome of interest for analyses.

To identify a national comparison group for breast cancer treatment rates, we used the publicly available Surveillance, Epidemiology, and End Results (SEER) data for comparison. The SEER program (a National Cancer Institute-supported database) contains tumor registries in 17 geographic areas covering approximately 25 percent of the U.S. population. We accessed breast cancer cases for women with SEER historic stage A (a composite staging variable in SEER that synthesizes changes in the staging system over time) for localized disease.

**Statistical Analysis**

We summarized the demographic characteristics of the cohort using means and proportions. We used Cochran-Armitage test of trend to determine whether there was a change in proportion of eligible women choosing BC rather than mastectomy over time. To assess whether these trends were substantially affected by implementation of BPN, we performed an interrupted time series (ITS) analysis for treatment modalities. These models were expanded to include factors associated with choice of treatment: cancer stage (DCIS or stage I/II), age, and RUCA score as continuous factors. Likelihood ratio tests were used to simultaneously test the statistical significance of either the change in trend and/or the instantaneous change in rate modeled by ITS. In addition, BC rates over time were compared to a population baseline in the publicly available SEER database. Institutional Review Board approval was obtained from our institution to conduct this study. All analyses were performed using R version 3.0.1.

**Results**

Table 1 lists characteristics of the 1,466 patients analyzed in this cohort stratified by era before and after implementation of our comprehensive BPN. The distribution of age, race, and rural versus urban residence were similar in the two treatment eras. We did observe a slightly longer time until initiation of treatment in the BPN era.

The BC rates were 56 percent versus 37 percent for navigated and non-navigated cohorts, respectively (95 percent CI for difference: 14.8 to 25.6 percent). Patients demonstrated a consistent and significant annual increase in treatment with BC versus a mastectomy (+2.9 percent/year, p-trend < 0.001).

Breast conservation rates over time are depicted in Figure 1. Using ITS analysis documented in Table 2, predictions from this model are presented as the solid line with linear trends depicted as the dashed line. Aggregate annual BC rates from our patient cohort are presented using points. There is a positive trend demonstrating an improvement in BC rates that continues to improve over the entire duration of the study period. The ITS analysis did not demonstrate that the change in BC rates over time were significantly attributable to implementation of the BPN using this model. These data suggest that other secular trends contributed to the change in BC rates over time.

Figure 2 demonstrates that in the later years of the study window, the BC rates became similar to those observed the SEER database which showed BC rates of 60 percent in 2012.
Discussion

In our study, among an under-served population of patients who previously experienced low rates of BC therapy for localized breast cancer – 22 percent in 2000 – we demonstrated a significant improvement in BC rates to 60 percent by 2012. This improvement in breast cancer outcomes corresponded to the initiation of a dedicated breast cancer patient navigation program. While our initial analysis suggested that implementation of a breast cancer treatment patient navigation program was associated with the reduction in mastectomy rates, more advanced adjusted modeling revealed that there were secular trends contributing to lower mastectomy rates for localized breast cancer. Reasons for improvements in breast conservation therapy rates over time were multifactorial. They likely included increased public awareness associated with national campaigns for breast cancer awareness that have grown over the past two decades. Furthermore, contemporarily-trained surgeons and radiation oncologists are more comfortable with breast conservation therapy, as

Table 1. Demographics of the Breast Cancer Navigation Cohort

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at diagnosis years; mean (sd)</td>
<td>60.9 (13.5)</td>
<td>60.8 (13.1)</td>
<td>0.89</td>
</tr>
<tr>
<td>RUCA at diagnosis; n (%)</td>
<td></td>
<td></td>
<td>0.07</td>
</tr>
<tr>
<td>Urban</td>
<td>451 (53.2)</td>
<td>357 (57.7)</td>
<td></td>
</tr>
<tr>
<td>Large rural</td>
<td>62 (7.3)</td>
<td>57 (9.2)</td>
<td></td>
</tr>
<tr>
<td>Small rural</td>
<td>155 (18.3)</td>
<td>89 (14.4)</td>
<td></td>
</tr>
<tr>
<td>Isolated</td>
<td>179 (21.1)</td>
<td>116 (18.7)</td>
<td></td>
</tr>
<tr>
<td>Time to initiation of treatment months; mean (sd)</td>
<td>0.7 (0.8)</td>
<td>0.9 (0.7)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>AJCC stage; n (%)</td>
<td></td>
<td></td>
<td>0.179</td>
</tr>
<tr>
<td>DCIS</td>
<td>108 (12.8)</td>
<td>95 (15.3)</td>
<td></td>
</tr>
<tr>
<td>Stage I/II</td>
<td>739 (87.2)</td>
<td>524 (84.7)</td>
<td></td>
</tr>
<tr>
<td>Treatment characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mastectomy; n (%)</td>
<td></td>
<td></td>
<td>0.001</td>
</tr>
<tr>
<td>Lumpectomy; n (%)</td>
<td></td>
<td></td>
<td>0.001</td>
</tr>
<tr>
<td>Radiotherapy; n (%)</td>
<td></td>
<td></td>
<td>0.001</td>
</tr>
<tr>
<td>Radiotherapy type; n (%)</td>
<td></td>
<td></td>
<td>0.104</td>
</tr>
<tr>
<td>EBRT</td>
<td>281 (88.1)</td>
<td>269 (83.3)</td>
<td></td>
</tr>
<tr>
<td>Brachytherapy</td>
<td>38 (11.9)</td>
<td>54 (16.7)</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Interrupted Time Series Unadjusted and Adjusted Model Results

<table>
<thead>
<tr>
<th>Unadjusted</th>
<th>Mastectomy</th>
<th></th>
<th>Lumpectomy</th>
<th></th>
<th>Radiotherapy</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Effect</td>
<td>95% CI</td>
<td>Effect</td>
<td>95% CI</td>
<td>Effect</td>
<td>95% CI</td>
<td></td>
</tr>
<tr>
<td>Time trend</td>
<td>-0.04 (-0.05, -0.02)</td>
<td>0.04 (0.02, 0.05)</td>
<td>0.04 (0.02, 0.05)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Program</td>
<td>0.18 (-0.09, 0.45)</td>
<td>-0.20 (-0.47, 0.07)</td>
<td>-0.10 (-0.37, 0.17)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time after</td>
<td>-0.01 (-0.04, 0.02)</td>
<td>0.02 (-0.02, 0.05)</td>
<td>0.00 (-0.03, 0.03)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>overall p ITS1</td>
<td>0.250</td>
<td>0.245</td>
<td>0.176</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Adjusted

| Time trend | -0.04 (-0.05, -0.02) | 0.04 (0.02, 0.05) | 0.03 (0.02, 0.05) |
| Program | 0.16 (-0.11, 0.43) | -0.17 (-0.44, 0.10) | -0.15 (-0.42, 0.13) |
| Time after | 0.00 (-0.01, 0.01) | 0.00 (-0.01, 0.01) | 0.01 (-0.03, 0.04) |
| Age | 0.00 (0.00, 0.01) | 0.00 (-0.01, 0.00) | -0.01 (-0.01, 0.00) |
| AI race | 0.11 (0.01, 0.20) | -0.11 (-0.20, -0.10) | -0.11 (-0.21, -0.02) |
| RUCA | -0.16 (-0.23, -0.09) | 0.15 (0.08, 0.23) | -0.16 (-0.23, -0.09) |
| DCIS | -0.01 (-0.04, 0.02) | 0.01 (-0.02, 0.04) | -0.01 (-0.04, 0.02) |
| overall p ITS1 | 0.247 | 0.250 | 0.173 |

1 p-value calculated from likelihood ratio test fitting a reduced model in which the program effect and its interaction with time are omitted.
well as introduction of more convenient adjuvant radiation therapy options also likely contributed. For example, the brachytherapy techniques implemented in 2002, allowed possibility shortening the recommended radiation therapy course from four to six weeks of external beam radiation to one week of breast brachytherapy.

Patient navigation programs established and published to date have demonstrated improved cancer screening rates with earlier-stage detection of cancer, lower breast cancer mortality rates, and enhanced patient experience during cancer treatment. Current PN programs for cancer patients have dual missions as recently discussed by Hendren: to assist patient coordination with the complexities of cancer treatment, and to optimize patient-reported outcomes. Our study suggests an association with increased uptake of guideline concordant cancer treatment, as breast conservation therapy rather than mastectomy for eligible patients is considered a quality of care measure by the American College of Surgeons.

Our BPN program originated from a previously established patient navigation program that was established in 2002 as part of a NCI-funded cancer disparity program. Our breast cancer patient navigator facilitated immediate referrals to both surgery and radiation oncology after a diagnosis of breast cancer so as to enhance patient education about breast cancer treatment options. Our breast conservation therapy rates now mirror the population baseline data available in SEER. This represents improvements in care for this community whose breast conservation rates previously fell well below the observed SEER breast conservation therapy rates. This suggests our comprehensive approach of improving treatment options in combination with a targeted patient navigation effort to enhance patient education and treatment choice agency effectively improved overall breast cancer care in this community cancer center.

Nationally, there is an emerging trend toward higher utilization of mastectomy, including contralateral prophylactic mastectomies. This is attributable to a number of factors: patient anxiety with retaining the breast(s), use of MRIs that have led to false positive findings of breast pathology, and appropriate genetic concerns with BRCA 1 and 2 results. A recent study by Farhenwald, et al. examined the differences in treatment decision-making for breast conservation therapy rates for women in both urban and rural South Dakota. Breast conservation rates were 57 percent for urban women and 50 percent for rural women. In this study, the three most important factors for these women in choosing breast conservation was the surgeon’s opinion, personal choice, and fear of recurrence. The implementation of a dedicated breast PN, and a genetic counselor, may further assist patients with these complex treatment decisions.

The geographic distance from the cancer center may influence patients to choose a mastectomy, even if they are appropriate candidates for breast conservation. As part of our cancer disparities research program initiated in 2002, we began offering breast brachytherapy as part of a phase II clinical trial in order to mitigate the effect of distance to travel for treatment, as many of our patients live 100 to 150 miles from the cancer center. This program has been successfully implemented with 70 patients treated and recurrence rates similar to that observed for whole breast radiation (less than 5 percent) and minimal toxicities (D. Peteriet, personal communication, May 2015.) During our breast PN era, 17 percent of early-stage breast cancer patients underwent breast brachytherapy. Presley, et al. recently published a report analyzing national breast brachytherapy practice patterns in the US. The majority of these programs were located in high population density regions. However, western South Dakota where our center is located, was a high-use area, and one of the few lower population density regions where this treatment modality was implemented in order to meet the needs of our community.

In 2002, we postulated that a comprehensive, multi-faceted approach to lowering overall cancer mortality rates, including breast cancer mortality, for Northern Plains American Indians, could be effective. Our approach in this rural, medically-underserved population consisted of a comprehensive patient navigation program and increased availability of technological advancements that reduce the overall radiation treatment time (i.e., breast brachytherapy as discussed above). In prior publications we have reported the following positive outcomes: higher patient satisfaction rates, improved patient compliance with less treatment interruptions, and higher clinical trial accrual rates. Recent analyses suggests that AI cancer patients with screen detectable cancers are now presenting with earlier stages of disease and higher cure rates. (Peteriet, personal communication, May 2015.)

Our model of a dedicated breast cancer treatment navigation program may have value not only for rural patients regardless of race as we have shown here, but also for other...
disease sites where complex treatment decisions for equivalent treatment options exist. Another site for which this may be particularly true is prostate cancer, where multiple treatment options exist for early-stage disease. Furthermore, treatment-based navigation may offer better patient outcomes in other complex oncology treatment regimens for other cancer sites, especially where combined modality therapy is the standard of care that often is associated with increased treatment toxicities. Further research is needed to investigate the role of patient navigation during cancer treatment as a delivery innovation that might improve care quality, reduce unnecessary care utilization, and improve patient outcomes.

In conclusion, our model of comprehensive, multi-faceted approach to addressing documented cancer-related health disparities in a rural and under-served population has been effective in improving breast cancer outcomes in a patient with previously high mastectomy rates for localized breast cancer. Our analyses show that disparate cancer-related outcomes in these communities can be overcome, but it likely requires a multi-faceted health services approach to affect improvements in outcomes. Resources and efforts are required to facilitate diffusion of technological, psycho-social, and decision-support advancements in cancer care to underserved communities.

This study was funded by NIH grant 5 U56 CA09010.

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18. Guadagnolo AG, Mollot K, Cina K, Helbig P, Reiner M, Peterit DG. Trust and satisfac-

Please note: Due to limited space, we are unable to list all references. You may contact South Dakota Medicine at 605.336.1955 for a complete listing.

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Delirium is a common neuropsychiatric syndrome that occurs in hospitalized patients, and has a propensity to impact the elderly, those with underlying cognitive impairment, and patients with a high severity of illness. It typically presents as an acute confusional state that is characterized by disturbed mentation or perception, and results in alterations in attention, disorganized thinking, disturbed psychomotor activity, an abnormal sleep-wake cycle and an altered level of consciousness. It typically develops over a period of hours to days, and also tends to fluctuate throughout its course. Delirium has been found to occur in up to half of hospitalized elderly patients and hip surgery patients, and up to 80 percent of mechanically ventilated ICU patients. Even though the development of delirium is a common occurrence in hospitalized patients, its pathogenesis is still poorly understood. Overall, it is felt to be precipitated by a complex interaction of factors including: predisposing patient characteristics (e.g., cognitive impairment, hypertension, advanced age, underlying alcohol or drug dependence), features of the acute illness (e.g., level of acuity; presence of hypotension, sepsis, or pain), and factors related to treatment or the hospital environment (e.g., medications, sleep deprivation, isolation, mechanical ventilation, immobility). It has also been postulated that disturbances in the sleep-wake cycle and alternations in melatonin levels might play a role in its development. Factors such as advancing age, surgery, and sleep deprivation have all been associated with lower melatonin levels and higher rates of delirium.

Aside from being severely distressing for patients and their family members, delirium also has a tremendous detrimental impact upon the health outcomes of patients. The development of delirium has been associated with increased morbidity, mortality, length and cost of hospital stay, likelihood of post-hospital institutionalization, and long-term cognitive impairment. Given the severe consequences of delirium, effective strategies are needed to prevent it from occurring in vulnerable patients.

Although several pharmacologic approaches (e.g., antipsychotics, cholinesterase inhibitors) have been tried to prevent delirium in hospitalized patients, the most recent clinical practice guidelines from the American College of Critical Care Medicine (ACCM), the Society of Critical Care Medicine (SCCM), and the American Society of Health-System Pharmacists (ASHP) provide no recommendations for pharmacologic prevention due to lack of compelling evidence and potential for patient harm. Instead, it is generally recommended to focus preventative efforts on non-pharmacologic alternatives for patients who are deemed high risk. (Table 1)

### Table 1: Non-pharmacologic approaches to prevent delirium

<table>
<thead>
<tr>
<th>Approach</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequent, scheduled reorientation</td>
<td></td>
</tr>
<tr>
<td>Cognitive stimulation (e.g., regular family/friend visits)</td>
<td></td>
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<tr>
<td>Sleep promotion (e.g., avoidance of nighttime interruptions and noise)</td>
<td></td>
</tr>
<tr>
<td>Early mobilization and minimal use of physical restraints</td>
<td></td>
</tr>
<tr>
<td>Provision of vision and hearing aids when needed</td>
<td></td>
</tr>
<tr>
<td>Avoidance/minimization of problematic medications (e.g., benzodiazepines, opioids, dihydropyridines, antihistamines)</td>
<td></td>
</tr>
<tr>
<td>Effective pain management and assessment</td>
<td></td>
</tr>
</tbody>
</table>

Since one of the cardinal features of delirium is a disruption of the sleep-wake cycle, melatonin supplementation has been studied as a possible preventative strategy. Melatonin is an endogenous hormone that is secreted by the pineal gland of the brain during times of darkness; it plays a pivotal role in the regulation of the circadian rhythm and the sleep-wake cycle. Because of this, melatonin is a popular dietary supplement that is taken by over 1 million Americans as a natural sleep aid, and for a variety of other less studied indications, with typical doses ranging from 0.5 to 6 mg. Although melatonin alterations have been postulated to be associated with the development of delirium in hospitalized elderly patients, the data regarding supplementation to prevent this disorder is sparse.

Two melatonin studies were the first controlled trials to find a pharmacologic intervention that was effective in preventing the onset of delirium in hospitalized elderly patients. The first trial was a randomized, double-blinded,
controlled trial done in Egypt that evaluated 300 patients, age 65 and older, undergoing hip arthroplasty. These patients were randomized to four different premedication groups: no premedication, oral melatonin 5 mg, oral midazolam 7.5 mg, or oral clonidine 100 mcg. Premedications were given the night before surgery and again 90 minutes prior to surgery. The number of patients who developed delirium by the third post-op day was significantly lower in the melatonin group (9.43 percent) compared to no premedication (32.65 percent), midazolam (44 percent), or clonidine (37.25 percent), p < 0.05. Melatonin was well tolerated in this study, and no adverse effects were reported.

In the second trial, Al-Aama and colleagues performed a randomized, double-blinded, placebo controlled study of 145 Canadian patients, age 65 and older, who were admitted to a medical floor of the hospital for an acute illness. Patients were randomized to low dose melatonin, 0.5mg to approximate physiological levels, verses placebo every night for 14 days, or until discharge. In this study, melatonin was again associated with a lower incidence of delirium compared to placebo (12 percent vs. 31 percent, p = 0.014). Similar results were seen when patients with preexisting delirium were excluded (3.6 percent vs. 19.2 percent, p < 0.02). No differences were noted in the incidence of sleep disturbances or subjective sleep quality. Melatonin was well tolerated; one patient reported nightmares and it was discontinued. Overall, both of these studies showed that melatonin supplementation effectively decreased the incidence of delirium in elderly surgical/medical inpatients, and was also well tolerated.

More recently, emerging evidence from Japan has suggested that the prescription drug ramelteon, a melatonin agonist, has a beneficial effect in the prevention of delirium. A randomized, rater-blinded, placebo-controlled trial showed a significant prophylactic effect on the incidence of delirium in 67 medical ICU patients, age 65 to 89. In this study, ramelteon 8 mg (the approved dose for insomnia) for seven days was associated with a significantly lower risk of delirium compared to placebo (3 percent vs. 32 percent, p = 0.003). Once again, this beneficial effect occurred despite a lack of effect on observed sleep parameters. No adverse events related to the drug were reported.

It is important to note, however, that not all studies have demonstrated efficacy. In a double-blind, randomized trial of 452 elderly patients undergoing hip surgery in the Netherlands, patients receiving melatonin 3 mg every evening for 5 days experienced similar rates of delirium compared to those receiving placebo (29.6 percent vs. 25.5 percent, p = 0.4). There are a number of potential reasons for these inconsistent results. First of all, these studies were performed in various countries throughout the world, which could introduce pharmacogenetic variability in patients, as well as pharmacologic differences in the actual products used. In the US, melatonin is only available as a dietary supplement, thus it is not subject to FDA regulation. As a result, products can vary significantly in their strength and purity. The prescription drug ramelteon overcomes the issue of dosage standardization, but at a cost of over $12 per tablet compared to less than a nickel for a dose of melatonin. And finally, there is very little data available regarding the optimal dose for the prevention of delirium. Although the physiologic dose of melatonin is considered to be 0.3 mg, a typical dose of 3 mg can result in serum levels that are up to 20 times higher than normal. Some researchers have suggested that lower doses that produce serum concentrations closer to physiologic levels may actually be more effective.

In conclusion, melatonin is a popular dietary supplement that has shown promise in the prevention of delirium in high risk patients. These trials suggest a potential role for its short-term use in the prevention of delirium in elderly medical/surgical patients. It is currently unclear what the optimal dose is, and whether these beneficial effects are related to melatonin’s sleep altering effects. Although the studies were relatively small in size and the results were not entirely consistent, melatonin’s low cost and favorable side effect profile make it an attractive option to consider, in combination with non-pharmacologic interventions, for the prevention of this potentially devastating complication. Further studies are needed to determine the optimal dosage and product formulation.

REFERENCES

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By E. Paul Amundson, MD
Chief Medical Officer

If you recall columns from this section last year, which were heavily focused on QUALITY of health care, I made a statement promising a similar series of articles in 2016 focused on the other part of the VALUE equation in our industry: COST. This month we’ll focus on generalities, and then dive into more specifics on various specific topics/conditions over the next few months. My intent here is to educate, as I’ve heard frustrations and alarm from other physicians at the cost (not the value) of select goods and services associated with our profession, thus my interest in expanding on this topic at this time. Our colleagues representing DAKOTACARE’s Quality and Pharmacy & Therapeutics Committees have seen firsthand examples, such as new entries into the high-dollar specialty pharmacy market and expensive tests. Also, of prime importance to those of you in more direct patient care responsibilities, as patients face increased exposure to health care costs they require meaningful and transparent information regarding the cost of services they will incur.

To be effective, price/cost transparency must offer clear and reliable information that is readily accessible to patients and enables them to make meaningful comparisons among providers. To accomplish this goal, we’re going to need to remove certain historic barriers and collaborate between providers (you), payers (us), and employers to develop tools which are useful and actionable by patients. This is certainly a “work in progress” within this company, but please know we’re working diligently within our resource capacity to ensure the following elements will be included:

- Total estimated price of the service
- Accurate and up-to-date provider locator feature
- Clear description to the patient/member of their out-of-pocket responsibility
- Mutually agreed upon objective clinical quality measures

I may sound like a broken record, as I know I have advocated for increased transparency of health care information in this publication by referencing Johns Hopkins noted surgeon Dr. Marti Makary’s text “Unaccountable.” Similarly, I recently came across a more technical, but nonetheless interesting, treatise on this subject from the Healthcare Financial Management Association. This body formed a task force composed of representatives from provider, payer, and other vested interests to approach this subject from a more objective, financial viewpoint. Their findings mirror those I have seen on this subject from strictly clinical grounds, therefore would like to share some of their findings for your benefit.

- Price transparency should empower patients and other care purchasers to make meaningful price comparisons prior to receiving care.
- Any form of price transparency should be easy to use and easy to communicate to stakeholders.
- Price transparency information should be paired with other information (e.g., quality…sound familiar?) that defines the true value of services for the purchaser of care.
- Price transparency should provide patients the information they need to understand the total price of their care and what is included in that price.
- Price transparency will require the commitment and active participation of all stakeholders.

DAKOTACARE feels that for our fully-insured (HMO and Individual plans) members, we should be the owners of this process and principal source of pricing information. We are currently evaluating various platforms to identify the one best suited for our members and our market. You may not be aware, but many of our clients are self-funded. These employers are encouraged to use and expand transparency tools which assist to shape benefit design and help people understand their health care spending and/or benefits. For those patients who are uninsured or seeking care from a provider on an out-of-network basis, the provider should be tasked with providing that information.

As you make referrals, please keep in mind your responsibility to assist your increasingly price-sensitive patients in making an informed decision. They not only are assuming greater financial responsibility for their health care costs, but are increasingly savvy shoppers of these services. You should anticipate questions to your evaluation and treatment recommendations, including which providers you recommend for referral, such as “who offers the best price at the patient’s desired level of quality?” It’s a new world out there, quickly changing but filled with opportunities. DAKOTACARE wants to partner with you in developing physician and patient-friendly processes and I promise we will keep you updated on our progress.

Next month we’ll discuss DIABETES and inherent cost drivers for this increasingly common chronic condition. Now get outside and enjoy your summer!
Quality Focus:
Improving Cardiac Health and Reducing Cardiac Healthcare Disparities

By Stephan D. Schroeder, MD, Medical Director, South Dakota Foundation for Medical Care

South Dakota Foundation for Medical Care and the Great Plains Quality Innovation Network (QIN) have as one of their projects a goal of implementing evidence-based medicine to improve cardiovascular health and support the Million Hearts campaign. Million Hearts is an initiative aimed at reducing myocardial infarction and stroke. It is the Center for Disease Control (CDC) and Centers for Medicare & Medicaid Services (CMS) working in partnership with federal, state, and private organizations to prevent one million heart attacks and strokes by 2017. In addition, the project will focus on cardiac disparities relating to ethnicity, health literacy, language, culture, as well as race and gender. Alignment with home health agencies will help provide information for data registries and assist with beneficiary and family engagement.

The project will strive to facilitate the use of new and/or existing best practices, resources, and protocols. Boosting EHR reporting on cardiac quality measures and translating quality data into strategy for improvement will be some of the goals of this activity. Great Plains QIN will provide technical assistance as well as Learning and Action Networks (LAN), webinars, and site visits. Home health agencies, physician offices, and clinics, including those reporting through the Physician Quality Reporting System (PQRS), will be recruited. Patients and families will be offered screenings, health fairs, and LAN participation to help improve outcomes.

Million Hearts® (millionhearts.hhs.gov) hopes to reduce the 1.5 million cardiac events and 800,000 deaths from heart disease that occur each year in our country. In addition to reducing the annual cost of over $300 billion for this disease, it will strive to decrease the number of smokers by 6 million and have 10 million more patients on medication to help improve these staggering costs. Million Hearts will support the use of the ABCS: Aspirin therapy as appropriate, Blood pressure control, Cholesterol management, and Smoking cessation.

One in three Americans have some degree of hypertension, and of those there is a concern that only half of them have a good control rate. That group also includes those with insurance coverage and a primary care provider. Developing protocols or care pathways will help ensure consistent care and reduce disparate outcomes, especially in the relatively young and otherwise healthy patients in this element of the population. The Million Hearts website referenced above has protocol templates to help achieve the value and benefits of improved blood pressure control.

Home health agencies afford an option and an opportunity to reach patients in both urban and rural areas. Home Health Quality Improvement (homehealthquality.org) is a national campaign tool dedicated to improving the care of these patients. Factors such as cultural diversity as well as distance from providers present a significant challenge. Hopefully home health agencies can help promote patient engagement with support and education. This will help improve overall health care in conjunction with primary care providers. Social determinates can contribute to poorer health and outcomes. Lifestyle changes are a crucial element for improvement in cardiovascular disease. Please contact me at Stephan.schroeder@area-a.hcqis.org if you would desire further information about this project.

“Quality Focus” is a monthly feature sponsored by SDFMC, South Dakota’s Quality Improvement Organization. For more information about the SDFMC, visit their website at www.sdfmc.org.
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The American Medical Association Economic Impact Study, completed in conjunction with the South Dakota State Medical Association, shows how much physicians add to the economic health of South Dakota.

Check the effect physicians have on the U.S. economy by viewing the national report from the AMA, as well as highlights from the South Dakota study, at ama-assn.org/go/eis.

AMERICAN MEDICAL ASSOCIATION

SOUTH DAKOTA STATE MEDICAL ASSOCIATION

Please activate your 2016 AMA membership through the South Dakota State Medical Association by calling (605) 336-1965.
Baby boomers are coming-of-age – retirement age, that is; and many are wondering what's going to happen.

Starting about nine months after soldiers returned home from World War II, there was a birth rate boom coming almost as celebration of soldier survival, and this has profoundly affected our culture. Baby boomers, and I'm one of them, are those born from 1946 to 1964 during a successful post-war economy. This very large group of kids were the most privileged of all time.

Often raised with a soldier-disciplined father and a not-always-happy work-at-home mother, we grew up watching too much TV, listening on transistor radios to Beach Boys and Beatles, and facing a new and real threat of atomic warfare. Still, life was quite idyllic for boomers during those first 18 years.

But after graduating from Easy & Perfect Boomer High, we were slapped into reality by the draft, Vietnam, and a newly realized inequality happening between sexes and races. This generation often labeled as one of sex, drugs, and rock-n-roll, grew into an angry government-distrusting counter-culture that fought for social justice, for causes like feminism, civil rights, and the Vietnam War. These all reflect the boomer ethic: the right of self-direction.

It is no surprise this group has also achieved the most wealth. Accounting for 24 percent of the total U.S. population, boomers hold onto something like 80 percent of our country's wealth, account for 80 percent of all leisure travel, and dole out 50 percent of consumer spending. Seventy-six million boomers in the U.S. are a huge cultural demographic.

Like a pig swallowed by a python, the big bump is moving through the snake and is now coming to retirement age. As we become very elderly, sick, and disabled, there will be a smaller cadre of youth to care for us. Will we become a huge societal burden?

First of all, the elderly are usually not that burdensome. Research finds 75 percent of those aged 85 or older still drive, 70 percent are not depressed, 60 percent do not experience significant memory loss, and on average the elderly are as happy as any age group. When boomers turn 85, although potentially a demanding group, I predict we will find the resources to care for those who need it, and some might still be active enough to even physically help.

More importantly, most boomers will have an advanced directive, bypassing futile efforts and reducing the risk for a prolonged, expensive, and painful death.

And, oh, in these next years as boomers have more time on their hands, expect intolerance to social injustice, and an increasing demand for the privilege of self-direction.
Legal Brief Highlight: Licensure

The South Dakota Board of Medical and Osteopathic Examiners has jurisdiction over the licensure and discipline of doctors of medicine and doctors of osteopathy. Except as otherwise provided by federal law, any person desiring to practice medicine or osteopathy in South Dakota must apply to SDBMOE for the appropriate license or permit. The Board is authorized by law to issue four types of licenses or permits:

1. **A license to practice medicine or osteopathic medicine, surgery, and obstetrics in all of their branches without limitations** – grants the privilege to practice medicine or osteopathic medicine, surgery, and obstetrics, in all their branches and without limitations. However, the license must state on its face whether it is granted to practice medicine and surgery or osteopathic medicine and surgery.

2. **A temporary permit** – may be granted by the SDBMOE if it appears that an urgent need exists in any state-owned and operated medical institute for the services of a practitioner of medicine, surgery, and obstetrics and their branches.

3. **A locum tenens certificate** – allows the practice of medicine in South Dakota for a time period not exceeding 60 days to an applicant who is a current holder of a valid license to practice medicine or osteopathy in any state or territory of the U.S., the District of Columbia, or province of Canada, or who has graduated and received a diploma from an approved medical or osteopathic college and who has completed at least one year of an approved internship or residency program or its equivalent.

4. **A resident certificate** – issued to an applicant currently enrolled in an accredited residency program. A resident certificate is valid for one year from the issue date.

For more information, download the SDSMA legal brief *Licensure* at www.sdsma.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 and programs for members in the area of practice management, leadership and health and wellness.

*Source: SDSMA staff*
Member Updates Needed for 2017 Directory

In order for the SDSMA office to provide members with timely information, it is important members regularly review their current contact information on file with the SDSMA. Is your email correct? Have you changed employers? Is your mail going to the right place?

Please take a few minutes to review your profile information on the SDSMA website and make any necessary updates.

- Visit sdsma.org;
- Select Member Login at the top right-hand corner;
- Enter your Username and Password;
- If you do not remember your login information, select “Forgot my username” or “Forgot my password” to select a new one.
  - Enter your SDSMA-preferred email where you receive SDSMA communications.
  - All members have an existing account. Do not create a new user account.
- Once logged in, go to Update My Profile at the top of the page;
- Review the information on each of the tabs. This is the information the SDSMA has on file for you regarding addresses for work and home, email(s), and phone number(s);
- Clicking the ‘pencil’ on each tab, you can update the information, if needed.
- Do you have a new photo? Updated photos can be uploaded to your user account as well or emailed to membership@sdsma.org.

The information listed in your profile is what the SDSMA uses for contacting you by mail, email, or phone, and this information is listed in our annual Member Directory.

Any questions about updating your information, or assistance is needed accessing your account, can be directed to membership@sdsma.org or by calling Member Services at 605.336.1965.

Source: SDSMA staff

H. Thomas Hermann, Jr., MD, Sworn in as SDSMA President

H. Thomas Hermann, Jr., MD, became the 135th president of the SDSMA on June 3. Dr. Hermann has been a member of the SDSMA since 1986.

Dr. Hermann is a family medicine physician at Massa Berry Regional Medical Clinic in Sturgis. He is also a clinical professor of family medicine at the University of South Dakota (USD) Sanford School of Medicine and is a fellow in the American Academy of Family Practice.

Dr. Hermann has been involved in the leadership of organized medicine for several years. Prior to becoming SDSMA president, he held offices within the SDSMA’s Executive Committee and served as a councilor for the Black Hills District Medical Society. He also served as the SDSMA Foundation Board chair. Dr. Hermann has also volunteered for the SDSMA’s advocacy efforts during state legislative sessions by participating in the SDSMA Doctor of the Day program.

Dr. Hermann received his medical degree from USD School of Medicine in 1984 and completed his residency in 1987 at Sioux Falls Family Practice. Dr. Hermann and his wife Terry have a son Joel and a daughter Molly.
Confronting a Crisis: An Open Letter to America’s Physicians on the Opioid Epidemic

By Steven J. Stack, MD
Emergency Physician and the 170th President of the American Medical Association

The medical profession must play a lead role in reversing the opioid epidemic that, far too often, has started from a prescription pad.

For the past 20 years, public policies – well-intended but now known to be flawed – compelled doctors to treat pain more aggressively for the comfort of our patients. But today’s crisis plainly tells us we must be much more cautious with how we prescribe opioids.

At present, nearly 2 million Americans – people across the economic spectrum, in small towns and big cities – suffer from an opioid use disorder. As a result, tens of thousands of Americans are dying every year and more still will die because of a tragic resurgence in the use of heroin.

As a profession that places patient well-being as our highest priority, we must accept responsibility to re-examine prescribing practices. We must begin by preventing our patients from becoming addicted to opioids in the first place. We must work with federal and private health insurers to enable access to multi-disciplinary treatment programs for patients with pain and expand access for medication-assisted treatment for those with opioid use disorders. We must do these things with compassion and attention to the needs of our patients despite conflicting public policies that continue to assert unreasonable expectations for pain control.

As a practicing emergency physician and AMA president, I call on all physicians to take the following steps – immediately – to reverse the nation’s opioid overdose and death epidemic:

• Avoid initiating opioids for new patients with chronic non-cancer pain unless the expected benefits are anticipated to outweigh the risks. Non-pharmacologic therapy and non-opioid pharmacologic therapy are preferred.
• Limit the amount of opioids prescribed for post-operative care and acutely-injured patients. Physicians should prescribe the lowest effective dose for the shortest possible duration for pain severe enough to require opioids, being careful not to prescribe merely for the possible convenience of prescriber or patient. Physician professional judgment and discretion is important in this determination.
• Register for and use your state prescription drug monitoring program (PDMP) to assist in the care of patients when considering the use of any controlled substances.
• Reduce stigma to enable effective and compassionate care.
• Work compassionately to reduce opioid exposure in patients who are already on chronic opioid therapy when risks exceed benefits.
• Identify and assist patients with opioid use disorder in obtaining evidence-based treatment.
• Co-prescribe naloxone to patients who are at risk for overdose.

As physicians, we are on the front lines of an opioid epidemic that is crippling communities across the country. We must accept and embrace our professional responsibility to treat our patients’ pain without worsening the current crisis. These are actions we must take as physicians individually and collectively to do our part to end this epidemic.

Together we can make a difference.
## CME Events

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

### CME Events

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

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

### Don’t forget to send in your favorite scenic photo for South Dakota Medicine front cover consideration.

Send photos to ereiss@sdsma.org.
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