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STUCK IN TRAFFIC
ROSS POLKING, CFP®, AIF®, MBA, Lead Advisor

We’ve all been there. Stuck in traffic. Blood pressure rising, four lanes apparently nowhere near enough to efficiently funnel all the vehicles to their intended destination. You cannot fathom what in the world is causing this crawl of a pace. In a desperate attempt to expedite your trip, you quickly weave into the paralleling lane that appears to be moving faster than yours. Five seconds later, you are hitting the brakes and coming to a standstill while – you guessed it – the lane you just left has now picked up the pace. You, of course, would have been better off staying in your original lane. How’s your blood pressure? Probably elevated just from reading this.

Getting from one place to the next requires some degree of patience and, certainly, a plan. Trying to guess which lane is the fastest often leads to undesirable results with significantly elevated risks, namely, accidents, tickets, and maybe a twinge of road rage. Investing is much like this traffic scenario. Drivers and investors both tend to make small and inefficient moves from one lane or investment to the next. After a short period of time, if one is not meeting our expectations, we quickly try to switch to another option. Ironically, most everyone else is trying to do exactly the same thing you are … gain an advantage … at significantly increased risk and cost.

In the end, more times than not, we do not arrive at our destination nor realize our goals as quickly as we would have had we just stayed the course. What’s more, it is unquestionably less dangerous and less stressful to drive and invest this way.

The message is simple – stick to your lane, stay diversified, and enjoy the ride!

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By Robert E. Van Demark, Jr., MD
SDSMA President

Gearing up for the Legislative Session

As I write this month’s article, the 2018 Legislative Session is about to start and the SDSMA staff are preparing and advocating on behalf of physicians and our patients. Session opens on Jan. 9 and continues through March 26. The Legislature is the state’s lawmaking body. It is a bicameral legislative body, consisting of the Senate, which has 35 members, and the House of Representatives, which has 70. The two legislative chambers are similar in most respects; however, the Senate holds the authority to confirm gubernatorial appointments. The Legislature's primary function is to enact laws to provide for the health, welfare, education, environment, and economic and general well-being of the citizens of South Dakota. It also establishes public policy through the passage of bills and resolutions and proposes amendments to the state constitution, which are then submitted to the voters for approval or disapproval. State senators and representatives are elected from the state’s 35 districts every even-numbered year to serve a two-year term. Since 1993, legislators have been limited to serving four consecutive terms in a legislative chamber.

On behalf of our physician, resident and medical student members, and our patients across the state, the SDSMA advocates to improve the health and well-being of the public and our patients, and create the best environment for our physicians to advance the delivery of quality health care. The SDSMA has developed a roadmap on state and federal advocacy initiatives for 2018 and beyond; you can review the SDSMA’s legislative advocacy agenda at www.sdsma.org. The agenda articulates specifically and directly what we are asking of the South Dakota Legislature, the U.S. Congress, and state and federal regulators. The recommendations range from the simple – directly what we are asking of the South Dakota Legislature, the U.S. Congress, and state and federal regulators. The recommendations range from the simple – increasing the purchase age for tobacco products to 21 years of age – to the complex – providing health care to low-income South Dakotans while ensuring realistic payment to physicians, to the most fundamental – ensuring protection of the patient-physician relationship.

To support the efforts of our SDSMA, we also need you to become politically active. As physicians, we have a natural role as patient/health care advocate, and quite often advocate on behalf of our patients by working with their insurance companies, meeting with their families, coordinating care with ancillary providers, and interacting with their employers. Patient and health care advocacy is a responsibility we all have and should take seriously. Over the years, I have come to realize that physicians also have a role and responsibility in protecting and improving the health and well-being of the public, as well as our patients. I provide for your review, the following excerpt from the AMA Ethics Policy H-140.900, Declaration of Professional Responsibility. “...As physicians, we are bound in our response by a common heritage of caring for the sick and the suffering. Through the centuries, individual physicians have fulfilled this obligation by applying their skills and knowledge competently, selflessly and at times heroically. Today, our profession must reaffirm its historical commitment to combat natural and man-made assaults on the health and well-being of humankind. Only by acting together across geographic and ideological divides can we overcome such powerful threats. Humanity is our patient.

Declaration

We, the members of the world community of physicians, solemnly commit ourselves to: (1) Respect human life and the dignity of every individual; (2) Refrain from supporting or committing crimes against humanity and condemn any such acts; (3) Treat the sick and injured with competence and compassion and without prejudice; (4) Apply our knowledge and skills when needed, though doing so may put us at risk; (5) Protect the privacy and confidentiality of those for whom we care and breach that confidence only when keeping it would seriously threaten their health and safety or that of others; (6) Work freely with colleagues to discover, develop, and promote advances in medicine and public health that ameliorate suffering and contribute to human well-being; (7) Educate the public and polity about present and future threats to the health of humanity; (8) Advocate for social, economic, educational, and political changes that ameliorate suffering and contribute to human well-being; (9) Teach and mentor those who follow us for they are the future of our caring profession. We make these promises solemnly, freely, and upon our personal and professional honor.”

This declaration defines medicine as a profession. There are numerous opportunities to advocate on behalf of our patients and to further our calling as physicians. One way to make a difference is to simply get to know, and hopefully form relationships with, elected officials in your district. Research has shown that constituents who develop relationships with their legislators are more likely to be identified by legislators as being relevant to an issue, and with only a handful of legislators who possess a background in health care, your involvement is important. I encourage you to become politically active.
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Cardiovascular disease (CVD) is the leading cause of death worldwide. Hypertension (HTN) is strongly associated with CVD, and it is a modifiable risk factor. Guidelines for the management of HTN have been changing, and many expert groups release periodic recommendations with treatment goals. Although JNC 8 (Report from the Panel Members Appointed to the Eighth Joint National Committee) had relaxed the threshold for treatment of HTN in 2014, the SPRINT trial demonstrated marked benefit of targeting lower blood pressures in certain high-risk populations soon afterward. The results of the SPRINT trial – along with well-established epidemiologic data showing a graded relationship between blood pressure (BP) and outcomes above a BP of 115/75 mmHg – have led the American College of Cardiology and American Heart Association to release new guidelines for definition and treatment of HTN. These recommendations seek to unify all emerging data and guide optimal clinical practice.

The new guidelines are summarized in Table 1. These updated diagnostic strata will result in an increased number of patients diagnosed with HTN, increasing prevalence from 32 percent of the U.S. adult population to 46 percent. This will undoubtedly lead to a shift in prescribing patterns. It is also likely that the lowered threshold will result in a higher proportion of patients being counselled regarding life style changes. Thus, four specific changes in practice are anticipated: (1) therapeutic lifestyle changes, (2) increased monotherapy, (3) increased combination therapy, and (4) closer monitoring in the elderly.

Table 1. Categories of BP in adults

<table>
<thead>
<tr>
<th>BP Category</th>
<th>Systolic BP</th>
<th>Diastolic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt; 120 mm Hg</td>
<td>&lt; 80 mm Hg</td>
</tr>
<tr>
<td>Elevated</td>
<td>120-129 mm Hg</td>
<td>&lt; 80 mm Hg</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Stage 1</td>
<td>Stage 2</td>
</tr>
<tr>
<td></td>
<td>130-139 mm Hg</td>
<td>&gt;= 140 mm Hg</td>
</tr>
<tr>
<td></td>
<td>or 80-89 mm Hg</td>
<td>or &gt;= 90 mm Hg</td>
</tr>
</tbody>
</table>

Patients with stage 1 HTN (systolic 130-139 mmHg or diastolic 80-89 mmHg) need to be informed about their increased CVD risk and about the positive benefit of lifestyle changes. Key life style changes include weight loss, moderating alcohol consumption, increasing physical activity and consuming a heart healthy diet. Additionally, the 10-year risk of developing atherosclerotic cardiovascular disease (ASCVD) should be calculated and pharmacological intervention should be recommended if their 10-year risk of ASCVD is greater than or equal to 10 percent. This can be done with the same automated decision support (risk calculator) used to assess statin benefit. The presence of CVD, diabetes, chronic kidney disease or heart failure also warrant pharmacological intervention. The goal for the vast majority of patients (regardless of their ASCVD risk or initial BP) is to achieve a BP of less than 130/80 mmHg. The choice of first line medications did not change, with calcium channel blockers, angiotensin converting enzyme inhibitors/angiotensin receptor blockers and thiazide diuretics recommended.

The new lower thresholds are also associated with more aggressive initial intervention in patients with severe disease. According to the new guidelines most adults with stage 2 HTN (systolic BP exceeding 140 mmHg, or diastolic BP exceeding 90 mmHg) will need combination therapy with initiation of pharmacologic therapy. Further, the new guidelines also suggest more aggressive blood pressure management in the elderly (age greater than 65). The new recommended target systolic BP is less than 130 mmHg, the same as the younger population. This is consistent with the common observation that many elderly patients have a 10 percent risk or greater of developing CVD in the next 10 years if they do not already carry the diagnosis of CVD. Many trials have demonstrated both the safety and efficacy of this practice.

Where are we Heading?

The new combined treatment guidelines for HTN indicate that “lower is better.” There will now be a surge in the number of patients meeting diagnostic criteria for HTN,
Editorial

and assessment of their CVD risk with automated decision support (risk calculators embedded in electronic medical records) will guide our recommendations to start medications or simply recommend lifestyle interventions with longitudinal follow up. For optimal effectiveness, clinicians should adopt these changes into their practice in the context of shared decision making with patients and their families. This approach will be particularly important in very old patients with frailty and a higher risk of medication side effects.

Acknowledgments: The authors would like to thank Dr. Russell Wilke, MD for his valuable comments during the preparation of this manuscript.

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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State of South Dakota’s Child: 2017

By Ann L. Wilson, PhD; Tyler Hemmingson, MPH; and Brad Randall, MD

Abstract
There was a slight decrease in 2016 from 2015 in the total number of live births in South Dakota, but it was the fifth consecutive year that there were more than 12,000 newborns in the state. Nearly one-quarter of South Dakota’s births represent minority populations mirroring what is observed nationally. Infant mortality in South Dakota dropped to its lowest ever rate per 1,000 live births (4.8) in 2016. Fewer births of less than 500 g newborns, increased survival of very low birth weight newborns, and a decrease in deaths due to congenital anomalies contributed to this low mortality rate. Though there is little progress observed in decreasing the trend in rate of sudden unexpected infant death (SUID), 2016 brought a decrease in the rate of these deaths from a previous spike in 2015. While positive indicators are observed in the 2016 data, South Dakota’s mean mortality rate of 6.6 for 2012-16 is statistically higher than the U.S. rate of 5.8 for 2014. Further, consistent with previous trends, the South Dakota mean 2012-16 mortality rate for minority infants (11.8) was significantly higher than that for white infants (5.1). These observations are discussed with strategies to prevent infant deaths.

Natality
The year 2016 greeted 12,270 newborn residents to the state of South Dakota. This number is slightly less than the previous year’s births in the state, and as noted in Figure 1, is the fifth consecutive year with more than 12,000 live births of residents in the state. Figure 2 shows that the trend toward greater racial diversity among the annual cohort of South Dakota newborns is continuing. Since 2003, the state’s percent of minority newborns has been similar to what is observed nationally with approximately one-quarter of all births minority newborns. Currently, 64 percent of these newborns in...
South Dakota are American Indian compared to 87 percent in 2000. While there have been changes in how births are coded by race, the greater diversity of the state’s population is apparent in these data. The most current birth rate (births per 1,000 population) for South Dakota is 14.4 compared to the provisional rate of 12.4 for the U.S.

An important parameter of health of a cohort of newborns is revealed by data on birth weight. Figure 3 compares low birth weight (LBW; less than 2,500 g) of newborns in South Dakota with those of the U.S. Though the state's percent of LBW in 2016 increased to approximately 7 percent, over time this percent has consistently been less than the national rate. Figure 3 demonstrates a trend of noticeably increasing low birth weights nationally and in South Dakota in the 1990s followed by a decrease in recent years.

The data presented in Figure 4 allow an examination of trends in specific South Dakota LBW cohorts. Apparent in this figure is how in 2016 the percent of infants in the two cohorts of mid low birth weight (MLBW; 1,500-1999 g and 2,000-2,499 g) slightly increased. Alternately, the percent of very low birth weight (VLBW; less than 500 g, 500-999 g, and 1,000-1,500 g) newborns decreased or remained similar to what was observed in recent years.

Very apparent is the decrease in the sub-group of VLBW newborns who weighed less than 500 g. In the previous five years there has been an average of 19 newborns in this weight category, and in 2016 there were only seven. This is noteworthy as typically survival of newborns in this weight group is not likely, thus affecting overall infant mortality rates (IMR).

**Infant Mortality**

Noted in Figure 5 is how the 59 infant deaths in South Dakota in 2016 yielded an IMR of 4.8 per 1,000 live births which is the state’s lowest ever rate. This rate is also lower than the most recent available U.S. 2015 rate of 5.9. The previous lowest state rate (5.5) was observed in 2000. We must recognize, however, the variation inherent in South Dakota’s infant mortality rate. The variation from year to year demonstrates how fluctuations in annual deaths express themselves in rates calculated with the small numbers of South Dakota’s births. Nonetheless, this low 2016 rate is a dramatic fall from the 7.3 IMR observed in the state in 2015. The factors that contributed to it will be explored below.

To better understand mortality during the first year of life, mortality rates are examined as occurring in two time periods. Neonatal mortality includes deaths that occur
between birth and the 27th day of life and post neonatal mortality includes deaths that occur between the 28th day and the end of the first year of life. Presented in Figure 6 are neonatal mortality rates (NMR) for South Dakota and the U.S. Very apparent in this graph is how the NMR decreased in 2016 for both South Dakota’s white and minority populations from previous observations. While it reached its lowest ever rate (2.5) for the total South Dakota population, a lower rate for the white population was previously recorded in 1996. In 2016, South Dakota’s white and minority populations had lower neonatal mortality rates than those most recently recorded for the U.S. (3.9) in 2015.14 The decrease in the number of less than 500 g infants born in South Dakota in 2016 (86 percent of whom died) likely contributed to the state’s low neonatal mortality observed in this year.

Figure 7 presents a different pattern in the post neonatal mortality rates (PNMR). The South Dakota 2016 white PNMR dropped to 1.2, its lowest ever recorded rate, which is also lower than what was most recently recorded for the U.S. in 2015 (2.0).14 The South Dakota minority 2016 PNMR for minorities matched its previous five year (2011-2015) mean of 5.9 that exceeds the U.S. rate (2.9) in 2014.5 In 2016, one-third of South Dakota white deaths occurred in the post neonatal period of time, while this was true of approximately two-thirds of the minority deaths.1 This differs from what was observed in 2014 for the U.S. when approximately one third of both the white and minority infant deaths occurred in the post neonatal period.1

Between 2012 and 2016, 56 percent of infants who died in South Dakota were LBW, with mortality highest among those in the sub groups of VLBW newborns. Figure 8 presents the birth weight specific mortality for the VLBW and MLBW infants for 2016 compared to the means for 2011-15.1 Noted in this figure is how mortality decreased for the VLBW infants to approximately 15 percent from its previous five year mean of 28 percent. The MLBW mortality slightly increased from 1.7 to 1.8 percent. The most recent national data show that in 2013, mortality for VLBW was 22 percent and 1.3 percent for MLBW,6 which was statistically lower than birth weight specific mortality noted in South Dakota between 2011 and 2015.7

The improved survival of VLBW newborns and decrease in births of less than 500 g newborns is reflected in the 2016 rate of death for infants due to perinatal causes. Noted in Figure 9 is how this rate of death dropped to its lowest rate (1.4) in the past 10 years.1 This decrease and the lower rate of SUID in 2016, compared to 2015, parallel this year’s drop in total IMR.1 SUID is a term that combines deaths from three causes: sudden infant death, ill-defined and unspecified causes, and accidental
suffocation and strangulation in bed. Also noted in Figure 9 is how the rate of infant death due to congenital anomalies declined in 2016 to its lowest rate in 10 years.1

To further characterize causes of infant death, Figure 10 presents their 2016 distribution in South Dakota.1 Perinatal causes (primarily complications of prematurity) and SUIDs equally contribute to nearly 60 percent of all infant deaths this year. Congenital anomalies were the cause of 22 percent of the infant deaths. Accidents/homicides and other causes comprised the remaining 20 percent of the deaths.

Figure 11 presents 2012-16 data on causes of infant death broken down by racial groups and occurrence in the neonatal and post neonatal periods of the first year of life. Between 2012 and 2016, deaths due to perinatal causes comprise 56 percent of white versus 71 percent of minority neonatal deaths which is a significantly different distribution (p<0.05). Alternately, during these years, congenital anomalies comprise 37 percent of white and 16 percent of minority neonatal deaths. This also represents a significantly different distribution (p<0.001). A similar pattern of distribution by cause is apparent among the post neonatal deaths for the white and minority populations.

While Figure 11 enables racial comparisons of causes of death in the state, it does not present their rates per population of live births. Table 1 presents the 2012-16 mean rates for causes of death for the total population of infants by white and minority groups and the statistical differences noted between them. With the exception of
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Amy
AWC! Participant

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congenital anomalies, the higher rates of death for minorities than whites in South Dakota are statistically significant for all causes of death. The higher total IMR for minorities than whites in South Dakota is also statistically significant. In addition, Table 1 allows comparisons of the mean 2012-16 rates of death for the total South Dakota populations with the most recent 2014 data for the U.S. The higher rates of death for congenital anomalies, SUID, and accidents/homicides in South Dakota versus U.S. are statistically significant. Further, South Dakota’s mean total infant mortality rate for 2012-16 is significantly higher (p<0.01) for the state than it was for the U.S. in 2014.

**Discussion**

The IMR in South Dakota for 2016 is the lowest ever observed. This rate (4.8), for the first time in nearly a decade, is also lower than that observed nationally. While this is a very positive indicator of improved perinatal outcome for the state, caution must always be exercised when interpreting data from one year in South Dakota. As noted in Figures 5-7, there are pronounced annual fluctuations in IMRs that reflect the relatively small numbers of births and deaths in the state. Overall, however, the trends reveal decreasing mortality for infants in South Dakota.

Analyses of the dynamics associated with this low 2016 IMR show that rates of infant death due to perinatal causes in 2016 were the lowest noted in the decade. While there was an increase in the percent of LBW infants, this was accounted for in the MLBW cohort that has a low (approximate 2 percent) mortality. The 2016 decrease in the mortality for VLBW infants and the drop in the number of newborns weighing less than 500 g also were likely to have contributed to the low 2016 IMR.

Another low that appears in the 2016 infant mortality data is the rate of infant death due to congenital anomalies. South Dakota’s rate of infant death attributed to this cause has typically been higher than that noted nationally.

In 2016, the rate of infant death due to congenital anomalies (1.1) was similar to that noted in the most recent 2014 national mortality data. One group of infant deaths that yields a distinct opportunity for prevention includes those identified as caused by SUID. This cause of death in South Dakota decreased in 2016 from the highest rate noted in the past decade in 2015, but reveals a slightly increasing trend over the past decade and is higher than noted nationally. Further, in 2016, close to half of all post neonatal deaths for both the white and minority populations are attributed to SUID, though the rate for the minority population of infants is more than four times higher than it is for white infants. Data from the Regional Infant Mortality Review Committee that reviews deaths for 10 counties in South Eastern South Dakota show that most of these deaths in the past five years are associated with known positional or environment risks to infants’ sleep. Messaging and active direct modeling for parents about safe sleep must continue if these deaths are to be avoided. While for many years this message focused on putting babies to sleep on their backs, today it must also include the vital necessity of babies sleeping *alone* on a firm flat mattress with a fitted sheet in a safety-approved crib devoid of other materials such as soft bedding, bumper pads or toys. Recent guidance from the American Academy of Pediatrics (AAP) describes that infant sleep is unsafe when it occurs in car seats, swings, strollers, carriers or slings. Further, it

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**Table 1. Causes of infant death**

<table>
<thead>
<tr>
<th>Causes</th>
<th>Total population</th>
<th>White</th>
<th>Minority</th>
<th>P White vs. Minority</th>
<th>SD 2012-16</th>
<th>U.S. 2014</th>
<th>P SD vs. U.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perinatal causes</td>
<td>1.39</td>
<td>2.08</td>
<td>4.38</td>
<td>&lt;0.01</td>
<td>2.81</td>
<td>3.0</td>
<td>ns</td>
</tr>
<tr>
<td>Congenital anomalies</td>
<td>1.06</td>
<td>1.62</td>
<td>1.74</td>
<td>ns</td>
<td>1.63</td>
<td>1.1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Sudden unexpected infant death</td>
<td>1.39</td>
<td>0.77</td>
<td>3.27</td>
<td>&lt;0.01</td>
<td>1.34</td>
<td>0.9</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Accidents and homicides</td>
<td>0.41</td>
<td>0.26</td>
<td>1.11</td>
<td>&lt;0.01</td>
<td>0.46</td>
<td>0.1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Other</td>
<td>0.57</td>
<td>0.37</td>
<td>1.32</td>
<td>&lt;0.01</td>
<td>0.59</td>
<td>0.7</td>
<td>ns</td>
</tr>
<tr>
<td>Total infant mortality</td>
<td>4.81</td>
<td>5.1</td>
<td>11.83</td>
<td>&lt;0.01</td>
<td>6.63</td>
<td>5.8</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

*All South Dakota data is from South Dakota Department of Health
ns = nonsignificant
recommends that babies sleep in the parents’ room but not in their bed for the first year of life, or at least for the first six months. More specifically, babies should sleep in a safe crib, portable crib, play yard or bassinet to avoid the risks of suffocation, strangulation and entrapment. These recommendations must accompany the message that compliance with them becomes a habit. Deviation from these recommendations “just once” can be fatal for an infant and tragic for a family.

Questions may be raised regarding the optimal timing for safe sleep education. As nearly three out of 10 infant deaths in South Dakota were attributed to SUID in 2016 (see Figure 10), strategic education and intervention has the potential to significantly impact infant mortality. Currently, postpartum instruction routinely covers the topic of safe sleep, but is this the most opportune time to share this information? New parents often describe feeling rushed during the hours following delivery of a new baby with so much to learn as skills in caring for the newborn are being practiced, often for the first time. Checklists of instructions are provided to parents, but questions can be raised about how much of this information is absorbed. Perhaps information on safe sleep could be best presented during the prenatal period of time when baby equipment is being purchased and plans for modifying a home to accommodate a baby are being made. Well baby checks are another time when discussion of safe sleep can take place. Further, the message of “safe sleep” must also reach an entire community, especially those who provide routine or periodic child care. When a community develops expectations that babies will sleep in safe environments, baby care practices may become more compliant with recommendations that prevent these tragic deaths. This does not, however, minimize the power of modeling of baby care practices may become more compliant with recommendations that prevent these tragic deaths. This does not, however, minimize the power of modeling of baby care practices to become more compliant with recommendations that prevent these tragic deaths.

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A persistent theme that emerges as data on infant mortality are examined in South Dakota is the discrepancy in survival of white and minority infants. This disparity is especially apparent as rates of death are compared in the post neonatal period. Though data are not available that provide direct correlations between family resources and infant survival, assumptions can be made about the prevalence of poverty and its association with survival among infants of minority racial groups. Not only is the rate of SUID four times higher for minorities than for whites, this is also true for the rate of accidents and homicide. Why is this so?

As noted in these previous publications, life during the first year is fragile. Not until age 55 will the rate of death be as high as it is during its first 12 months. Infant mortality has long been considered a mirror of society’s investment in its future. Yet, parents’ warmest dreams and emotional investment in the planning and support of a developing baby during pregnancy and early months of postnatal life, even when accompanied by the best of health care, can tragically end with an infant’s death. Reviews of the circumstances surrounding infant death, however, often accompany descriptions of chaotic environments for families with limited financial and social resources. The challenge of protecting survival during this first year can be seen as reaching babies with care and education for their parents in these difficult circumstances. How can this be done? There are no easy answers. The AAP, however, reports that the risk of sudden infant death is lower when mothers receive regular prenatal care. One can speculate that education on healthy beginnings for babies is one part of what is (or should be) received during prenatal care. Perhaps equally important, though more difficult to measure and assess, are the relationships that form between care providers and parents that facilitate responsiveness to education. Providing parents and caregivers information on safe sleep or even a cost-free safe crib, cannot assure risk-free sleep for a baby. Messages, however, that are shared within the context of a responsive, warm, and consistent health care provider-parent relationship may be better heard and heeded by expectant or new parents. This suggestion recognizes the optimism it reflects, but is offered with respect for the complexity of changing behavior that may improve survival during the first year of life.

**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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A Rare Cause of Pancreatic Head Mass: Metastatic Prostate Cancer – A Case Report and Review of Literature

By Khalil Aloreidi, MD; and Robert T. Lapp, MD

Abstract
Metastases to the pancreas account for less than 5 percent of all malignancies affecting the pancreas. The most common secondary malignancy of the pancreas is renal cell carcinoma. We report a patient presented with abdominal pain and weight loss. Computed tomography (CT) imaging showed pancreatic head mass. Biopsy from the mass showed prostate metastasis after 13 years from radical prostatectomy and Leuprolide therapy. This case demonstrates a rare location for prostate metastasis which was the pancreas. To our knowledge there are only six cases reported in literature. Due to increased long-term survival of prostate cancer patients, the frequency of metastases to the pancreas will likely increase. Therefore, clinicians need to be aware the pancreatic tumor may be secondary to an extrapancreatic malignancy.

Introduction
The majority of pancreatic malignancies are primary tumors. Metastases to the pancreas are rare, accounting for 2-5 percent of all pancreatic malignancies. Prostate cancer metastases to the pancreas are very unexpected. To our knowledge there are only six published cases of prostate metastasis to the pancreas. At the time of diagnosis, prostate cancer metastasized disease in 4 percent of patients. Secondary tumors of the pancreas are difficult to differentiate from primary tumors given the similar clinical and radiological features. In patients with a history of extra-pancreatic malignancy presenting with a pancreatic mass, metastatic disease should be considered in the differential diagnosis. Tissue acquisition is the standard to differentiate between primary and secondary pancreatic tumors.

Case Presentation
An 80-year-old man presented with lower back and abdominal pain associated with unintentional weight loss of about 15 pounds for the last two months. He had a history of prostate cancer diagnosed 13 years ago for which he underwent radical retropubic prostatectomy demonstrating T2N0 Adenocarcinoma with Gleason score of 7. Upon examination, he was alert oriented with no scleral icterus. His lungs were clear and his heart sounds were normal. Abdominal examination showed normal bowel sounds. He had mild discomfort to deep palpation in epigastric area with no guarding or rebound. He had no palpable masses or hepatosplenomegaly. Laboratory studies showed elevated liver enzymes with alkaline phosphatase of 691 IU/L, aspartate aminotransferase 78 IU/L, and alanine transaminase of 91 IU/L. Prostate-specific antigen (PSA) level was 149 (0-6.21) ng/mL. Subsequent abdominal CT scan showed an ill-defined, irregular 3.7 cm x 3.3 cm pancreatic head mass (Figure 1).
with upstream dilation of the common bile duct to 1.4 cm, these findings were suspicious for primary pancreatic adenocarcinoma. Biliary decompression with endoscopic retrograde cholangiopancreatography (ERCP) demonstrated a 10 mm distal biliary stricture with upstream dilation of the common bile duct to 1.5 cm. The biliary stricture was brushed for cytology and placement of a temporary biliary stent was performed. Cytology brushings were negative for malignancy. Subsequent endoscopic ultrasound (EUS) demonstrated a 4.9 cm x 3.5 cm hypoechoic pancreatic head mass (Figure 2). Fine needle aspiration (FNA) of the pancreatic head mass was consistent with adenocarcinoma during on-site rapid interpretation (Figure 3). Given the elevated PSA, Immunohistochemistry was done which showed cells positive for PSA and prostate-specific acid phosphatase (PSAP) consistent with metastatic prostate cancer (Figures 4 and 5). Initial treatment with docetaxel for metastatic prostate cancer was tolerated poorly. Therefore, the patient was transitioned to hospice care.

Discussion

Metastatic disease to the pancreas is rare; however, it is being observed with increasing frequency, especially in high-volume centers. Renal cell, colorectal, and lung carcinomas are known to metastasize to the pancreas. Prostate cancer most commonly metastasizes to bone, in particular the axial and proximal long bones, which represent 90 percent of distant metastases. An increase in the cases of metastatic prostate cancer to the pancreas are expected, given the five-year survival rate of prostate cancer of 98.9 percent and 29.3 percent for metastatic disease.

Clinical differentiation of primary and secondary tumors of the pancreas is difficult due to similar presentations. Secondary pancreatic tumors are asymptomatic in more than 50 percent of cases. If present, symptoms are...
typically vague, including, abdominal/back pain, weight loss and fatigue.\textsuperscript{9}

It may be challenging to distinguish between primary and secondary pancreatic tumors by cross sectional imaging alone. Primary tumors appear as hypo-attenuating lesions on cross sectional imaging. Secondary tumors may be hyper-, iso- or hypo-attenuating making a diagnosis difficult.\textsuperscript{10-12} In addition, pancreatic tumors may lead to pancreatitis impeding adequate cross sectional imaging. Secondary tumors appear uniformly throughout the pancreas, while primary tumors are most commonly found in the head of the pancreas.\textsuperscript{12-14}

Serum biomarkers have been studied in pancreatic cancer.\textsuperscript{15} The most studied is the carbohydrate antigen 19-9 (CA 19-9). The sensitivity and specificity of CA 19-9 for the diagnosis of pancreatic cancer is 70-92 percent and 68-92 percent, respectively.\textsuperscript{16} Limitations for the sensitivity of CA 19-9 are tumor size, requires the presence of Lewis antigen, and may be elevated in patients without pancreatic cancer.\textsuperscript{17,18} PSA is highly sensitive for the diagnosis of prostate cancer. Higher levels of PSA are associated with an increased likelihood of metastasis.\textsuperscript{19} A serum PSA less than 20 ng/ml have high negative predictive value in ruling out bone metastasis.\textsuperscript{20}

The similar clinical, radiological and laboratory findings of both primary and metastatic pancreatic tumors demonstrate the need for tissue acquisition to differentiate between primary and metastatic tumors to the pancreas. EUS with fine needle aspiration is commonly used for tissue acquisition given the minimally invasive nature of this test. The majority of primary pancreatic tumors and metastatic tumors to the pancreas are adenocarcinomas, which makes differentiation difficult. Immunohistochemical stains may help in differentiation, which were used in this case to confirm a final diagnosis. In addition, metastases to the pancreas may occur years after the initial diagnosis, with median time between primary tumor diagnosis and metastatic diagnosis of nine years.\textsuperscript{1} Therefore, metastatic disease to the pancreas should be considered when evaluating any pancreatic mass, even years after a primary tumor has been diagnosed and/or treated.

\textbf{REFERENCES}


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Robert T Lapp, MD, University of South Dakota School of Medicine, Department of Gastroenterology, Sioux Falls, South Dakota.
Recurrence In-stent Restenosis in the Area of Previous Stent Fracture: A Management Dilemma

By Udit B. Bhatnagar, MD; Josh Rezkalla, MD; Tomasz Stys, MD; and Adam Stys, MD

Abstract
Drug-eluting stents (DES) have been increasingly being used for treatment of coronary artery disease (CAD) and have been shown to be very effective in prevention of primary in-stent restenosis (ISR). However DES have been increasingly associated with acute to subacute risk of stent fractures (SF). There is also a paucity of data about different management strategies for SF, especially in the long term. We present a case of recurrent ISR in an area of previous acute SF at the touchdown of saphenous venous graft (SVG) to first diagonal artery (D1). In our knowledge this is the first case reported of recurrent ISR due to prior acute stent fracture in a saphenous venous graft. It presents an interesting management dilemma with multiple layers of stent in the area of recurrent ISR which was managed with balloon angioplasty with good results.

Introduction
Drug-eluting stents (DES) have become a cornerstone in coronary percutaneous revascularization. The novel design of DES has lowered the restenosis rates of coronary arteries when compared to bare-metal stents (BMS). However, as the usage of DES continues to grow we see some challenges affecting them in similar fashion to bare metal stents, such as stent fracture (SF). The incidence of SF varies from 0.84-8.4 percent but an autopsy study revealed that DES SF often goes unrecognized and incidence may be as high as 29 percent. SF can occur acutely (during stent placement) or be detected during routine surveillance angiography months or years later. Both acute stent fracture and chronic SF have been consistently associated with increased major adverse cardiac events (MACE). A higher number of stent fractures have been reported in the right coronary artery (RCA), which has been postulated to be due to increased motion and shear stresses in the RCA and there is paucity of data regarding long term management of stent fractures in drug eluting stents. We present a case of recurrent in-stent restenosis (ISR) in the saphenous vein graft (SVG) touchdown to the first diagonal branch of left anterior descending artery (D1) at the site of previous stent fracture.

Case Report
A 76-year-old female presented with non-ST elevation myocardial infarction (NSTEMI). Her history included a four-vessel coronary artery bypass over 10 years ago. She had previously undergone PCI to the distal SVG graft to D1 8 years ago then returned one year ago with crescendo...
angina due to severe i-stent stenosis (95-99 percent) of the distal touchdown of SVG to D1. An everolimus eluting DES (2.5 mm x 16 mm) was placed crossing from distal SVG extending to the first diagonal (stent in stent). The stent balloon was inflated at 22 atmospheres (atm) for 10 seconds and then again at 24 atm for 20 seconds. After balloon withdrawal, an acute stent fracture was noted on angiography (Figure 1). Thus, another everolimus DES (3 mm x 18 mm) was deployed over the fractured stent segment (Figure 2).

Approximately one year later the patient presented again with NSTEMI. Coronary angiogram revealed ISR (90%) at the distal SVG to D1 at the site of previous stent fracture. A 2 mm x 12 mm noncompliant coronary balloon was inflated at 18 atm for 15 seconds followed by another balloon 3 mm x 12 mm inflation at 18 atm for 15 seconds.
The result was satisfactory (Figures 3-5) and as there were already three layers of stents in this area, a decision was made to not add another stent. Following the procedure the patient was placed on dual antiplatelet therapy and did very well clinically on six months follow up.

**Discussion**

DES have been shown to be very effective in prevention of primary ISR; however, their use in this setting is associated with increased risk of SF. Stent fractures are associated with significantly increased rates of ISR and thrombosis. In some studies, about 38 percent of patients with SF causing ISR required target lesion revascularization compared to only 8 percent of patients with ISR without SF. However, there is little data about the efficacy of different management strategies for SF, especially long term. SF has been associated with longer stent length, overlapping stents, and vessel segment tortuosity. The incidence of SF has also been shown to be higher in sirolimus eluting stents (SES) which is thought to be secondary to their closed-cell strut design, decreasing their flexibility. A majority of SF has been reported in the RCA, possibly due to exposure to more excessive radial stresses during the cardiac cycle. Our case was one of the first cases of acute stent fracture reported in a saphenous vein graft. The shear and tear forces at the graft touchdown plus its connection angle could be the reason for acute SF. The patient presented later with recurrent ISR in the area of previous stent fracture.

In our case, the patient already had three layers of stent in the area of ISR which, as with previously described cases, is likely secondary to increased “hinge” movement in the touchdown point from SVG to D1 and so the areas are exposed to higher radial forces and longitudinal stent deformation. Although DES placement in the area of ISR has shown to decrease the rate of further restenosis compared to balloon angioplasty, the majority of available data has evaluated up to one recurrence of restenosis. Our patient had already had three layers of stents which created a unique management dilemma. We subsequently managed this with balloon angioplasty, with so far good result.

**Conclusion**

Our case outlines the unique dilemma of management of recurrent restenosis in the area of multiple overlapping stents due to prior acute stent fracture at the touchdown point of SVG to D1. Stent fractures are frequently subclinical and missed until ISR develops. The management of recurrent ISR is not clearly defined. Our patient had ISR with previous three layers of stents which was managed by balloon angioplasty alone good clinical result.

**References**


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AngioVac System Used as an Adjunct Treatment for Intra-Cardiac Lead and Valvular Vegetations

By Vishesh Kumar, MD; Shahbaz A. Malik, MD; Maryam Sheikh MD; and Adam Styx MD

Abstract
Infections are known complications of cardiovascular implantable electronic devices (CIEDs). We describe a case of a 62-year-old male who presented with pulseless electrical activity (PEA) cardiac arrest and respiratory failure. He had a history of cardiac resynchronization device and defibrillator (CRT-D) implantation for non-ischemic cardiomyopathy. After resuscitation, he was found to have methicillin sensitive Staphylococcus aureus (MSSA) bacteremia on blood culture and large vegetations on the CRT-D lead and tricuspid valve found on echocardiography. The patient underwent extraction of the leads, but several large vegetations were present adherent to the tricuspid valve on intra-cardiac ultrasound (ICE). Due to comorbidities, the patient was not a candidate for surgical removal of these vegetations. Thus, he underwent percutaneous extraction of tricuspid and right atrial vegetations with the AngioVac device.

Introduction
We describe a patient who presented with encephalopathy, respiratory failure, cardiac arrest, septic shock, MSSA bacteremia due to CRT-D lead vegetation and tricuspid valve endocarditis. Despite lead extraction, the patient still had vegetations in his right atrium and on the tricuspid valve. Since surgery was considered too high risk due to his comorbidities, a percutaneous approach was pursued with successful removal of the bulk of the vegetations. This case illustrates the safe and effective use of the AngioVac cannula and device system as an adjunctive treatment for large intra-cardiac vegetations.

Case Report
A 62-year-old male was found to be confused, lethargic, covered in feces and urine by his home nursing staff for an unknown period of time. The patient was immediately transferred to an emergency department. The patient had a history of non-ischemic cardiomyopathy with implantation of cardiac resynchronization device and defibrillator (CRT-D) implanted seven years prior to presentation. He was a non-insulin-dependent diabetic and morbidly obese. Shortly after presentation to the emergency department, his respiratory status deteriorated. He required intubation with mechanical ventilation followed by sustained pulseless electrical activity (PEA) cardiac arrest. Cardiopulmonary resuscitation (CPR) was performed with return of spontaneous circulation. The patient was then admitted to the ICU requiring vasopressor support with norepinephrine. Blood cultures collected on admission, were reported to be growing gram-positive cocci; these were eventually identified as methicillin sensitive Staphylococcus aureus (MSSA). The patient had been started on vancomycin and piperacillin-tazobactam and eventually antimicrobial therapy was adjusted to intravenous nafcillin according to final culture results. During the course of hospitalization, the patient developed a maculopapular rash, attributed to nafcillin; thus, antibiotic was switched to intravenous cefazolin.

A transesophageal echocardiogram showed 2 cm x 2 cm mobile masses on the atrial side of the ICD lead, traversing the tricuspid valve, consistent with vegetations, shown in Figure 1. The left ventricular ejection fraction was 50-55 percent. Interrogation of the CRT-D revealed no recent ventricular tachyarrhythmias or device discharges. Removal of the device and extraction of leads was successfully performed, but despite removal of all leads, several large intra-cardiac...
vegetations were still seen in the right atrium adhering to the tricuspid valve on ICE, shown in Figure 2. Cardiothoracic surgery was consulted; however, the patient was deemed a poor surgical candidate on account of his elevated Society of Thoracic Surgeons mortality score; thus, a percutaneous approach to retrieve of the vegetations was suggested.

The patient underwent venovenous cardiopulmonary bypass with the AngioVac device. The access for this device was obtained through the right internal jugular vein with a 26 French sheath. The left and right femoral veins as well as right femoral artery access was used for cardiopulmonary bypass. Unfractionated heparin was used as anticoagulation as the AngioVac cannula was inserted.

The procedure was performed under fluoroscopic and echocardiographic guidance with numerous passes made within the right atrium. The vast majority of the vegetation burden was removed from the tricuspid valve and right atrium, shown in Figure 3. There was no significant tricuspid regurgitation seen after the procedure.

Pathological and microbiological analysis of the specimens revealed fibro-purulent tissue with numerous gram-positive cocci. These were eventually identified as MSSA.

He was eventually extubated and discharged to a skilled nursing facility for antibiotic management and physical therapy. He had a peripherally inserted central catheter (PICC) line placed and was discharged on IV cefazolin 2 g every eight hours to complete total six weeks of antimicrobial therapy. He has thus far remained in good health.

**Discussion**

To our knowledge, there have been only three other cases reported, in a single case series, describing the use of the AngioVac device for removal of percutaneous extraction of large vegetations (greater than 2 cm) from ICD/pacing leads.¹

Use of CIEDs have increased 49 percent in the U.S. between 1999 and 2003.² One of the major complications of CIEDs is infection of intra-cardiac device leads, with an incidence of around 10 percent.³ Lead infection is frequently complicated by formation of large vegetations.⁴ Mortality in these patients remains high – 18 percent despite antibiotic treatment and device removal and 66 percent without such treatment.⁵ Since many of these patients have significant comorbidities which may deter them from being candidates for open thoracotomy there is room for consideration of percutaneous removal of intra-cardiac vegetations.
The AngioVac Cannula is indicated for use as a venous drainage cannula during extracorporeal bypass for up to six hours. The cannula is also indicated for removal of fresh, soft thrombi or emboli during extracorporeal bypass for up to six hours. The AngioVac device has a cannula with a proprietary balloon-actuated, expandable, funnel-shaped distal tip to enhance flow, prevent clogging of the cannula and facilitate en bloc removal of undesirable intravascular material. An extracorporeal bypass circuit is created outside the body consisting of an outflow line, a centrifugal pump, a filter and an inflow line. This creates a one-way flow to provide suction at the tip of the AngioVac cannula. The circuit reinfuses the filtered blood back into the body to minimize blood loss without causing hemodynamic instability. This device was used off-label for valvular vegetation extraction in our case.

In summary, we suggest this novel approach may be considered for removal of large intra-cardiac vegetations as an adjunctive treatment after infected lead extraction in patients who are high risk to undergo open thoracotomy.

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3. To reduce the risk of suffocation and SIDS, infants should be placed on a firm sleep surface (e.g., mattress in a safety-approved crib) with a fitted sheet and no other bedding, bumper pads, or soft objects.

4. Soft objects and loose bedding should be kept away from infants’ sleep area to reduce risk of SIDS, suffocation, entrapment, and strangulation.

5. Infants should sleep in parents’ room, close to the parents’ bed, but on a separate safe surface, ideally for the first year, but at least for the first six months of life.
Safe and Effective Triglyceride-Lowering Therapy for Hypertriglyceridemia Associated Pancreatitis: Insulin Mono-therapy in a Non-Diabetic Patient

By Chencheng Xie, MD; Dennis C. Stevens, MD, MS; and Tamera Sturm, DO

Abstract

This report was prepared to describe a case in which insulin monotherapy was efficacious for the management of hypertriglyceridemia-associated pancreatitis (HGTP) in a patient who was not diabetic. Currently, there are no definite clinical guidelines or standards of practice for nondiabetic HGTP. Apart from insulin infusion, other regimens include plasmapheresis and heparin administration, both of which carry significant risks. We reported a case of a non-diabetic male with HGTP (triglyceride greater than 3,000 mg/dL) who was successfully managed with an insulin infusion. This resulted in achieving a level below 1,000 mg/dL within 28 hours of the initiation of therapy. The patient was discharged without additional incident in 72 hours. We believe that insulin monotherapy for HGTP is efficacious in non-diabetic patients and should be the regimen of choice.

Introduction

Hypertriglyceridemia (HTG) is the third most common cause of acute pancreatitis (AP) with alcohol use and biliary tract stones being most common. This disorder is rapidly increasing in frequency and accounts for as many as 10 percent of all AP cases. A triglyceride value greater than 1,000 mg/dL (11.2 mmol/L) is an important risk factor for pancreatitis. Early identification of HTG associated pancreatitis (HTGP) and initiation of therapy are critical, decreasing complications of this disorder and decreasing the duration of hospitalization.

No universally accepted guideline for the management of HTGP has been established. Plasmapheresis is generally recommended except for patients with diabetic ketoacidosis (DKA), in whom insulin therapy is used for glucose levels of greater than 500 mg/dL. Plasmapheresis has its limitations including: high cost of treatment, lack of availability, invasiveness, discomfort, infection, deep vein thrombosis, and bleeding. Few cases have reported the use of insulin as single therapeutic agent for nondiabetic HTGP. In this report, we report the successful treatment of nondiabetic HTGP with insulin monotherapy.

Case Presentation

A 31-year-old man presented to the emergency department with excruciating abdominal pain of two days duration. The pain was localized in the right upper quadrant (RUQ) and radiated to his back. He rated the pain as 8 of 10, indicating intense pain with severely limited physical activity. The patient did have a history of moderate alcohol abuse; however, he has been well controlled, consuming a moderate amount of beer with his last drink three days prior to onset of his pain. The patient had a family history of hypercholesterolemia in three paternal uncles. He did not have a history of diabetes mellitus.

The patient was in moderate distress due to acute abdominal pain. His vital signs were within normal limits. He had abdominal pain without rebound tenderness to palpation in the right upper and lower quadrants.

His admission laboratory values are found in Table 1. A computer tomographic scan of the abdomen revealed acute inflammation around the uncinate process of the pancreas without calcification (Figure 1), accompanied by adjacent duodenal wall thickening, and hepatic steatosis. Abdominal ultrasound did not show any stones in the gallbladder, or biliary duct dilation.

The patient was admitted to the hospital with the diagnosis of acute pancreatitis. He was placed on bowel rest, pain management with fentanyl and hydromorphone and an insulin drip. Initially, the rate of insulin infusion
was 0.1u/kg/h (8 units/hour) with 5 percent dextrose by intravenous infusion; however, the patient subsequently experienced an episode of hypoglycemia. The insulin drip was stopped and subsequently re-started at three units/hour. His triglyceride and cholesterol level started decreasing immediately, with the triglyceride levels dropping to less than 1,000 mg/dL by within 28 hours and 597 by 55 hours of insulin treatment at which time the cholesterol was 313 mg/dL (Figure 2). His pancreatic lipase was normal. At this time, the patient’s abdominal pain was improved, the insulin drip was stopped and his diet resumed. The lipoprotein electrophoresis testing indicated type III hypolipoproteinemia. Atorvastatin and gemfibrozil were started for the long-term triglyceride management. The patient was discharged after 72 hours of hospitalization.

Discussion

In HTGP patients, the initial goal is to lower the triglyceride to less than 500 mg/dL. Maintaining the triglyceride below this level in conjunction with aggressive hydration and analgesia can improve clinical symptoms of acute pancreatitis. Among the therapeutic strategies proposed for HTGP are plasma exchange, a combined insulin and heparin drip, and insulin monotherapy. The optimal therapeutic strategy remains controversial. No conclusive randomized controlled trials have directly compared the efficacy of available therapies on the outcomes of patients with HTGP.

In 2013, the American Society for Apheresis Guidelines recommended plasmapheresis for HTGP. They rated the evidence level as Grade 2C (weak recommendation, low quality evidence) and Category III (optimum role of apheresis therapy is not established, decision making should be individualized) due to limited data and conflicting reports. Some of its adverse effects, such as bleeding, infection and thrombosis, must be weighed against the benefits and the therapeutic index. Heparin can also be administered. Its mechanism of action is thought to be the release of lipoprotein lipase (LPL) from endothelial stores which enhances triglyceride clearance by enzymatic degradation. It carries the risk of exacerbating hemorrhage in patients suffering from pancreatitis making its use controversial. Some of its adverse effects, such as bleeding, infection and thrombosis, must be weighed against the benefits and the therapeutic index. Heparin can also be administered. Its mechanism of action is thought to be the release of lipoprotein lipase (LPL) from endothelial stores which enhances triglyceride clearance by enzymatic degradation. It carries the risk of exacerbating hemorrhage in patients suffering from pancreatitis making its use controversial. Some of its adverse effects, such as bleeding, infection and thrombosis, must be weighed against the benefits and the therapeutic index. Heparin can also be administered. Its mechanism of action is thought to be the release of lipoprotein lipase (LPL) from endothelial stores which enhances triglyceride clearance by enzymatic degradation. It carries the risk of exacerbating hemorrhage in patients suffering from pancreatitis making its use controversial.

Insulin infusion also decreases triglyceride levels. Insulin theoretically increases LPL activity which degrades chylomicrons that transport ingested lipids and reduces serum triglycerides. If the patient has diabetes mellitus and glucose levels above 500 g/dL, current guidelines recommend insulin therapy, however, for
non-diabetic patients or those with glucose levels below 500 g/dL, the efficacy of insulin treatment is not well documented, and the therapeutic strategy remains controversial. A number of case reports in the recent literature support the use of insulin monotherapy as the treatment of choice for nondiabetic patients with HTGP.10,11

**Conclusion**

Our case reveals that insulin can be administered safely and efficiently in a non-diabetic HTGP patient, who does not have signs of worsening pancreatic inflammation and dysfunction. This clinical case supports the use of insulin monotherapy as a potentially credible regimen for nondiabetic patients with HTGP and should be considered as the regimen of choice for future clinical practice.

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**REFERENCES**


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Pediatric Viral Myocarditis – A Review

By Teresa Frey, DO; and Nofil Arain, MD

Abstract
Pediatric myocarditis is a common pediatric illness most commonly secondary to a preceding viral infection. It is a leading cause of acquired heart failure, cardiomyopathy, and cardiac transplantation in pediatrics. Due to the variability in presentation, the diagnosis is often unrecognized until later in the disease course. It should be considered in the differential diagnosis of all children presenting with respiratory distress, since this is the most common presentation. Imaging modalities, such as cardiac magnetic resonance imaging have become a useful diagnostic tool in recent years; however, endomyocardial biopsy remains the gold standard diagnostic test. Treatment of viral myocarditis is mainly supportive, with debatable role of anti-inflammatory, immunosuppressing immunomodulators and anti-viral therapy. Although, the outcome is generally favorable, delay in supportive care may be associated with a worse prognosis. We focus on the clinical presentation, review diagnostic and treatment options of viral myocarditis.

Case
A 16-day-old full-term male presented to the emergency department with respiratory distress. He was born following an essentially uneventful pregnancy. His postnatal course was unremarkable and he was discharged from the nursery without any concerns. He was in a normal state of health until the day prior to presentation, when he started experiencing progressive tachypnea and substernal retractions. There were no other reported signs or symptoms attributable to an acute illness. The family denied any ill contacts and reported the infant had adequate oral intake as well as urine output. A chest X-ray performed in the emergency department (Figure 1) was concerning for cardiomegaly and increased pulmonary vascularity with small pleural effusions.
His initial physical exam aside from sinus tachycardia (P 162 bpm), tachypnea (RR 48 bpm) and mildly increased work of breathing, was not suggestive of significant cardiopulmonary compromise. An EKG showed sinus tachycardia with T-wave inversion in the inferior and lateral leads (Figure 2). An echocardiogram (Figure 3) revealed no structural anomalies, moderately diminished left ventricular systolic function with an ejection fraction of 20 percent, mild to moderate left atrial and ventricular dilatation, and a small pericardial effusion. A complete blood count with differential, point of care glucose and comprehensive metabolic panel were essentially normal, yet he had a markedly elevated BNP at 8473 pg/ml (normal less than 100 pg/ml) and an elevated troponin I at 0.22 ng/ml (normal less than 0.03 ng/ml). Despite absence of inflammatory markers, there was a high suspicion for myocarditis based on his respiratory distress with elevated troponin I and BNP level with no other signs of an acute infectious process. Viral panels were obtained that included coxsackie, echovirus, enterovirus, Epstein Barr virus, parvovirus, hepatitis A, B and C.

The patient was admitted to the pediatric intensive care unit for supportive care where he responded well to intravenous diuretics (furosemide) and inotropic support (milrinone). He was discharged in stable condition after a seven-day hospitalization on oral medications (ACE inhibitor and furosemide). A few days after discharge, his enterovirus PCR returned positive, establishing a diagnosis of viral myocarditis.

He has been followed closely since hospital discharge. His cardiac evaluation three months after the illness revealed normal cardiac function and dimensions. There has been complete resolution of his T-wave abnormalities on EKG (Figure 4) and normalization of his troponin and BNP levels.

**Introduction**

Myocarditis is inflammation of the myocardium, an acquired process resulting from various etiologies. While there are many causes of myocarditis, this article will focus primarily on viral etiologies. Because the clinical presentation of myocarditis in children can be highly variable, it continues to be a challenging diagnosis in pediatrics. It remains a common cause for morbidity and mortality in pediatric patients, as well as the leading cause of acquired heart failure and cardiomyopathy in previously healthy children.1 Viral infections remain the most common cause of myocarditis in children and adolescents. Based on the limited pediatric studies, there continues to be significant debate over appropriate diagnosis and treatment.

**Epidemiology**

The true incidence of pediatric myocarditis is difficult to ascertain because a definitive diagnosis requires an endomyocardial biopsy. Biopsies are not done on a routine basis because of test limitations and its associated risks.
The true incidence, reported at roughly 0.05 percent, is likely underestimated because of limited biopsy specimens and because sudden death is a common presentation of undiagnosed myocarditis. Among pediatric patients, neonates have the highest incidence with a second peak in incidence occurring around mid-adolescence. There is a higher prevalence in males, with one study citing four times higher rates in this population subset.

**Etiology**

Among the various causes of pediatric myocarditis (Table 1), the most common etiology is viral infections. In the past, enteroviruses, such as coxsackie A and B were considered the leading cause. However, the most common viral cause has continued to change over the years. Recently, other enteroviruses, parvovirus and adenovirus have been detected more frequently. There is an opinion that adenovirus may be the most common etiology, but may have a low detection rate due to generation of a decreased inflammatory response within the myocardium. Other viruses, including respiratory syncytial virus, cytomegalovirus, influenza viruses, echovirus, human herpesvirus 6, Epstein-Barr virus, rabies, varicella, mumps, yellow fever, rabdovirus, rubella and HIV have also been implicated.

**Pathophysiology**

The pathophysiology of myocarditis depends highly on the etiology. This could involve immune mediated damage to myocytes by infiltrating immune cells, autoimmune mediated damage of infected cells by circulating autoantibodies, or direct virus related myocardial damage. The process of myocardial damage evolves through a stage of viral replication, acute myocardial injury, chronic changes of fibrosis, remodeling and scarring leading potentially to a dilated cardiomyopathy. Studies have failed to demonstrate a definitive correlation between the severity of illness at presentation and irreversible myocyte damage.

**Clinical Presentation**

The clinical presentation of viral myocarditis in pediatrics can be highly variable, ranging from asymptomatic patients with subtle clinical findings, to fulminant heart failure and even sudden death. Due to this variability, viral myocarditis is difficult to diagnose and is often underdiagnosed. The most common presenting symptoms for all ages are respiratory in nature, predominantly shortness of breath and difficulty breathing. There is often a history of a preceding viral illness one to two weeks prior to the onset of symptoms. In infants, lethargy, emesis, irritability and feeding intolerance are common concerns. In general, infants are more severely affected, often presenting in respiratory failure and circulatory shock.

Tachypnea, increased work of breathing, wheezing and possibly cough are the usual signs apparent on physical exam. Tachycardia, although not as frequent in older children, is present in the majority of children younger than 10 years age. Older children and adolescents will often complain of chest pain and fatigue. Patients will also often exhibit signs of venous

### Table 1. Various causes of pediatric myocarditis

<table>
<thead>
<tr>
<th>Viral</th>
<th>Bacteria</th>
<th>Protozoa and Parasites</th>
<th>Fungi</th>
<th>Non-infectious</th>
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<tr>
<td>Varicella</td>
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<td>Trypanosoma</td>
<td>Candida</td>
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<td>Haemophilus influenza</td>
<td>Toxoplasma</td>
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<td>Leishmaniais</td>
<td>Nocardia</td>
<td>Ulcerative Colitis</td>
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<td>Babies</td>
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<td>Amebiasis</td>
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<td>Penicillin</td>
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<td>Mumps</td>
<td>Treponema pallidum</td>
<td>Schistosomiasis</td>
<td>Blastomyces</td>
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<td>Yellow fever</td>
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<td>Respiratory syncytial virus</td>
<td>Mycoplasma pneumonia</td>
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<td>Cytomegalovirus</td>
<td>Corynebacterium diphtheriae</td>
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<td>Influenza A</td>
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<td>Coxsackie A and B</td>
<td>Tuberculosis</td>
<td>Cysticercosis</td>
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<td>Legionella pneumophila</td>
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<td>Human herpesvirus 6</td>
<td>Meningococcus</td>
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<td>Epstein-Barr virus</td>
<td>Rickettsia</td>
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<td>Human Immunodeficiency virus</td>
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<td>Hepatitis A, B, C</td>
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<td>Measles</td>
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<td>Herpes simplex virus (HSV)</td>
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<td>Influenza H1N1</td>
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congestion, jugular venous distention, pallor and peripheral as well as pulmonary edema. Pediatric patients may also present with gastrointestinal symptoms, vomiting, nausea and abdominal pain, often associated with fever. An overwhelming majority of patients (84 percent), will require multiple visits to the primary physician prior to the diagnosis, with respiratory illness the most frequently given diagnosis. Patients often have findings not fitting a typical respiratory illness, such as hepatomegaly, prolonged respiratory findings and/or an abnormal cardiac exam, including a gallop or murmur.

If the diagnosis is delayed and patients develop fulminant myocarditis, rapidly progressive heart failure or shock, they are less likely to respond to standard supportive measures. Patients with a more severe presentation have a higher risk for cardiac transplantation, sudden cardiac death and mortality in general.

**Diagnosis**

Aside from clues to the diagnosis from the clinical findings, the diagnostic workup should include basic lab analysis (complete blood count with differential, comprehensive metabolic panel, C-reactive protein, erythrocyte sedimentation rate) to look for evidence of infection, inflammation or end organ dysfunction. Nasopharyngeal, blood and urine specimens for cultures or viral PCRs could also be obtained, but may not yield positive results, especially if patient presentation is delayed beyond a week following the onset of viral illness symptoms.

Despite the non-specificity of inflammatory markers, an elevation of B-natriuretic peptide and cardiac enzymes (troponins, creatine kinase-MB) helps to rule out non-cardiac causes of inflammation. However, it is common for laboratory data to be normal and even with normal cardiac markers the diagnosis of myocarditis cannot be completely excluded. There is ongoing research to find specific biomarkers and cardiac autoantibodies to aid in the diagnosis of all forms of myocarditis.

Upon presentation, a chest X-ray is performed in the majority of cases because respiratory symptoms are so prevalent. Although chest films may be normal early in the course of the disease, findings such as cardiomegaly, pleural effusion, pulmonary edema and possibly pulmonary infiltrates may be seen.

Electrocardiography should be performed on all patients suspected of myocarditis, particularly those presenting with chest pain as this may be the only laboratory screen-

Due to improving imaging techniques and availability, cardiac MRI (cMRI) has emerged as probably the most helpful non-invasive diagnostic, as well as prognostic tool for myocarditis. Standard protocols of T1-T2 weighted images, relaxation techniques and delayed contrast enhancement are very accurate in delineating sub-endocardial, as well as myocardial spaces. Certain imaging protocols have attained sensitivity and specificity for signs of myocarditis as high as 78 and 91 percent, respectively, especially if performed in the first two weeks of disease onset. While cardiac MRI can show cardiac chamber sizes, wall thicknesses or ventricular function, it can also very precisely depict areas of focal myocyte edema, ischemia, hyperemia, necrosis and fibrosis. By utilizing established MRI diagnostic criteria, detection of patchy myocardial involvement is achieved due to the excellent correlation between cMRI findings and endomyocardial biopsy results.

However, the diagnostic gold standard is an endomyocardial biopsy (EMB), which is required for a definitive diagnosis prior to delivering definitive treatments (pacemaker, AICD implantation or cardiac transplantation). Limiting factors for preforming a biopsy include the invasive nature of the procedure, increased risk of cardiac perforation and arrhythmias. The patchy distribution of inflammatory changes and mononuclear cells accounts for sampling error and lower sensitivity (3-63 percent), along with the need for multiple biopsies. Additionally, EMB is unable to identify the exact viral etiology in cases of viral myocarditis and is of low yield in cases where the virus predominantly injures the endothelium (e.g., Parvovirus B19, herpes
virus 6). The biopsy sensitivity however, is improved if EMB is performed within 72 hours of presentation.10

Due to biopsy limitations, the American Heart Association and the American College of Cardiology have established guidelines to perform EMB only in certain cases. These include: new onset unstable heart failure without dilated left ventricle, ventricular arrhythmia, advanced second or third degree AV block, and chronic heart failure of two weeks to three months duration with dilated left ventricle, that is not responsive to treatment in one to two weeks.12

Treatment
Overall, the mainstay of treatment for viral myocarditis is supportive and not targeted to the specific etiology. Patients who have mild cases may recover spontaneously without any intervention. Those with moderate to severe forms of the disease may require respiratory support in the form of oxygen supplementation, positive airway pressure or even mechanical ventilation. Diuresis, afterload reduction, intravenous inotropic therapy, as well as vasopressor therapy, may become necessary to support cardiac function and circulation in cases of circulatory shock. Such affected patients often require transitioning to oral anti-congestive and inotropic medications. There has been much debate over the use of anti-inflammatory, immunosuppressing (steroids, azathioprine, cyclosporine), immunomodulator intravenous immunoglobulin) and anti-viral agents.13 Similar to adult literature, limited small scale pediatric studies have revealed mixed results.

Overall, the most current research has failed to find a statistically significant difference in outcomes of combination therapy with steroids, intravenous immunoglobulin (IVIG) and anti-virals.14 Promising results (improved ventricular function and better survival) in certain cases of viral myocarditis has led some centers to propose treatment with immunosuppressive and immunomodulator agents, especially high dose IVIG, in cases of documented viral myocarditis early in the course of disease.9

ECMO is often required to support circulation in patients with heart failure secondary to myocarditis. Left ventricular ejection tends to improve after one week of ECMO. Those patients requiring ongoing ECMO support after two weeks have a survival rate of less than 50 percent.9 Overall, ECMO survival rates were better for individuals with isolated cardiac failure versus those with multiple organ failure. As such, worldwide use of ventricular assist devices (VADs) for pediatric myocarditis is increasing. The Berlin EXCOR trial showed a mortality rate of 8 percent, and of those placed on a VAD 87 percent of patients were successfully bridged to cardiac transplantation. The hope for the future is that patients on VAD will eventually be able to recover enough to allow VAD explantation without requiring transplantation.15

CPR training and education is encouraged to all families of pediatric patients with myocarditis because of existing potential for cardiopulmonary compromise. Depending on the patient’s clinical status at discharge, continuous home monitoring (e.g., apnea/bradycardia monitor) may be considered. However, discharge on these monitors or with an automated external defibrillator (AED) is not routine practice. If clinically indicated, some patients (e.g., continuous milrinone therapy, enlisted for cardiac transplantation, documented ventricular arrhythmias) may require a wearable cardioverter/defibrillator (WCD) versus implantation of an automated implantable cardioverter-defibrillator (AICD).

Allowing time for myocardial recovery in the form of rest is a crucial component of management. Typically, two to four weeks of rest has been proposed before returning to routine activities. However, based on guidelines, young athletes should not be allowed to return to competitive sports and strenuous exercise for at least six months after resolution of myocarditis. Return to activities requiring near maximal effort any sooner is associated with increased overall mortality.

Prognosis
Overall, the prognosis of myocarditis is good with survival rates reported between 60-93 percent at one year. Although the mortality rates are higher in patients with more severe illness at presentation and for those who need inotropic as well as mechanical support, those surviving an acute fulminant myocarditis have a better long term prognosis. The mortality is higher in infants (33-45 percent).16 Patients who survive can take months to recover spontaneously. For patients who do not improve, progression to heart failure may occur either acutely or take several years, with continued histological changes predisposing to a higher risk of developing chronic myocarditis.

Of those requiring mechanical support, up to 46 percent of patients progress to a dilated cardiomyopathy and up to 8 percent require cardiac transplantation. Sudden cardiac death remains a feared complication and reportedly occurred in 57 percent of patients with myocarditis on whom an autopsy was performed. This has led certain
sudden infant death studies to link viral myocarditis as an etiology of sudden infant death syndrome. Despite spontaneous recovery, patients require long term follow up due to concern for chronic complications of heart failure, arrhythmias, conduction abnormalities and cardiomyopathy.

Summary
Viral myocarditis should be on the differential diagnosis for all children who present acutely ill, especially in lieu of a preceding viral illness and have new onset respiratory issues that do not respond to conventional therapy and/or have new onset cardiac findings on exam. With such a wide variation in clinical presentation, viral myocarditis is often misdiagnosed leading to a delay in treatment. Although, the outcome is generally favorable, delay in supportive care with pharmacological and/or mechanical cardiopulmonary support as needed, may lead to a worse prognosis of the disease.

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Depression and Pregnancy: Treatment Considerations

By Jeremy Daniel, PharmD, BCPS, BCPP

It doesn’t take that long searching the internet to find mixed “evidence” on the use of antidepressants in pregnancy. There are countless blogs with stories about terrible adverse reactions that happen to babies of mothers who take antidepressants during pregnancy followed by numerous comments calling them life-savers. It is extremely important for providers, pharmacists, and other health care workers who have direct contact with patients to be familiar with the evidence behind psychotropic use, especially as new articles are published frequently.

New fuel for those against the use of antidepressants in pregnancy was published in JAMA Pediatrics in February 2016 when a population-based cohort study of 145,456 singleton full-term infants demonstrated a hazard ratio of 1.87 (95 percent CI 1.15-3.04) for developing an autism spectrum disorder if the mother took antidepressants during the pregnancy. The article further concluded that selective serotonin reuptake inhibitors (SSRIs) were the largest risk of all antidepressants, especially during the second and third trimester. There were many limitations to the article, however, and they started with the background characteristics. Mothers who took antidepressants were more likely to be older, have a lower education level, live alone (less support), have a lower socioeconomic status, be diagnosed with a psychiatric disorder, develop gestational diabetes or hypertension, and deliver a baby with a lower birth weight. Only some of these differences were statistically adjusted, and as it is known these are all risks for the development of autism. This left possible doubt that SSRI exposure is a true signal for autism spectrum disorder development.

From the time this article made the nightly news on the three major networks until now, there have been several other studies published looking for the same link between antidepressant exposure and autism. The most recent was published in the British Medical Journal in July of 2017. An observational prospective cohort study of 254,610 individuals living in Sweden were grouped into three cohorts including mothers with no mental health diagnosis and no antidepressant use, mothers who took antidepressants, and mothers who had a mental health diagnosis but used no antidepressants. The results of the study demonstrated a 1.45 adjusted odds ratio (95 percent CI 1.13-1.85) of developing autism with antidepressant exposure. Propensity score matching eliminated many of the potential sources of error discussed in the 2016 JAMA Pediatrics study. The authors concluded “the association between antidepressant use during pregnancy and autism might not solely be a byproduct of confounding” and while this appears to be true, the authors also comment on the small number of autism cases in the study and thus the small number of preventable cases. This study is currently the strongest possible link between SSRIs and autism.

With this new potential link, it is also important to focus on the benefits. It is common medical knowledge that untreated mental illness during pregnancy results in increased fetal complications, risk for malformations, and potential increased suicidality in the mother secondary to regular hormonal changes during pregnancy. Thus, treating depression in the mother is key for healthy delivery as 14-23 percent of pregnant women experience a depressive episode and up to 70 percent of pregnant women report depressive symptoms according to the American Congress of Obstetricians and Gynecologists (ACOG).

The ACOG guidelines recommend psychotherapy for the management of mild or moderate depression as defined by various standardized rating scales. In patients with moderate to severe depression, or with a history of recurrent depressive episodes, antidepressant therapy is recommended. Several medication management principles guide treatment: monotherapy management is preferred over polypharmacy, use the lowest effective dose to minimize risk, taper the medication close to delivery to minimize neonatal serotonin withdrawal syndrome, and medication selection should be based on a history of...
efficacy (past antidepressant trials). In a patient with no previous antidepressant exposure, sertraline and citalopram are the two preferred agents due to their pharmacokinetic profiles. It should be noted that of the 29 antidepressant medications, 27 are listed as pregnancy category C (paroxetine listed as a category D, phenelzine is unknown) meaning risk cannot be ruled out, so almost any agent could potentially be used when considering patient-specific factors. However, these pregnancy categories will be completely eliminated by June 29, 2020 as the new pregnancy requirements are phased in by the Food and Drug Administration due to concern for the ambiguity in letter classification.

As health care professionals, we know talking about studies to patients often leads to a “glazed over” look or “tuning out” after a few minutes, and with the elimination of pregnancy categories to demonstrate potential relative risk to patients, risk/benefit discussions may become more difficult. Couple this with the increase in technology and information available to the population, and the need for patient-friendly, accurate information becomes paramount. There are two reputable sources to provide to your patients. The first site is called “Treating for Two” and is run by the Centers for Disease Control and Prevention. A second source is “Mother To Baby” run by the Organization of Teratology Information Specialists (OTIS). Both of these sites provide non-biased information about specific medications written in non-medical language.

Overall, antidepressants currently appear relatively safe during pregnancy compared to the potentially disastrous outcomes if depression is left untreated. Psychotherapy is indicated for those with less severe depression while reserving medication options for those with severe depression or recurrent episodes. Choose sertraline or citalopram in the treatment naive patient, or consider continuing a previously effective therapy in those with medication history if their medication is considered safe. It is necessary to have a risk and benefit discussion with every pregnant patient, and providing them with reliable, evidence-based sources for personal review is key to building a strong provider-patient relationship and ensuring the best outcomes for the mother and baby.

### References


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Jeremy Daniel, PharmD, BCPP, BCPP
Assistant Professor, College of Pharmacy and Allied Health Professions, South Dakota State University; Psychiatric Clinical Pharmacist, Avera Behavioral Health.
A friend gave me feedback a few days ago, “Some people say you are arrogant, but I tell them ‘No – he is just self-confident.’” He followed, “We need that in a doctor, but, you know, there is a fine line between those two characteristics.”

Uff da! That threw me for a bit. After thinking about it, I took it as an honest comment and a chance for me to improve myself. A physician/philosopher once said: “A true friend will help you grow by pointing out your warts. Instead of getting angry, one should take it as an opportunity to get better.”

What is arrogance? The dictionary’s definition is harsh: “An offensive display of superiority or self-importance; overbearing pride.” I see it in people who treat others poorly, especially those who are lower on some hierarchal level. Examples would include an employer who expects sexual favors of some kind, a prison guard who harasses a prisoner hatefully, a teacher or parent who supervises a student or child unjustly, or, to make the point, a doctor who treats a nurse or patient poorly. I believe nothing indicates the true color of an individual more than how he/she treats someone who may be lower on the totem pole.

I have seen examples of physicians acting this way: when a surgeon threw a scalpel across the room, when a specialist spoke negatively to the patient about a primary care physician, and when a surgical resident treated a young inner-city woman, infected by gonorrhea, with disdain and contempt. I am not proud that my profession probably deserves some of its reputation for being arrogant. On the other hand, part of the value provided by a physician comes from the sense that she or he is competent and knowledgeable. A humble physician is one thing, but an unsure and uncertain doctor is another.

Perhaps sometimes I have come off as a know-it-all. I need to work on that because I do not know it all. In fact, it seems the more I do know, the more I realize my inadequacies. My folks came from humble backgrounds, and I was drilled on the Golden Rule. The last thing I want to do is to portray myself as a physician who thinks he is more important than anyone else. Rather, I would like to be known as someone who is both competent and cares.
Small Providers Benefit in Quality Payment Program Year Two

By Holly Arends, CHSP, CMQP
Program Manager, Great Plains Quality Innovation Network

The Medicare Access and CHIP Reauthorization Act of 2015 (MACRA) established the Quality Payment Program (QPP) for eligible clinicians. Under the QPP, eligible clinicians can participate via one of two tracks: Advanced Alternative Payment Models (APMs) or the Merit-based Incentive Payment System (MIPS). The Centers for Medicare & Medicaid Services (CMS) began implementing the QPP through rulemaking for calendar year (CY) 2017. CMS released a final rule with comment period to provide updates for the second and future years of the QPP.

In addition, CMS is issuing an interim final rule with comment period that addresses extreme and uncontrollable circumstances MIPS eligible clinicians may face as a result of widespread catastrophic events affecting a region or locale in CY 2017, i.e., Hurricanes Irma, Harvey and Maria.

These provisions of the final rule with comment period and interim final rule with comment period are effective on Jan. 1, 2018. Comments must be received no later than 5 p.m. ET on Jan. 2, 2018. Providers are encouraged to comment on these rules to ensure their voice is heard and their perspective is considered in future regulations. CMS has been listening to the received comments. New CMS Administrator Seema Verna announced two new initiatives at the Health Care Payment Learning Action Network Summit in October of this year. The initiatives are Patients Over Paperwork and Meaningful Measures, both of which were based on the final rule comments received.

Patients Over Paperwork is a cross-cutting, collaborative process that evaluates and streamlines regulations with a goal to reduce unnecessary burden, increase efficiencies and improve the beneficiary experience. The changes to year two of QPP included in this initiative are as follows: (1) An increase in the low-volume threshold – Individual MIPS eligible clinicians or groups with less than or equal to $90,000 in Part B allowed charges or less than or equal to 200 Part B beneficiaries are excluded from the 2018 participation year; (2) Concessions for extreme and uncontrollable circumstances – Natural disasters and hurricanes, among other circumstances, for both 2017 and 2018 are addressed and concessions are provided for those effected providers; (3) Virtual group option for year 2 – Solo providers and groups with 10 and under eligible clinicians will have the option to submit aggregate data as a virtual group; (4) Easier clinician qualification – Criteria for qualifying for incentive payment through participation in APMs that begin or end in the middle of the year will be easier for participating clinicians.

The first year of QPP, 2017, was transitional and offered flexibilities to ensure eligible clinicians could participate. In year 2, CMS is keeping some of those flexibilities and building upon them by offering more bonuses and flexibilities for small, rural providers. The new and continuing options for small practices, 15 or fewer clinicians, are as follows: (1) An increase in the low-volume threshold (see previous description); (2) Ability to join virtual groups – CMS has developed a toolkit to assist practices; (3) Small practice adjustments – The addition of five bonus points to the final score of small practices; Continue to award small practices three points for measures in the Quality performance category that don’t meet data completeness requirements. That requirement was 50 percent in 2017 and will be 60 percent in 2018; (4) Clinician exceptions – Providers practicing in ambulatory surgery centers, hospitals and groups with 15 or fewer clinicians can apply for a hardship exception for the Advancing Care Information performance category; MIPS eligible clinicians with an EHR decertified by the Office of the National Coordinator may also apply for exception; (5) New weight of the Cost Category in the MIPS track – In 2017, the Cost Category was set at 0 percent. For 2018, the category will be weighted at 10 percent of the final MIPS score. This is a significant change that will require understanding of how CMS looks at costs. Providers are encouraged to access the feedback reports provided by CMS. The Quality Resource Use Report is available using the CMS Enterprise Identity Data Management account.

CMS and the Great Plains Quality Innovation Network (QIN) know healthcare professionals and organizations feel the strain of trying to adhere to CMS program requirements. Due to that reality, the Great Plains QIN makes it their mission to provide assistance to all Medicare providers to be successful in these programs.
In Memoriam 2017

By Rev. Mark Tracy

Bob Johnson, SDSMA CEO from 1972-1999

We have lost a dear loved one. Robert D. Johnson passed away on Nov. 27, 2017 at Sanford Ava’s Hospice House in Sioux Falls. In the days following Bob’s death, I spoke to some incredible folks who knew Bob. Dr. Mary Carpenter, a past president of the SDSMA, said she’ll miss this great gentleman who was an incredible leader and visionary who had an amazing insight for looking forward. Those who knew Bob describe him as a man who had complete dedication to the physicians of South Dakota. Another past SDSMA president, Dr. Steve Schroeder, told me that he never ran across anyone who didn’t trust Bob’s judgement on issues. Jan Anderson, former SDSMA vice president, said Bob was the best boss and coworker anyone could have. His laugh was hearty and his smile was genuine. The one thing that everyone said about Bob was that Bob loved doctors. Bob believed in doctors.

When Dr. Robert L. Ferrell came to South Dakota and first applied for his medical license, the SDSMA, the Foundation, and the SDBMOE were all under one roof with a total of four employees, including Bob. Dr. Ferrell said it was obvious that the organization was run like a business, and he was impressed. Under Bob’s leadership, the SDSMA became one of the most respected and powerful medical associations in the country. In addition, Bob developed the concept and idea behind what would become DAKOTACARE.

Bob was a man who was kind and polite and never talked bad about anybody – not that he would back away from talking about things he disagreed with. His son, Chris, said Bob “liked the argument more than the fight.” He loved getting together with intelligent people to debate politics and healthcare. Bob’s family was unanimous – debate was a serious hobby for Bob. His wife, Teri, said Bob had a firm belief that people should get together with one another and that will build community and true, real friendships. That’s what makes an organization strong. This was the core principle upon which the SDSMA blossomed. We’ll miss this man who took his work seriously, who was a great leader, and who liked to have fun.

Honoring physician members of the SDSMA who passed away in 2017

[Images of physicians]
What is a confidential monitoring program?
When an applicant or a licensee has been determined by an evaluation to be impaired in a manner where the individual demonstrates the inability to practice in their health-related profession with reasonable skill and safety due to mental health issues, physical issues, or substance use related disorders (alcohol or drug abuse, dependency, or addiction), the applicant or licensee is enrolled in a confidential monitoring program. The confidential monitoring program is an agreement between the participant (applicant or licensee) and the BMOE staff review panel to defer any recommendation for discipline on a license as long as the participant can be monitored to ensure their ability to safely practice. See the flowchart graphic for the process.

What is the purpose of the BMOE staff review panel?
The review panel administers the program for the individual. The participant’s case stays at the staff level and does not go to the full BMOE unless the participant is unable to comply with the MBMP. The review panel consists of the BMOE executive director and one BMOE board member who will, in effect, “be considered one of the staff” in order to make the recommendation as to whether the individual is eligible for the MBMP.

What are the eligibility requirements for the MBMP?
The MBMP monitors impaired healthcare providers. The potential participant will have undergone an evaluation that demonstrates their inability to practice in one’s health-related profession with reasonable skill and safety due to mental health issues, physical issues, or substance use related disorders (alcohol or drug abuse, dependency, or addiction).

Do I need to enroll in the MBMP if I already participate in a monitoring program administered by my employment or other entity?
You can continue in your current monitoring program without enrolling in the MBMP. However, you must report your participation in any and all monitoring/wellness programs, other than the MBMP, on or before you submit your annual license renewal application. You also need to be aware that the MBMP is the only state BMOE approved confidentially protected program for South Dakota licensees.

I am licensed in another state and am enrolled in that state’s monitoring program. Now I am also licensed in South Dakota where I currently practice. Do I have to enroll in the MBMP, and how do I keep my previous monitoring program informed that I am in compliance?
Yes, you do need enroll in the MBMP as it is the only state BMOE approved monitoring program. The MBMP will then contact your previous monitoring program and send reports regarding your compliance.

Do the BMOE members know who is in the MBMP?
The MBMP participation list is only known to designated BMOE staff and the BMOE investigative review panel. The BMOE members do not know who is in the MBMP except for the one board member who is assigned to the BMOE.
investigative review panel. All monitoring will remain confidential to the BMOE board members as long as the participant is compliant and doing well in the monitoring program.

**Does the public know who is in the MBMP?**
An individual’s participation in the MBMP is confidential to the public as long as the participant is in compliance with program requirements. The public does not have access to information that would identify participants in the program, except in rare cases where the BMOE staff files formal disciplinary charges against a participant which may include noncompliance with an MBMP contract.

**Who administers the MBMP?**
The MBMP is administered by BMOE staff and the established review panel pursuant to authority granted by administrative rules promulgated by the BMOE.

**What happens when a licensee or applicant self-reports?**
The MBMP monitors participants struggling with impairment issues related to substance abuse, mental health issues, or physical disability, and is not a disciplinary program. The MBMP considers a licensee’s or applicant’s self-report to be a positive first step toward bringing a potentially harmful situation under control before their professional reputation is damaged.

The MBMP will gather information and make referrals for evaluation as needed. The MBMP then works with the licensee or applicant to put the required supports in place to ensure the participant is able to continue to practice safely. The majority of individuals participating in the program are actively practicing.

**Who evaluates the potential participants?**
Potential participants are evaluated depending upon the case history of each individual. Some individuals may self-report or apply to the MBMP after having been involved in the judicial system; for example, a DUI or other incident where evaluations are already available. In other cases, there may be questions as to whether a diagnosis indicative of impairment exists. That individual would be referred to the appropriate evaluator or evaluation team prior to entering the confidential MBMP.

**Who treats the participants?**
Once a determination of impairment is made and the participant enters the MBMP, the participant chooses a medical and support team. The MBMP participant is an active participant with their medical and support team. As determined by the previously mentioned evaluation recommendations, this team is comprised of a monitoring physician or other healthcare provider; a monitoring therapist, psychologist, or counselor; a work-site monitor, and an aftercare monitor. The medical and support team will be approved by the MBMP.

**Is it a good idea to have the licensing board administer the program?**
The BMOE has a legal obligation and mission to ensure safe medical practice for the protection of the public who seek professional medical services within the State. The BMOE has promulgated administrative rules that provide a transparent process for the investigation and regulation of medical practice while providing due process rights to applicants and licensees. While the process is transparent, participation remains confidential. Healthcare facilities have immunity when reporting employees to the BMOE staff.

**Who is the contact person?**
For the convenience of the licensee or applicant, one person on the BMOE staff is designated for contact purposes. Separate and dedicated phones, both mobile and land-line with a toll-free phone number option, are available. A separate and dedicated email is also accessible. A separate and dedicated website is being developed.

Please contact:
Randi Sterling
Medical Board Monitoring Program (MBMP)
Phone: 605-367-7700 | Toll free: 888-340-4371 |
Cell: 605-400-4542
Email: mbmp@state.sd.us

**Who answers general questions?**
Any general questions should be directed to the BMOE executive director, Margaret Hansen.

**Summary**
The BMOE has a long history of monitoring its licensees. The BMOE has agreements in place with the monitoring companies, Affinity eHealth and Soberlink to serve the MBMP. The BMOE is continuing to research other regional and national monitoring programs including protocols, processes, budgets, and program services, to identify best practices.
### UNITED STATES SENATORS

**MIKE ROUNDS (R)**  
Office: 502 Hart Senate Office Building  
Washington, DC  20510  
Phone: (202) 224-5842  
Website: rounds.senate.gov

**JOHN THUNE (R)**  
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Phone: (202) 224-2321  
Website: thune.senate.gov

### UNITED STATES REPRESENTATIVE

**KRISTI NOEM (R)**  
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Washington, DC  20515  
Phone: (202) 225-2801  
Website: noem.house.gov

### SOUTH DAKOTA GOVERNOR

**DENNIS DAUGAARD**  
Office: State Capitol; 500 E. Capitol Avenue; Pierre, SD  57501  
Phone: (605) 773-3212  
Website: sd.gov/governor

### SOUTH DAKOTA LIEUTENANT GOVERNOR

**MATT MICHELS**  
Office: State Capitol; 500 E. Capitol Avenue; Pierre, SD  57501  
Phone: (605) 773-3661  
Fax: (605) 773-4711

### SOUTH DAKOTA SENATE

State Capitol, 500 E. Capitol Ave, Pierre, SD  57501  

- **President Pro Tempore**  
  Sen. Brock Greenfield (R)

- **Majority Leader**  
  Sen. Blake Curd (R)

- **Asst. Majority Leader**  
  Sen. Ryan Maher (R)

- **Majority Whips**  
  Sen. Kris Langer (R)  
  Sen. Al Novstrup (R)  
  Sen. Bob Ewing (R)

- **Minority Leader**  
  Sen. Billie Sutton (D)

- **Asst. Minority Leader**  
  Sen. Troy Heimert (D)

- **Minority Whips**  
  Sen. Jason Frerichs (D)

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### SOUTH DAKOTA REPRESENTATIVES

State Capitol, 500 E Capitol, Pierre, SD  57501  

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- **Speaker of the House**  
  Rep. Mark Mickelson (R)

- **Speaker Pro Tempore**  
  Rep. Steven Haugaard (R)

- **Majority Leader**  
  Rep. Lee Qualm (R)
### 2018 South Dakota Legislative Directory

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Listed below are the SDSMA district medical societies and the state legislative districts contained within each medical society.

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For Your Benefit

Get Involved in the SDSMA

Thank you for your membership in the South Dakota State Medical Association. As a member, you have several direct opportunities to become more involved in the important work of the SDSMA.

- Doctor of the Day – serve as the physician for the South Dakota State Legislature for one day during session;
- Physician lobbyist – serve as a volunteer lobbyist during the legislative session;
- SDSMA PAC – help friends of medicine become elected officials and lawmakers;
- SDSMA committees and task forces – serve the organization and yourself;
- SDSMA appointments to state boards, committees and commissions – the SDSMA is asked to nominate and recommend physicians to fill positions to numerous vacancies every year;
- Districts and specialty societies – for local involvement.

If you’d like to get involved, give us a call at 605.336.1965 or visit www.sdsm.org for more information.

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

Legal Brief Highlight: Closing a Medical Practice

Before closing a practice, notify your patients and other professional organizations of the closure, and plan for proper handling of medical records and remaining prescription drugs. Plan enough time to handle internal accounting, legal and employee-related matters.

Active patients must be given adequate time to locate another physician. If possible, notify your active patients two to three months before your closing date. It is a good idea to run an ad in the newspaper to announce the closing of the practice for the benefit of inactive patients and others. Take care in maintaining the confidential nature of the physician-patient relationship. For example, unless required by law, physicians may not transfer copies, summaries of records or other information in a person’s medical records to anyone without the patient’s prior written permission.

More information is available in the SDSMA legal brief, Closing a Medical Practice at www.sdsm.org. Through the SDSMA Center for Physician Resources, the SDSMA has developed more than 50 legal briefs that are available to members. In addition, the Center develops and delivers programs for members in the areas of practice management, leadership and health and wellness.

SDSMA Launches Website on Prescription Drug Abuse

The SDSMA is working with the South Dakota Department of Health to reduce prescription drug diversion, abuse and overdose deaths.

To aid in the battle against this ongoing epidemic, the SDSMA has launched a website at www.sdsm.org for both providers and patients to learn more about prescription drug diversion and abuse, addiction, and recovery/treatment services. Information on pain management, alternatives to controlled substances and drug take-back programs is also available.

Preventive actions, treatment for addiction, and proper response to overdoses can help patients struggling with addiction. We are excited to be partnering with the South Dakota Department of Health to increase awareness among not only physicians, but patients, too. It is important to remember that addiction is a disease, but it can be overcome – people struggling with addiction can, and do, recover.

We hope the physicians, patients and families of South Dakota find this information helpful.

We encourage all prescribers to take a look at the information that we have made available to you and to assess your individual practice. While South Dakota continues to have one of the lowest numbers of opiate deaths when compared to other states, prescription drug abuse is a problem to which South Dakota is not immune, and without the help of physicians, this epidemic will worsen.
Final Rules on QPP and Physician Fee Schedule Released

The Centers for Medicare and Medicaid Services (CMS) has released both the final 2018 Quality Payment Program rule and the final 2018 Physician Fee Schedule rule. Combined, the regulations are approximately 3,000 pages in length.

The QPP Final Rule continues to build upon CMS’ transition year policies and address elements of the Medicare Access and CHIP Reauthorization Act of 2015 (MACRA) that were not included in the first year of the program. It also includes an interim final rule with comment period to establish an uncontrollable circumstance policy for the 2017 MIPS performance period that recognizes recent hurricanes Harvey, Irma, and Maria, and other natural disasters that could effectively impede a Merit-based Incentive Payment System (MIPS) eligible clinician’s ability to participate in the program.

CMS makes payments under the 2018 Physician Fee Schedule for services furnished by physicians and other practitioners in all sites of service. The Final Rule’s overall update to payments based on the finalized 2018 rates will be +0.41 percent. The Final Rule also includes the addition of several codes to the list of telehealth services and the elimination of a telehealth reporting requirement.

For the QPP and PFS regulations in their entirety, visit www.cms.gov.

Source: CMS

New Guidelines Lower Definition of High Blood Pressure

Nearly half of American adults, 46 percent, will be determined to have hypertension under a new clinical guideline on the prevention, detection, evaluation and management of high blood pressure (BP), up from 32 percent under the old benchmark. Previously, high blood pressure was defined as BP readings persistently at or above 140 mm Hg systolic or 90 mm Hg diastolic, but is now defined as persistently at or above 130/80 mm Hg.

This new guideline does not mean these newly classified patients with hypertension will face dramatic new risks or that physicians need to immediately begin medication treatment for most. The guideline is meant to prevent strokes, heart attacks and other cardiac problems through earlier action—a combination of lifestyle changes for all of these patients, and medications for some, depending on the circumstances—to control high BP.

The 2017 hypertension guideline comes from a joint task force formed by the American College of Cardiology (ACC) and the American Heart Association (AHA). The ACC and AHA partnered with many other organizations representing physicians and other health professionals to create the new guideline.

Read more about the new guidelines in this month’s editorial.

Source: AHA

Nominate a Colleague for 2018 SDSMA Awards!

Nominations for the 2018 SDSMA Awards are now being accepted. Nomination forms are available at www.sdsma.org.

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

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

The SDSMA is seeking award nominations for the following awards:

- Distinguished Service Award
- Community Service Award
- Young at Heart Award
- Outstanding Young Physician Award
- Media Award

Visit www.sdsma.org to review each award and complete an awards nomination form. Those with questions may contact Elizabeth Reiss at ereiss@sdsma.org.

Nominate someone today and help your colleagues and supporters get the recognition they deserve!
Members of the South Dakota State Medical Association (SDSMA) and the SDSMA Medical Student Section attended the American Medical Association (AMA) Annual Meeting in Honolulu in November with hundreds of others from across the country. The gathering was filled with activities and policy debate that will help shape the future of health care in the nation. Some policies adopted by the AMA House of Delegates (HOD) include the following:

**Oppose changes to Medicaid**

Delegates adopted policy opposing Medicaid changes that would undermine essential health benefits requirements. The policy opposes the removal of categories from the essential health benefits package and waivers of essential health benefits requirements that could lead to the elimination of protection against annual or lifetime benefit limits and out-of-pocket expenses. Several legislative proposals considered in Congress in 2017 included provisions that would allow for changes to essential health benefits, such as allowing states to waive them. These approaches, according to the reference committee’s summary, “could have caused a significant number of Americans to lose access to affordable health insurance coverage.”

**Eliminate burdens for controlled substances’ e-prescribing**

Barriers to implementing e-prescribing of controlled substances remain a significant burden for physicians. To further the AMA’s work in eliminating restrictions, the HOD modified current policy to “continue to advocate…for elimination of cumbersome, confusing and burdensome requirements relating to electronic transmission of physicians’ controlled substance prescriptions to pharmacies.”

**More Medicaid help sought for Puerto Rico, U.S. Virgin Islands**

It has been nearly four months since Hurricanes Irma and Maria, but Puerto Rico and the U.S. Virgin Islands continue to struggle with recovery. To help with disaster relief, the HOD directed the AMA to urge Congress to quickly pass legislation to adequately fund the islands’ Medicaid programs. To help restore access to health care services, the AMA also will encourage the Centers for Medicare & Medicaid Services to implement temporary emergency regulatory Medicare and Medicaid funding waivers.

**Protect clerkship spots**

There is an increased need for clerkship spots for U.S. medical students. The AMA passed a policy calling for greater capacity. The policy advocates for federal and state legislation to provide additional funding to support infrastructure and faculty development, capacity for medical school expansion, clinical clerkships and other educational experiences. The policy speaks to concerns about the availability of clinical clerkship training sites due to increases in the enrollment of U.S. allopathic and osteopathic medical schools, the growing number of U.S. medical schools, and the rising number of foreign medical schools that seek to place their students in clerkships in U.S. institutions.

**Shine light on why drugs cost so much**

The AMA will seek to ban unjustified prescription-drug “price gouging” and let pharmacists tell patients when their co-pay exceeds a drug’s cash price. Under the new AMA policies, federal and state advocacy efforts to further drug price transparency will be expanded that take drugmakers, pharmacy benefit managers, and health insurers to task for their roles they play in rising drug costs and less access to affordable treatment.

Respectfully submitted,

Mary S. Carpenter, MD  
SDSMA Delegate to the AMA  

Robert L. Allison, MD  
SDSMA Alternate Delegate to the AMA
The University of South Dakota’s Physician Assistant Program invites applications for a full-time Physician Assistant faculty position within the School of Health Sciences. Diversity and inclusiveness are values that are embraced and practiced at the University of South Dakota. Candidates who support these values are encouraged to apply.

This individual will have the opportunity to teach in a dynamic, accredited, 24 month Master of Science in Physician Assistant Studies program. The position will work in cooperation with the faculty to create and maintain an exceptional educational program for students that includes teaching, scholarly activity, and community engagement. The position is open to a Physician Assistant or Physician.

Review of applications will begin immediately and continue until filled. For further position details or to apply on-line: https://yourfuture.sdbsr.edu Posting Number 0009105

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