PANHANDLE HEALTH

A QUARTERLY PUBLICATION OF THE POTTER-RANDALL COUNTY MEDICAL SOCIETY

SPRING 2024 | VOL 34 | NO.2

Update in Cardiovascular Diesease

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President's Message

by Nicole Lopez, MD, FAAFP

County medical societies work together in meeting the needs of physician members at both the local and state level. There are currently 112 local medical societies in Texas. The Potter-Randall County Medical Society (PRCMS) was organized in 1903 by 18 local physicians. It currently consists of 352 active physicians, both primary care and specialists, as well as residents in training. The mission statement of the PRCMS is to "nurture the continued advancement of scientific knowledge and to maintain unity and harmony among all members of the profession."

Local medical societies also promote the art and science of medicine and the betterment of public health. The next issue of Panhandle Health (after this one) will explore how art in its many forms relates to the values and practice of medicine, as well as specific articles on the art of certain cultures. If you find this appealing and have any interest in writing, Dr. Steve Urban, our Editor-In-Chief, would love to hear from you (email address: steven.urban@ttuhsc.edu).

Medical societies also have a tradition of activism on behalf of practitioners. This fall, our local chapter hosted a CME event that featured Texas Medical Association president, Dr. Rick Snyder, who provided a well-attended legislative update. Dr. Snyder returned to Amarillo in January along with TMA Executive Vice President and CEO, Michael Darrouzett, for our Annual Meeting and officer installation ceremony.

The PRCMS elects delegates each year to represent the Panhandle in the TMA House of Delegates. As part of TMA's largest caucus, the Lone Star Caucus, our members advocate for issues that affect our state, physicians, and members of our community, working with our colleagues from around the state to advance TMA's mission to improve the health of all Texans. Last year, access to meaningful health care coverage, maternal health, fighting scope of practice creep, working to address the opiate and mental health crises, and physician burnout were all key issues that our delegates helped to address.

The PRCMS also supports addressing important issues impacting healthcare in the Amarillo area, such as protecting the physician-patient relationship and promoting public health. Our Medical Alliance supports our mission and participates in TMA's Hard Hats for Little Heads program every year at Christmas.

Our society is planning an event this April for women physicians in Amarillo as an opportunity to network with other female colleagues in our community. We hope to meet at least twice each year to help build these important relationships. Our retired physicians group also is very active and plays an important role in our society. If you have not read the Winter issue of Panhandle Health, I encourage you to take a look, as it focuses on many respected physicians in Amarillo who have contributed greatly to our community.

I hope that you will consider becoming more active in our local county medical society, and I would welcome inquiries about participation in the Board of Directors from those who are interested.

Happy Spring!

Our Next Issue Of *Panhandle Health* Features:

Medicine and the Visual Arts

Executive Director's Message

by Cindy Barnard, Executive Director

This issue of Panhandle Health features Cardiovascular Diseases. The World Health Organization ranks Cardiovascular Disease as the leading cause of death globally, taking an estimated 17.9 million lives each year. It is a devastating health problem in our community as well. The Spring 2024 issue of Panhandle Health includes articles on common cardiovascular conditions such as heart failure, coronary artery disease, congenital heart disease, peripheral arterial disease, and several more, written by some of our community's leading practitioners. We hope that you enjoy it and that you learn and profit from it.

On March 30th, we will celebrate Doctor's Day, which was first observed in 1930. A physician's wife, Eudora Brown Almond, decided to declare a day in honor of all doctors. The red carnation was chosen as the symbolic flower for National Doctors Day.

If you are one of the few physicians who have not paid your dues for 2024, note that the DROP DATE was MARCH 1, 2024. The physicians roster goes to press shortly thereafter and will include only members whose dues are current.

Finally, PRCMS appointments to our Boards and Committees are now ongoing. If you are interested in serving on one of these, please call the Society office at 806-355-6854 or email prcms@suddenlinkmail.com. The core of the Society is its volunteers. We truly need you.

Message from the Potter-Randall County Medical Alliance

by Alena Martin, President & Madeline Lennard, Co-President

A changing of the guard has occurred at the Alliance, and Madeline Lennard and I are excited to continue Tricia's good work to serve and support the Potter-Randall medical community alongside the board.

Planning is underway for a fun year of Alliance activities. Please join us on March 13 from 5:30-7:30 at Amarillo County Club to congratulate the 2024 medical school class on their residency matches and encourage them to return to Amarillo to practice. We would love to have as many specialties as possible represented. If you can make it, please come! We are also looking forward to celebrating Doctor's Day at the end of March.

If you and your spouse/partner are not current alliance members, please visit www.texasmedalliance.org to join and take advantage of the auto-renewal option. We love a one-and-done moment! There's no better way to support our local physicians, medical students, and their families!

Guest Editorial

by Sheryl Williams, MD

The CDC publishes yearly morbidity and mortality data through the National Center for Health Statistics. During the throes of the pandemic, we watched as the mortality numbers for Covid-19 rose precipitously, up to 416,893 deaths in 2021 (with provisional data for 2022 still pending publication). Yet, despite the horrible loss of life, Covid was not the major cause of mortality. Unfortunately, cardiovascular disease continues to be the number one cause of death in the United States, with the CDC attributing 695,547 lives lost in 2021. Globally, WHO data show that heart disease accounted for a third of all deaths prior to Covid-19. This issue of Panhandle Health is dedicated to exploring new concepts, therapies, and prevention strategies for treating and managing our number one health threat.

Although the problem is sometimes overlooked, children suffer from congenital and developmental cardiovascular disease. Dr. Srilatha Alapati and resident Dr. Sangeeta Dammu from Texas Tech have provided us with an extensive review on exercise recommendations for children with congenital heart defects.

Prevention is key to reducing the incidence of cardiovascular disease in the first place. We have an excellent article by Rachel Hutto delving into the complexities of diet and heart health, with specific recommendations from the American Heart Association Dietary Guidance for Cardiovascular Health. Along those same lines, Dr. Alan Keister has detailed the Primary Care Physician's role in cardiovascular health and prevention. However, not all cardiovascular disease is mediated by atherosclerosis. Dr. Scott Milton reviews the continuing impact of rheumatic fever and rheumatic heart disease on global morbidity and mortality from heart disease. And not to be ignored is the role of peripheral artery disease (PAD) as a marker for coronary artery disease. Dr. David Langley has contributed a detailed review of risk factor reduction and treatment for PAD.

Our local cardiologists have contributed to this issue by detailing their approaches to new and improved treatment modalities. Dr. David Brabham has a specialized clinic for congestive heart failure patients and details their approach for the major types of heart failure. Matt Parker, Vice President of Cardiovascular Services at BSA, provides an up-to-date account of the role that percutaneous transcatheter techniques are playing in the management of aortic stenosis. Dr. Rajesh Nambiar and his medical student co-author Jithin Kurian review the devastating occurrence of sudden cardiac death, as well as methods of treatment and prevention. Dr. Paresh Rawal describes a patient with refractory angina and unfavorable anatomy who responded to complex intervention, requiring support with the Impella® heart pump.

Finally, long-term contributors Dr. Rouzbeh Kordestani and Dr. Steve Urban take us on a journey into the history of the cardiac catheterization procedures. Rounding out the issue, our Texas Tech Internal Medicine resident representative, Dr. Basak Basbayraktar, has put together a case report and literature review of DC cardioversion and sinus node dysfunction.

Cardiovascular disease is the elephant in the room when discussing the most prevalent mortality threat in our nation and the world. New advances are being made every year to help prevent and treat the many faces of this chronic condition. We hope you enjoy this issue.

Management of Heart Failure in the New Millennium

by David Brabham, MD

Heart failure is a difficult clinical problem. The burden of disease on patients suffering from heart failure and on the healthcare system is high and is increasing as the population ages. Significant resources have been applied to develop therapy and strategies to mitigate the burden of heart failure on the population. Fortunately, over the past decade, there have been advances in the medical therapy, meaningful differentiation of the types of disease, and a better understanding of how to deliver medical care to this population. There are three different types of heart failure that will be discussed: heart failure with reduced ejection fraction (HFrEF), heart failure with preserved ejection fraction (HFpEF), and acute decompensated heart failure (ADHF). A brief review of the most important and most recent literature will be presented. Data providing a scientific rationale supporting full treatment will be presented, showing the benefit of more complete and thorough treatment. Lastly, discussion will focus on how our clinic approaches the complexities of meeting the needs of these patients as their disease progresses.

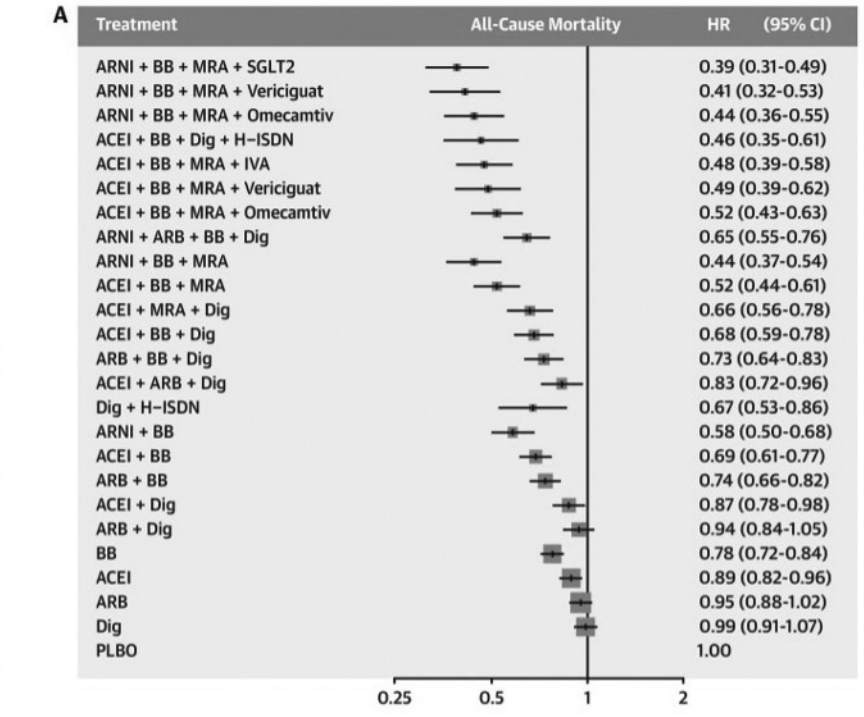
HEART FAILURE WITH REDUCED EJECTION FRACTION (HFrEF)

The impact of heart failure with reduced ejection fraction has been well recognized for decades. There have been steady advances in medical therapy starting in the mid-1980s. How to apply these advances in concert has not been as easy to discern. With choices of 8 to 10 possible medicines for heart failure, these questions arise: which medicine do we start first, how do we combine the therapy, and when have we reached maximal medical benefit from therapy? Difficult questions indeed. The concept of quad therapy has been advanced in the heart failure guidelines (1). Quad therapy includes: evidence-supported beta blocker, renin-angiotensin-aldosterone system blockade, an aldosterone antagonist, and SGLT 2 inhibitors.

Sacubitril/valsartan (Entresto) is the preferred therapy in the RAAS blockade family. Sacubitril has a novel mechanism; it inhibits neprilysin, a member of the neutral-endopeptidase family. Neprilysin is an enzyme that breaks down Brain Natriuretic Peptide (BNP), a peptide that is released as result of increased left ventricular pressure. BNP promotes sodium and thus volume excretion. Initially, BNP gained prominence as a diagnostic tool, first to differentiate heart failure from other causes of shortness of breath, and then as a prognostic tool to assist in guiding care of patients with heart failure. It is clear now that the purpose of endogenous BNP is to assist with fluid balance in the cardiovascular system. Inhibiting neprilysin prevents the breakdown of endogenous BNP, and the higher BNP level increases its benefit to the patient with heart failure. Furthermore, a second action of BNP is promotion of vasodilation, also a benefit to the struggling heart. But the action of sacubitril deleteriously promotes angiotensin (AT) as well; this necessitates the addition of valsartan to block the effect of AT.

The pivotal trial for Entresto was the PARADIGM-HF trial, reported in 2014 (2). Entresto was compared to enalapril in symptomatic patients with ejection fraction of 35% or less. The trial design included a run-in period where patients were given escalating doses of Entresto and then enalapril, so that only patients

CENTRAL ILLUSTRATION: Relative Risk Reduction of Different Pharmacological Treatment Combinations for Heart Failure



Tromp, J. et al. J Am Coll Cardiol HF. 2022;10(2):73–84.

who tolerated the maximum doses of therapy were randomized. The primary endpoint was composite of heart failure hospitalization (HFH) and CV death. After a mean follow-up of 27 months, the primary endpoint was reached in 21.8% of the Entresto group versus 26.5% in the enalapril group for a relative risk reduction of 20% and an impressive absolute risk reduction of 4.7%. Other interesting aspects of the trial were some of the inclusion criteria. New York Heart Association class II (symptomatic with usual activity) comprised 75% of the randomized patients. This emphasizes that this therapy is not designed for patients who have failed other therapies and are in a clinically difficult state but rather for patients who are less ill on the heart failure spectrum, with the goal of preventing progression of this disease. Secondly, it should be noted that patients had to have a systolic blood pressure of greater than 95 mmHg to be randomized in this trial. Finally, the individual end points (HFH and CV death) were each statistically significantly better in the Entresto group.

The guidelines discuss RAAS blockers as one of the four pillars of heart failure treatment. For more than 15 years, angiotensin converting enzyme inhibitors (ACEi) were considered the cornerstone of heart failure treatment. Angiotensin receptor blockers (ARB) were considered to be non-inferior to ACEi. In our practice, the use of an ACEi, namely lisinopril, was the standard for many years (although the benefit appears to be a class effect).. The use of ACEi has generally been preferred over evidence-based ARB, except when cough or other side effects preclude ACEi usage. As for evidenced-based ARB, valsartan and candesartan have the most robust data, with losartan having less robust effect on heart failure outcomes. The use of ACEi or ARB is now relegated in the guidelines to second-line therapy, especially in symptomatic patients. The cost of Entresto has caused some issues with access, especially in the first few years following its FDA approval. We have had significant success helping our patients access this therapy, but it has required directed resources to aid with approval.

Evidence-based beta blockers have been a second cornerstone of HFrEF care since the mid-1990s. The US carvedilol trial, CIBIS and MERIT trials (for carvedilol, bisoprolol, and metoprolol succinate, respectively) showed robust benefit in reducing heart failure hospitalization and CV death, with a fairly consistent magnitude of benefit. Multiple iterations of trials demonstrated relative risk reduction of 25% and absolute risk reduction of 5%, favoring beta blocker therapy. For the most part, beta blockers were tested as monotherapy, as the ACEi trials were conducted separately. It should be clearly stated that beta blockers that are not carvedilol, bisoprolol, or specifically metoprolol succinate should be avoided, as they lack data suggesting benefit. Numerous trials have showed no benefit outside of these 3 specific therapies; so a class effect should not be inferred.

Aldosterone antagonists were added to ACEi and evidence-based beta blockers in the early 2000s. The RALES trial studied spironolactone versus placebo in NYHA class III-IV patients who were on maximally-tolerated ACEi and many who were also on beta blockers (3). The primary endpoint was death from any cause. In this ill population, after only 24 months on the trial, death occurred in 46% of patients receiving placebo and in 35% of patients receiving spironolactone, for a relative risk reduction of 35%--an impressive reduction indeed. In the subsequent EMPHASIS trial (4), class II-IV patients with HFrEF were randomized to eplerenone or placebo and were followed for a median of 21 months. The primary endpoint was a composite of HFH and CV death. In patients receiving placebo, 25.9% experienced the primary endpoint compared with 18.3% of patients receiving eplerenone—also an impressive 37% relative risk reduction. RAA system blockers and beta blockers were concomitantly administered in greater than 90% and 85% of the trial patients, respectively. As a result, aldosterone antagonists have been added as a third pillar of medical therapy in the treatment of HFrEF. There had been some hesitation regarding the use of this important therapy because of the danger of hyperkalemia. In our clinic, we routinely check potassium and creatinine levels on the day we start therapy, the day we change therapy, and a week after either of these.

SGLT2 inhibitors have had an increasing role in reduction of cardiovascular outcomes over the past ten years. Initial pivotal trials were designed to show benefit in glycemic control and potential benefit in cardiovascular outcomes. From data in the initial trials, it became clear there were significant pockets of benefit, especially in preventing heart failure. From this initial data, numerous trials have been conducted to test the benefit of SGLT 2 inhibitors in patients with heart failure, both with and without diabetes.

Sodium-Glucose Transport 2 (SGLT 2) protein inhibitors are the final pillar of quad therapy. In 2020, the results of the EMPEROR-Reduced trial were published (5). Empagliflozin was compared to placebo in symptomatic patients with ejection fraction of 40% or less. After a median trial duration of 16 months, the primary endpoint of HFH or CV death occurred in 19.4% of patients receiving empagliflozin and in 24.7% in patients receiving placebo. There are several important observations from even a cursory review of this data. Despite so-called modern heart failure therapy, almost 25% of patients experienced either HFH or CV death; this brings attention to the significant residual burden of HFrEF despite the existing therapies. Furthermore, there was an additional 5.3% absolute risk reduction in clinically important events despite concomitant guideline-based care. The magnitude of this reduction is similar to the previous cornerstones of care, when patients had little in the way of concomitant therapy. The benefit was seen in patients with and without diabe-

tes. Hypoglycemia was not more common in the nondiabetic patients. The result was even more impressive because of the short follow-up of the trial; the trial was stopped early because of the significant findings.

There are clinical challenges to provide modern medical care to patients with HFrEF. We are tasked with instituting these four pillars of care in patients with variable financial access to these therapies. Furthermore, there is not clear direction on which therapy to institute first and on the stepwise addition of therapy. It is likewise challenging to escalate the dose of each medicine to its maximal observed benefit. Our clinic has an approach to these issues that will be further elucidated.

HEART FAILURE WITH PRESERVED EJECTION FRACTION (HFpEF)

It has been well established that heart failure with preserved ejection fraction causes significant morbidity and mortality. Indeed, several registries have shown that outcomes between HFrEF and HFpEF are similar. Furthermore, treatment for HFpEF has been dominated by negative trials since the study of the disease process was instituted. The cornerstone of treatment for HFpEF has been aimed at the presumed cause, such as hypertension, sleep apnea, or atrial fibrillation, for lack of better data. This past decade, however, has offered several new therapies that have only modest proven benefit but at least offer hope that something might help. The heart failure guidelines have adopted 3 therapies that should be considered, despite this modest statistical benefit: Entresto, SGLT 2 inhibitors, and aldosterone antagonists.

After numerous negative trials, the TOPCAT trial results were received eagerly (6). There was significant hope that aldosterone antagonists might offer good news in the treatment of HFpEF. In this trial, spironolactone was compared to placebo for a mean of 33 months in symptomatic patients with ejection fraction 45% or greater, but the trial was clearly and disappointingly negative. Interestingly, the results differed regionally. It was found that, in eastern Europe and Russia, the benefit was nonexistent but, in Argentina, Canada, Brazil, and the United States, spironolactone showed benefit. In review of the data, it was determined that a metabolite of spironolactone was lacking in around 30% of patients from Eastern Europe and Russia versus only 3% in the Americas. This, along with numerous other inconsistencies that were uncovered, suggested that compliance was entirely different between the two regions. In the Americas, the TOPCAT study showed statistically significant benefit (18% reduction in primary composite endpoint of HFH, CV death or aborted cardiac arrest). The road to understand the inconsistencies was long but ultimately led the conservative US Food and Drug Administration to approve spironolactone for use in HFpEF in 2020.

In the PARAGON trial, sacubitril/ valsartan was compared to valsartan (7). The trial results (reported in 2019) were disappointing. Symptomatic patients were included if the ejection fraction was 45% or greater and if they were able to tolerate high dose therapy in a run-in period. The primary end-point was a composite of HFH and CV death. The hazard ratio was 0.87 with p value of 0.06, narrowly missing statistical significance. The results of this trial initially discouraged the widespread use of this therapy in our clinic.

The results of the EMPEROR-Preserved trial were met with great excitement, as this was the first trial to show statistically significant benefit in the treatment of HFpEF. Patients were included if they had symptomatic heart failure with ejection fraction of greater than 40%. They were followed for a median of 26 months for the primary composite outcome of HFH and CV death. There was a statistically significant reduction in this composite end-point of more than 3% (from 17.1% down to 13.8), a 21% reduction in the sacubitril/valsartan cohort. The end-point was mainly driven by a reduction in heart failure hospitalization, rather than CV death. Again, this was the first trial of a medical therapy to show any statistically significant benefit in this prevalent and difficult-to-treat entity. While the numeric benefit is modest, the length of follow-up is fairly short for this disease process. The magnitude of benefit over a longer period is yet to be determined, but it is hoped that benefit will continue to increase.

In our clinic, we aggressively pursue treatment of HFpEF. With an EF greater than 40%, abnormal BNP and symptoms, we start Entresto and an SGLT 2 inhibitor. We routinely halve the loop diuretic frequency because of the observed potency in decongesting the patient with use of Entresto. We have found that, if we do not reduce diuretic frequency, patients have significant side effects from volume depletion with orthostasis or fatigue. The observed benefit in our clinic has been stark. It is certainly difficult to observe decrease in the endpoints of CV death and decreased heart failure hospitalization, but it has not been difficult to see the symptomatic improvement in patients. Prior to 2020, our approach was mainly to control blood pressure and volume status, and to add spironolactone. The effect was obvious in a small minority of patients, but most patients had little apparent benefit. It has been encouraging to see so many patients improving clinically since starting this more aggressive approach. We add spironolactone if vol-

ume remains a significant issue after the Entresto has been escalated to the highest dose, if the creatinine remains below 1.8 and if the potassium is less than around 4.5. We have seen in real time the development of successful treatment of a difficult-to-treat entity.

ACUTE DECOMPENSATED HEART FAILURE (ADHF)

In all the trials mentioned so far, heart failure has been a chronic problem. Patients included in the trials have had to be symptomatic, generally with class II-IV symptoms. These trials have not studied an entirely different phenotype of patients, those with acute decompensation or de novo heart failure. The outcomes of people with an acute decompensation are different than patients with chronic heart failure. Rehospitalization occurs in 25% of patients with ADHF in the first month and in 60% in the first 6 months following an acute decompensation. It is not a foregone conclusion that patients will respond to treatment for ADHF with reduced or preserved EF as they do for chronic HFrEF or HFpEF. This concept is an active area of research with several trials published in the past couple of years.

The EMPULSE trial studied the effect of empagliflozin on acute heart failure regardless of ejection fraction (8). In the EMPULSE trial, 566 patients were randomized to receive empagliflozin or placebo with median time to randomization 3 days from admission to the hospital. A win-ratio statistical analysis was used, so that fewer patients would be needed to show benefit. A full discussion of this new statistical strategy is beyond the scope of our topic but, briefly, the win-ratio analyzed 4 outcomes and gave preference in a hierarchical manner. Multiple outcomes can be tested at once to produce a general answer as to which therapy is most effective. In this trial, death from any cause, heart failure event frequency, time to heart failure event, or 5 point or greater difference in change of Kansas City Cardiomyopathy Questionnaire Total Symptom Score (KCCQ-TSS) were the outcomes tested. Each patient receiving empagliflozin was tested for each outcome in the mentioned order against a patient receiving placebo and, if the outcome was present, a winner was determined. If there was no winner then it was considered a tie. Ultimately, the win ratio of clinical benefit was 53.9% in the empagliflozin group versus 39.7% in the placebo group, indicating a 36% benefit for the empagliflozin group. The power of this win-ratio study was apparent in seeing a benefit in so few patients at only a 90 day follow-up period. While the results are not intuitive, it is provocative that benefit can be observed so quickly in so few patients.

Entresto was tested in patients with 40% or less ejection fraction who were hospitalized for acute heart failure in the PIONEER trial (9). Enalapril was given to the comparator group. This trial had a serologic endpoint. The primary endpoint was the time-averaged proportion change in the NT-proBNP concentration from baseline through 4 to 8 weeks. In our clinic we routinely use serologic changes to define risk for rehospitalization and to guide follow-up for these difficult-to-treat patients. There was a greater reduction in the NT-proBNP in the group receiving Entresto vs the group receiving enalapril.

There are few completed trials showing how to treat patients with acute heart failure with reduced or preserved ejection fraction. Presently, there are numerous medicines being tested in acute heart failure. Many of these will have significant overlap with our strategy in treatment of chronic heart failure. We eagerly await these results, as currently we must make assumptions that quad therapy for HFrEF or triple therapy for HFpEF is beneficial. Indeed, in our clinic, we apply significant resources to the institution of these therapies with clinical follow-up and escalation of the medicines to recommended doses.

OUR APPROACH

We have a program in our clinic that provides escalation of therapy, clinical follow-up, and screening for side effects or failure of therapy. In our pursuit to meet the needs of this ill population, it has become clear that the single most important feature to the care of these patients is the frequency of the follow-up. This has provided a strain on our system but by, using a multi-disciplinary team approach, we are better able to safely meet the patients' needs.

We have a team for our heart failure care. Two nurses work in the hospital to assist with care of all of the hospitalized patients. They are knowledgeable regarding the importance of identifying ADHF patients, so that appropriate medical therapy can be initiated and appropriate follow-up arranged. A case manager, specifically trained in heart failure, ensures a seamless transition from the hospital to the home setting. This includes a detailed medicine reconciliation and an appointment within 7 days of discharge, preferably 2 or 3 days. A physician or non-physician provider (NPP) such as a Physician Assistant or Nurse Practitioner sees the patient to discuss the adequacy of treatment after discharge. Most commonly, patients will be seen weekly for a month and then bi-weekly or monthly, depending on the success of management and the patient-specific understanding of the heart failure treatment. We have tried using heart failure-trained nurse phone calls as suggested in some studies, but this has been inadequate to convey the seriousness and high likelihood of decompensation to this patient population. For patients unable to keep this rigorous follow-up schedule, we offer telehealth visits to serve the process of escalating therapy and to convey the important information patients need to have clinical success. We provide a binder that explains the disease process, the signs of decompensation, the purpose of each medicine and allows them to track their weight, blood pressure, and medicine changes over time.

In general, patients remain on an intensive track for 3 months--sometimes more based on the support and educational needs of the patient. As the patients reach maximal medical benefit, we hope that they are at the highest tolerated recommended doses of each therapy: Entresto 97/103 twice daily, carvedilol

| *continued on page 14*

HAPPY.

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25mg twice daily, Empagliflozin 10mg once daily, and spironolactone 25mg once daily. In order to achieve these doses, the team practices protocol-driven care, so the nurses can titrate safely. Our protocol is less aggressive than suggested in the STRONG HF trial, but we believe our strategy to be safe and well tolerated. We start SGLT 2 inhibitors in the hospital as they have significant benefit and have minimal impact on blood pressure, kidney function, and serum potassium. We try to start Entresto at 24/26 twice daily in conjunction with carvedilol 6.25mg twice daily while the patient is still in the hospital. This strategy works well so long as the systolic blood pressure is greater than 100 mmHg. Frequently, aggressive diuresis impedes this approach as blood pressure and kidney function can be affected. We routinely decrease the diuretic frequency by half with the institution of this therapy. It is our habit to double either Entresto or carvedilol on a weekly basis so long as the patient is not having orthostasis, regardless of blood pressure. If the patient is having significant orthostasis or fatigue thought to be due to low blood pressure or volume depletion, we assess whether it would be appropriate to further decrease diuretic frequency, wait another week before doubling a medicine, or both. With this strategy, patients virtually always achieve maximal doses within 2 months. As stated, this is slower than the scheme shown to be effective in STRONG HF trial. Laboratory exam with chemistry and BNP are performed on the day of change of a medicine and a week later.

This is an intensive process that we have developed over the past 10 years. We have found that patients are often unable to report decompensation regardless of understanding the disease process or sophistication. Furthermore, patients struggle to be motivated to take higher doses of therapy. Achieving these therapeutic goals requires a large team of dedicated professionals and protocol-driven care. We realize that this is a work in progress, and all members of the team are encouraged to give input to improve efficiency, safety, or communication.

Our team had made significant progress to achieve our goals to maximize therapy. We had a belief that maximal doses of Entresto and carvedilol were important based on the clinical trials. Further, we believed that adding classes of therapy until quad therapy was achieved was important as long as patients remained symptomatic with careful history-taking. We were gratified to see the work by Tromp et al published in 2022 in JACC Heart Failure (10). They produced a meta-analysis compiling HFrEF trials since the 1980s. With this newer technique, magnitudes of benefit can be observed and compared between combinations and additions of therapies. We laminated the accompanying table and posted it throughout the clinic so that everyone who participates in the care of this needy population can have a visual reminder of why we spend so much of out resources in escalating care.

A brief look at how to interpret this forest plot is warranted (see Table 1). This particular graph refers to mortality in patients with HFrEF. If a patient receives the standard care of 2000 (ACEi and beta blocker), the hazard ratio is 0.69, a 31% reduction. If standard care for 2005 is adopted by adding spironolactone the hazard ratio is 0.52, a 48% reduction. If standard care in 2014, substituting Entresto for ACEi and keeping spironolactone and beta blocker, then the hazard ratio is 0.44, a 56% reduction. Lastly, if 2021 care is achieved by adding an SGLT 2 inhibitor to Entresto, beta blocker, and spironolactone, then the hazard ratio is 0.39, a 61% reduction. This stepwise look at escalation of care over time validates our approach to pursue full care as quad therapy.

There is no doubt that heart failure care is evolving. Differentiating the disease entities into HFrEF, HFpEF, and ADHF helps to better define treatment strategy and follow-up care. Additional therapies provide exciting but challenging opportunities to decrease the burden of heart failure on the patient, the hospital, and the financial system and to improve the length of life meaningfully. We have adopted a team approach that incorporates nurses, medical assistants, NPPs, and physicians. It is our hope to include pharmacists, nutritionists, and rehab specialists in the future.

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Hole in the Heart – Can I run with my Peers?

by Sangeeta Dhammu MD, PGY2 & Srilatha Alapati MD, FACC, FAAP

Congenital heart defects (CHD) are one of the most common congenital defects we encounter, occurring in eight per 1000 live births. We are seeing striking improvements in survival with palliative or corrective heart surgery, and we now have more adult patients than pediatric patients alive with congenital heart defects. While this is amazing news, it is the job of caregivers and providers to make their lives as close to normal as possible.

There is enough research to state that "exercise is for everyone", and that includes most children with congenital heart defects. Cardiologists agree that routine regular exercise is beneficial for all children including ones with complex congenital lesions. "You need to get out more, work out, play outside." This means something completely different when you have a child with CHD. While some caregivers are overly cautious and restrict all activity, others might not have enough information and allow activities that are unsafe and result in the feared complications associated with these conditions. Still, there is an overwhelming amount of information about benefits from exercise including reduced risk of heart attack, stroke, high blood pressure, and diabetes, to name a few.

Guidelines exist that help pediatricians guide caregivers and patients about participating in sports. It is crucial that pediatricians provide anticipatory guidance, discuss complications, and document the understanding of the patient and caregiver. Caregivers must be informed about these guidelines. Every caregiver should be aware of their child's specific lesion or defect, and what symptoms to watch for to judge their child's functional status.

Congenital heart defects can range from simple shunting lesions, where a structural defect in chambers of the heart leads to abnormal blood flow, to complex single ventricle physiology, where there is a single lower chamber in the heart. The structure, location, and extent of the defect or lesion affects how well or poorly the blood is oxygenated and pumped throughout the body.

When caregivers understand what they are dealing with at the anatomic or structural level, they can better understand the implications on the child and need for activity restrictions. The level of sports participation is dependent mainly on the training and the competitive aspects of the activity, and must be individualized to the child, meanwhile considering the child's functional status and surgical correction. Clinicians should encourage their patients to engage in healthy physical activities, keeping the specific features in mind like residual obstruction, pulmonary vascular disease, low systemic ventricular function, preexisting arrhythmias, and the presence of implanted cardiac rhythm devices such as pacemakers and implantable cardioverter-defibrillators.

Depending on clinical severity of the congenital heart defect, it is a shared decision-making process with patient and parents. The type of sports and the classification provided in Table 1 can help guide that decision. It can be safely said that CHD patients without clinical symptoms, and with good blood oxygenation, can participate in most low intensity sports.

Below are the simplified guidelines from the American Heart Association for sports participation for children with certain congenital heart defects:

SIMPLE SHUNTING LESIONS: ATRIAL SEPTAL DEFECT (ASD), VENTRICULAR SEPTAL DEFECT (VSD), PATENT DUCTUS ARTERIOSUS (PDA).

Patients with hemodynamically insignificant lesions such as VSD, ASD, and

Table 1. Classification of Sports

	A. Low Dynamic	B. Moderate Dynamic	C. High Dynamic
I. Low static	Bowling, Cricket, Curling, Golf, Riflery, Yoga	Baseball, Softball, Fencing, Table tennis, Volleyball	Badminton, Cross country skiing, Field hockey
II. Moderate static	Archery, Auto racing, Diving, Equestrian, Motorcycling	American football, Field events, Figure skating, Rodeoing, Rugby, Running, Surfing, Synchronized swimming, Ultra racing	Basketball, Ice hockey, Cross- country skiing, Lacrosse, Running, Swimming, Team handball, Tennis
III. High static	Bobsledding/Luge, Field events, Gymnastics, Martial arts, Rock Climbing, Sailing, Water skiing, Weightlifting, Windsurfing	Bodybuilding, Downhill skiing, Skateboarding, Snowboarding, Wrestling	Boxing, Canoeing, Kayaking, Cycling, Decathlon, Rowing, Speedskating, Triathlon

PDA who remain asymptomatic can participate in all sports without restrictions.

Children who have complications that are hemodynamically significant, like pulmonary hypertension, can develop acute symptoms, including reduced exercise capacity or, more importantly, arrhythmias, syncope, chest pain, or (the most feared of all) sudden cardiac death.

Further testing is recommended in symptomatic children and decisions must be made on an individual basis.

PULMONARY VALVE STENOSIS (PS):

Decisions for sports participation and activity restriction are based on estimated severity by detecting pressure gradients on each visit to the cardiologist. A peak instantaneous gradient <40 mm Hg indicates mild PS, 40 to 60 mm Hg indicates moderate PS, and >60 mm Hg indicates severe PS. Adequate relief after a valve procedure means resolution of symptoms or a reduction in gradient to <40 mm Hg.

Athletes with mild PS and normal right ventricle (RV) function can participate in all competitive sports. Annual reevaluation is recommended. Athletes treated by operation or balloon valvuloplasty who have achieved adequate relief of PS (gradient <40 mm Hg by Doppler) can participate in all competitive sports. Athletes with moderate or severe PS can consider participation only in low intensity sports (classes IA and IB).

Athletes with severe pulmonary insufficiency with marked RV enlargement can consider participation in low-intensity sports (classes IA and IB).

AORTIC STENOSIS (AS):

To grade these patients as mild, moderate and severe, we need to go by annual physical examination, electrocardiography (ECG) and Doppler echocardiography. In all cases, regardless of the degree of stenosis, patients with a history of fatigue, light-headedness, dizziness, syncope, chest pain, or pallor on exercise deserve a full evaluation. Children with mild AS can participate in all competitive sports without restriction. Athletes with moderate AS may be considered for participation in low static or low to moderate dynamic sports (classes IA, IB and IIA). Athletes with severe AS can participate only in low intensity sports (class IA) and are restricted from competitive sports.

COARCTATION OF THE AORTA:

The severity of the disease in this congenital heart defect is determined by a clinical examination that includes the arm/leg pressure gradient, exercise testing, echocardiographic studies, and magnetic resonance imaging. Athletes with coarctation and without significant ascending aortic dilation with a normal exercise test and a resting systolic blood pressure gradient <20 mm Hg between the upper and lower limbs and a peak systolic blood pressure less than 95th percentile of predicted blood pressure with exercise can participate in all competitive sports. Conversely athletes with a systolic blood pressure arm/leg gradient >20 mm Hg or exercise-induced hypertension or with significant ascending aortic dilation

may be considered for participation only in low-intensity sports.

PULMONARY HYPERTENSION ASSOCIATED WITH ANY CONGENITAL HEART DISEASE:

Patients with mean pulmonary artery pressure of <25 mm Hg can participate in all competitive sports. Patients with moderate or severe pulmonary hypertension, with a mean pulmonary artery pressure >25 mm Hg, should be restricted from all competitive sports, except for low-intensity sports. Complete evaluation and exercise prescription (physician guidance on exercise training) should be obtained before athletic participation.

VENTRICULAR DYSFUNCTION AFTER CHD SURGERY:

Ventricular dysfunction is a common complication after surgery for CHD, and this dysfunction understandably affects exercise performance. Before participation in competitive sports, all athletes with ventricular dysfunction after CHD surgery should undergo evaluation that includes clinical assessment, ECG, imaging assessment of ventricular function, and exercise testing.

Athletes with normal or near-normal systemic ventricular function (EF ≥ 50%) can participate in all sports. It is reasonable for athletes with mildly diminished ventricular function (EF 40%–50%) to participate in low- and medium-intensity static and dynamic sports. Athletes with moderately to severely diminished ventricular function (EF <40%) should be restricted from all competitive sports, except for low-intensity sports.

REPAIRED TETRALOGY OF FALLOT:

Before participation in competitive sports, it is recommended that all athletes with repaired Tetralogy of Fallot should undergo evaluation, including clinical assessment, ECG, imaging assessment of ventricular function, and exercise testing. Athletes without significant ventricular dysfunction (EF >50%), arrhythmias, or outflow tract obstruction may be considered for participation in moderate- to high-intensity sports. To meet these criteria, the athlete must be able to complete an exercise test without evidence of exercise-induced arrhythmias, hypotension, ischemia, or other concerning clinical symptoms. Athletes with severe ventricular dysfunction (EF<40%), severe outflow tract obstruction, or recurrent or uncontrolled atrial or ventricular arrhythmias should be restricted from all competitive sports, except for low-intensity sports.

TRANSPOSITION OF GREAT ARTERIES, AFTER ARTERIAL SWITCH REPAIR:

Before participation in competitive sports, athletes with transposition of great arteries after repair should undergo an evaluation that includes clinical assessment, ECG, imaging assessment of ventricular function, and exercise testing.

Participation in competitive sports in athletes with a history of clinically significant arrhythmias or severe ventricular dysfunction may be considered on an individual basis, based on clinical stability.

Athletes without clinically significant arrhythmias, ventricular dysfunction, exercise intolerance, or exercise-induced ischemia may be considered for participation in low- and moderate intensity competitive sports.

Asymptomatic athletes with TGA and without abnormalities on clinical evaluation may be considered for participation in moderate- to high-intensity competitive sports.

Athletes with severe clinical systemic RV dysfunction, severe RV outflow tract obstruction, or recurrent or uncontrolled atrial or ventricular arrhythmias should be restricted from all competitive sports, with the possible exception of low-intensity sports.

FONTAN PROCEDURE:

Palliative surgery performed in children born with only one functioning ventricle redirects the systemic venous blood to the pulmonary arteries directly. Cardiopulmonary exercise stress test should be done to evaluate for exercise intolerance along with routine ECG and cardiac imaging tests to evaluate for ventricular dysfunctions and arrhythmias. Evaluation and optimization of lung function is also very important in these patients. Patients who are on chronic anticoagulation should be restricted from high contact sports.

Athletes who have undergone the Fontan procedure and who have no symptomatic heart failure or significantly abnormal intravascular hemodynamics can participate only in low-intensity class IA sports.

In summary, most children with simple cardiac defects who have had successful surgical repair may safely participate in physical activities or sports at varying levels of classifications. Children with more complex conditions may need some restrictions in activities. For overall health and well-being, it is important and healthy to allow activity at a level safe for that child, and the cardiologist works with families to understand and decide what is safe and appropriate for an individual child.

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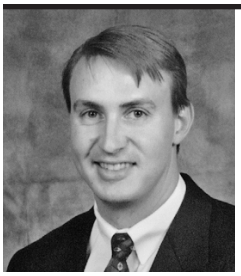

Peripheral Arterial Disease

by David Langley, MD

Consider this scenario. A husband and wife are walking to play a round of golf. The wife has just been recently diagnosed with breast cancer. The husband notes as he approaches the next hole that he has been getting an aching pain in his calf whenever he walks more than a block. The pain goes away when he stops, so he has not been particularly worried about it.

Out of these two, which one has the highest risk of dying in the next 5 years? According to the American Cancer Society, when all stages of newly diagnosed breast cancer are considered, the 5-year survival for breast cancer is 91% (1). On the other hand, one third of patients with peripheral vascular disease will die within 5 years. Moreover, 16% suffer from a myocardial infarction, TIA/stroke, angina, or cardiovascular death within one year after diagnosis, and almost one third within 3 years of diagnosis (2).

Peripheral arterial disease may manifest in the legs, but it is a marker of significant atherosclerosis throughout the body, providing an excellent opportunity to decrease morbidity and mortality in these patients. Interventions to decrease the risk of cardiovascular death, stroke, and limb loss in these patients are widely available and make a major impact on long-term outcomes.

RISK FACTORS

The priorities for treatment of peripheral artery disease are first, modification of the risk factors that lead to the disease and, second, treatment of the manifestations of the disease itself.

The major risk factors for peripheral artery disease are well-defined and include tobacco use, diabetes, hypertension, and hyperlipidemia. Cigarette smoking and diabetes are, by far, the most important of these. Continued tobacco use in patients with peripheral vascular disease contributes to an increase in disease progression and to a decrease in the longevity of interventions undertaken to treat the disease. In addition, for those who stop smoking completely, the risk of future limb loss drops significantly.

Diabetes causes multiple deleterious effects on limbs, including progression of atherosclerotic vascular disease, neuropathy with loss of sensation in the feet, and poor immune response related to hyperglycemia. While intensive control of hyperglycemia in Type 2 diabetics has been shown to decrease their cardiovascular risk, unfortunately the risk of peripheral vascular disease remains relatively unchanged (3).

Treatment of hypertension and hyperlipidemia reduce the risk of associated ischemic events in the coronary and cerebral circulation. The Scandinavian Simvastatin Survival study included patients with elevated cholesterol and a previous myocardial infarction. The benefits of statin therapy in this group included a 42% reduction in the risk of major coronary events as well as a 30% reduction in total mortality at an average follow up of 5.4 years (4). Control of hypertension has also been shown in large trials to decrease the risk of cardiovascular death and stroke.

TREATMENT

Once appropriate medical therapy is instituted, patients with peripheral vascular disease can be divided into 3 broad groups based on symptomatology--asymptomatic, intermittent claudication, and critical limb ischemia.

The asymptomatic patient requires nothing more than treatment of their risk factors and monitoring for progression of their peripheral vascular disease. Keep in mind that all interventions for treatment of vascular disease--whether the intervention is catheter-based treatment or open surgical treatment with bypass-- have a limited lifespan. Therefore, the notion of pre-emptively treating an area of stenosis to prevent future problems is almost never entertained. Once a procedure is undertaken, the clock starts on the development of recurrent stenosis in that area, and the resulting outcome may be worse than the patient's initial presentation, with more limited options for treatment.

Intermittent claudication indicates that patients have an arterial occlusion in the vascular tree of the legs significant enough that they are asymptomatic at rest but develop pain in the legs with walking. Claudication indicates that the perfusion to the limbs is adequate while resting but is unable to meet the increased needs of active muscles. This usually occurs predictably at about the same distance but may vary based on how fast the patient is walking or whether they are walking up an incline or stairs. The other hallmark of claudication is the reliable relief of the discomfort with rest. For patients with occlusive disease of the aorta or iliac vessels, their symptoms usually occur in the lower back, buttocks, or proximal thigh. Those with occlusions in the superficial femoral and popliteal arteries usually experience symptoms in their calves.

The presence of intermittent claudication, while an excellent indicator of the presence of systemic atherosclerotic disease, fortunately does not indicate a high risk of progression to amputation, with a projected risk of limb loss of 10% or less over 10 years. For patients diagnosed

with peripheral vascular disease who quit smoking, their risk of limb loss drops to essentially 0%.

Because the risk of amputation is low, the aggressiveness of treatment of the symptoms of claudication should be guided by their impact on the person's quality of life. The symptoms of claudication depend on the degree of arterial obstruction as well as the patient's activity level. Younger patients with lesser degrees of obstruction who are more active may experience greater hindrances in their daily activities of living than a sedentary elderly person with much more severe occlusive disease. Because of this, my approach has been to let the patient determine how much their symptoms affect their daily life and how aggressive they would want to be with treatment.

In many cases, the first line of therapy is a simple exercise regimen. The normal response of patients who experience discomfort in their legs with walking is to simply decrease their activity levels. This results in further weakening of their legs with worsening of their ability to ambulate. This can be reversed with a walking program. Patients are instructed to pick a length of time to start. During this time, they should walk until they experience discomfort and then rest until it is relieved. This cycle is repeated until the time they have selected is complete. For those who are committed to this, they should add a few minutes each week or every other week to their time. Disciplined patients in a walking program generally double or triple their walking distance prior to onset of discomfort. The addition of cilostazol may further enhance this effect (5).

Classically, there are 2 indications for more aggressive intervention in peripheral vascular disease--lifestyle limiting claudication symptoms and critical limb ischemia with either rest pain or ulceration. For patients with claudication, a full discussion of the options for treatment with either catheter intervention or open surgery allows them to make an informed decision of whether their symptoms are significant enough to warrant more aggressive treatment.

Critical limb ischemia is defined by the presence of either rest pain or ulceration of the leg. Rest pain occurs when the perfusion to a limb is poor enough that patients experience discomfort in the legs and feet without activity. Many times, this will be noted at night when lying supine and is relieved by hanging the foot dependent to allow gravity to augment blood flow. This may also be accompanied by neurologic compromise of the foot with development of numbness.

Not all cases of tissue loss in the leg and foot are related to vascular disease. Ulceration of the foot or leg can be due to many etiologies. In some cases, ulceration may occur due to poor perfusion of the foot. In others, lack of blood flow may contribute to an inability to heal an area of minor trauma to the foot.

For cases of critical limb ischemia, expeditious treatment of associated vascular disease is critical, as the risk of progression to amputation is high. Multiple modalities of treatment are possible, with the best option for each patient determined individually based on the pattern of their disease as well as their age and associated comorbidities.

CONCLUSION

A diagnosis of peripheral vascular disease provides an indication of the presence of systemic vascular disease. Risk factor management can decrease the risk for future cardiovascular events and stroke, as well as the risk of limb loss. Intervention for symptomatic patients—either with a dedicated walking regimen or with intervention (either catheter-based or with open surgery)—can usually improve symptoms and relieve limb-threatening ischemia.

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Rheumatic Heart Disease

by Scott Milton, MD

As the subject "Infectious Disease and the heart" is quite broad, I thought I would dedicate my article to rheumatic heart disease, as this condition, although uncommon in the US, has an interesting history and the preventive strategies of rheumatic fever can be confusing. Rheumatic fever still remains common in other parts of the world, and the general practitioner in the Texas Panhandle may be confronted with management issues from time to time. As rheumatic heart disease is a consequence of acute rheumatic fever, I'll need to discuss rheumatic fever in significant detail. Cardiac involvement will be discussed, and then management issues of individuals with rheumatic fever and preventive strategies will be reviewed.

The various clinical manifestations of rheumatic fever such as arthritis, carditis and chorea have been described for centuries, but it wasn't until the late 19th and early 20th century that the association between sore throat, fever, rash and acute arthritis (as well as the other symptoms associated with rheumatic fever) was found to be caused by a recent infection. During this time, bacteria were discovered as causative agents of disease and streptococcal bacteria, in particular, were identified and grouped based on their laboratory characteristics. Rheumatic fever was then clearly identified as an immune-mediated illness following certain group A strep infections. After World War II, antibiotic use became widespread and, with this, the development of strategies to prevent the disease.

ACUTE RHEUMATIC FEVER: PATHOGENESIS AND INCIDENCE

Acute Rheumatic Fever (ARF) occurs within several weeks of an upper respiratory infection caused by Group A streptococci. Curiously, cutaneous streptococcal infections do not induce ARF. Whether this is because of the rich pharyngeal lymphatics or an inability of cutaneous strain to induce a "rheumatic" immune response is not clear even today. Group A streptococci vary in their ability to induce a "rheumatogenic" response in that certain M serotypes are strongly associated with ARF. However, it is likely that these ARF-inducing serotypes display different degrees of virulence. Factors that favor person-to-person transmission obviously enhance virulence. The extensive research that addresses the various pathways of the immune response which eventually lead to end-organ damage is beyond the scope of this overview. It is clear, however, that both humoral and cell-mediated responses are involved. Pathologic findings show the presence of inflammatory lesions of connective tissue. In the heart, findings of pericarditis, myocarditis or endocarditis may be found. Endocarditis is almost exclusively left-sided. A "MacCallum's patch", a thickened, roughened area above the posterior leaflet of the mitral value in the left atrium may be seen. Inflammatory infiltration of the leaflets and chordae of the heart valves occurs, leading to edema and cellular infiltration. As inflammation abates and is replaced with healing. distortion of the valves and chordae occurs, leading to thickening of the valves and eventually to insufficiency and stenosis years later.

The incidence of ARF in the United States and Western Europe has drastically declined over the last several decades. The rate of decline was most dramatic during the 60's and 70's. Surveys conducted in the late 70's and early 80's indicated an incidence of 0.5 per 100,000 annually. ARF is more common in lower socioeconomic groups, with the incidence much higher in Blacks. Household crowding is felt to be more important than a genetic predisposition. The risk of recurrence is markedly higher in individuals after the initial bout of ARF. Further, this increased risk of recurrence is most pronounced within the first few years following the initial infection. The risk declines thereafter for unclear reasons. Preexisting heart disease and the magnitude of the antistreptolysin O (ASO) response are associated with recurrence.

The global incidence of ARF is still high, with endemic areas including the Middle East, the Indian subcontinent, and areas of Africa and South America. The World Health Organization estimates that 500.000 cases of ARF occur annually; of these, 300,000 develop rheumatic heart disease (RHD). More than 15 million people live with RHD, and more than 200,000 die annually from RHD. Extraordinarily high rates of ARF and RHD are seen in Aboriginal populations of New Zealand and Australia. The annual incidence of ARF in Aboriginal children in some parts of Australia is 150 to 380 per 100,000, with a point prevalence of RHD approaching 2%.

ACUTE RHEUMATIC FEVER: DIAGNOSIS

The clinical manifestations of rheumatic heart disease are the result of the chronic inflammatory changes of the heart tissues. Endocarditis involves the mitral valve more commonly than the aortic valve. Acute rheumatic carditis is associated with three characteristic murmurs: a mitral regurgitation murmur (high-pitched, holosystolic, apical and blowing), the Carey Coombs murmur (a low-pitched, apical, mid-diastolic mitral flow murmur), and, finally, a high pitched decrescendo diastolic murmur of aortic

regurgitation (best heard at the primary and secondary aortic areas). The tricuspid valve is rarely involved and the pulmonic valve almost never. Conduction disturbance (first degree or greater heart block) is associated with ARF but is not in itself diagnostic of rheumatic carditis. Subcutaneous nodules are rare and associated with severe carditis. They are most commonly found over extensor surfaces and tendons. Rheumatic nodules are painless, firm and vary in size from a few millimeters to 2 centimeters; their numbers vary from one to a few dozen, and they persist for several weeks. Rheumatoid arthritis can cause similar lesions, although nodules with this disease are more persistent.

The Jones criteria are utilized to diagnose ARF. They include five major criteria, four minor criteria and serological evidence of an antecedent group A streptococcal infection. The major Jones criteria include carditis, arthritis, chorea, erythema marginatum and subcutaneous nodules. The four minor criteria include fever, arthralgias, elevated acute phase reactants (ESR and CRP) and prolonged P-R interval. Diagnosis requires one major and at least two minor criteria or two major criteria and evidence of antecedent group A streptococcal infection.

The echocardiogram has been utilized in diagnosing "subclinical carditis" in individuals with ARF and no murmur on physical exam but who are found to have valvular regurgitation on 2D doppler and color flow Doppler. At present, this "echocarditis" is not considered diagnostic of carditis. However, in areas of the world where ARF is much more common, it is used to satisfy one of the major Jones criteria (i.e., carditis).

The antistreptolysin test (ASO) should be positive at least 80% of the time if drawn within two months of the onset of ARF. Therefore, a negative ASO test drawn during this time frame makes the diagnosis of ARF doubtful. However, if the diagnosis of carditis was delayed or went unrecognized, then the ASO test may be negative, as this failure to suspect ARF may delay ordering the test.

ACUTE RHEUMATIC FEVER: PREVENTION

As RHD is the only long-term sequela of ARF, therapy to prevent recurrent attacks is extremely important. Acute symptoms are treated with salicylates and corticosteroids. Neither of these agents prevents or modifies the development of RHD. The prognosis of an individual attack correlates to the severity of carditis during the acute phase. The echocardiogram is much better at recognizing RHD than conventional screening in children in developing countries, and, in areas with high prevalence, should likely be utilized if possible.

Prevention of recurrent attacks of ARF in individuals with previous rheumatic fever is critical to minimize recurrent carditis and long-term damage to the heart tissues. Prophylaxis in areas of low incidence of ARF is intramuscular benzathine penicillin G every 4 weeks. Oral sulfadiazine or penicillin V are also acceptable, although less effective. A macrolide may be substituted in those allergic to penicillin or sulfadiazine. Duration of therapy is controversial. The risk of recurrent ARF declines with age and the number of years since the most recent attack. The presence and severity of RHD and the number of recurrent attacks increases the likelihood of ARF. The decision to discontinue prophylaxis by the clinician is thus complex, and includes determining the risk an individual may have in acquiring a new streptococcal infection as well as the consequences if this were to occur. Generally speaking, prophylaxis should be continued until at least 5 years have elapsed since the most recent rheumatic attack and the individual is in their 20's. Some patients may require longer duration, possibly even life-long. Please see the recommendations of the American Heart Association for further details.

Rheumatic heart disease is much less common in North America than other parts of the world. However, with global travel and immigration, the clinician in the Texas Panhandle should be aware and look for this disease in foreign-born individuals who may have relocated to this area. Prophylactic treatment can be for long periods of time, and the decision to stop prophylactic therapy is complex. Again, guidelines are available for reference through the American Heart Association.

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A PCP Looks at PCP (Primary Cardiac Prevention)

by Alan Keister, MD, FACP

Mr. H arrives at the ER with chest pain and signs of an acute heart attack. As I look over the chart, I see the subtle things: the slightly elevated blood pressure, the borderline blood sugar, the high cholesterol that was not treated because of his fear of statin drugs. Although he is a successful businessman, he does not exercise and is known to enjoy cigars and indulge in a traditional American diet. So many opportunities to intervene and prevent this outcome, but the story is all too familiar.

As a primary care doctor, I am challenged to be proactive in the lives of my patients. Primary prevention of cardiovascular disease is the art of decreasing suffering for patients by intervening to avoid heart disease. I hope this article will help patients have a better understanding of the role of the primary care doctor in prevention by discussing the tools we can use in shared decision-making.

RISK FACTORS

The best way to decide how aggressive to be in primary prevention is to do an assessment of risk factors.

Family History. One of the most important things a patient can do to help their doctor is to provide a good family history. Knowing if their parents had heart disease and at what age it became evident are significant data points in a cardiovascular history.

Tobacco Use. Checking the status of tobacco use is a routine part of questioning each year. Tobacco use means everything from cigarettes and cigars to dipping snuff; they all increase the risk of a future cardiac event. Avoiding tobacco is a simple intervention that can decrease heart disease risk exponentially.

Physical Activity. Another important factor is determining how active the patient is and whether they are engaged in regular exercise- The American Heart Association Guidelines recommend 150 minutes of moderate exercise or 75 minutes of vigorous exercise a week.

Diet. What kind of diet is a patient eating? From keto to paleo to plant based, patients are often confused about which diet is the best. Many of these diets are more focused on weight loss than prevention of heart disease. The American Heart Association (AHA) recommends a Mediterranean diet focused more on vegetables, fruits, nuts, whole grains, lean animal protein, fish, and olive oil. The dietary recommendations also suggest limiting refined carbohydrates, meats, and processed foods. In general, a simple recommendation is to shop around the outside of the grocery store and avoid the more packaged processed foods on the aisles.

Diabetes. Most people do not recognize how significant a risk factor diabetes is in cardiovascular disease. A person who has diabetes has the same risk of having a heart attack as someone who has already had a heart attack. While metformin remains first line therapy, primary care doctors have new options for drug therapy with GLP-1 receptor agonists and sodium glucose co-transporter 2 inhibitors to treat diabetes. Thes newer agents have the bonus effects of lowering the HbA1C and protecting the heart as well.

High Blood Pressure. The latest recommendations target the blood pressure at < 130/80. Lifestyle changes including diet, exercise and weight loss remain first line therapy. Medications should be added when blood pressure is persistently elevated to decrease progression to heart disease.

High Cholesterol. Elevated cholesterol increases the risk of heart disease by producing plaques in the arteries of the heart that can rupture and cause an inflammatory cascade that results in a heart attack. By lowering cholesterol and decreasing inflammation, cardiovascular risk can be lowered. Drug therapy is indicated in all patients with very high LDL cholesterol levels (> 190 mg/dL) and in many patients with LDLs between 100 and 160 if they have with other risk factors. (Patients with known coronary artery disease may be treated even with lower levels of LDL cholesterol.) Determining who should be treated and how aggressively to treat are common issues that arise in primary care office visits.

Some lesser-known risk factors include chronic kidney disease, inflammatory rheumatologic diseases like lupus and rheumatoid arthritis, and history of premature menopause (<age 40).

KNOW YOUR NUMBERS

A recent campaign by the American Heart Association to raise awareness of risk factors is the Know Your Numbers program. Patients are challenged to know their blood pressure, their lipids, their HbA1C, and their body mass index. As a patient, you can be your own advocate in knowing these numbers and asking your physician how to improve them.

After the risk factors are assessed, the process of developing an optimal prevention plan requires shared decision making and often a collaborative team approach.

ASSESSING RISK IN THE INDIVIDUAL PATIENT

One of the most important tools is doing a **10-year cardiovascular risk assessment** with a Pooled Cohort Equation. While no calculator is perfect, a race and sex specific risk estimator helps stratify which patient needs more aggressive risk factor modification. The American College of Cardiology website uses an ASCVD calculator that includes age, sex, race, blood pressure, cholesterol levels, diabetic status, smoking status, and BP treatment status to determine risk. For asymptomatic patients aged 40-75, risk can be calculated as low (< 5%), borderline (5%- <7.5%), intermediate (7.5%-20%), or high (>20 %).

Initiating lipid lowering therapy with a statin drug is considered standard of care in the high-risk groups, but what about the intermediate group? Current studies suggest performing a **cardiac calcium score** to further stratify risk. The cardiac calcium score is a specialized CT scan that looks at the amount of calcium in the walls of the blood vessels in the heart. Again, results are reported as very low risk (score 0), low risk (1-10), intermediate risk (11-100), and high risk (> 100). Those with a cardiac calcium score of 0 are very low risk of a cardiac event and are unlikely to benefit from a statin. Patients with an intermediate ASCVD score but a high-risk cardiac calcium score will often benefit from statin therapy. Cardiac calcium scores are relatively inexpensive at $100, but they often give supporting data to clarify which patients will benefit from starting lipid-lowering therapy. I have used this tool with my patients to help make the best collaborative treatment plan.

What about **aspirin**? Interestingly, aspirin is not routinely recommended in the latest guidelines. Most recent studies do not show significant benefits of aspirin in primary prevention of strokes and heart attacks. In patients > 70, prophylactic aspirin may be harmful.

The newest considerations in risk factor modification involve looking at the **social determinants of health** and how they play a role. Understanding a person's life circumstances—income level, access to healthy foods, race and ethnicity, lifestyle practices—can be very important in deciding how best to deliver health care. Education and health literacy can also provide a challenge to primary prevention. Social support for tobacco cessation and exercise are often missing in less resourced communities.

So, what do I tell my patients? How can I help them avoid having a heart attack?

Exercise almost every day!
(Things in motion stay in motion.)

Eat Right
(Mediterranean diet, every day.)

Quit all tobacco products
(yesterday!)

Diabetes (treat aggressively.)

Know your numbers
(BP, HbA1C, lipids and weight/BMI- treat to optimize- lifestyle changes first!)

Cardiovascular health is a team sport! Let's work together to keep your heart strong.

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Prescription Nutrition:
Diet and Heart Health

by Rachel Hutto, MS, RD, LC, CNSC

One in four. Twenty-five percent. The odds of dying from heart disease.

Physical inactivity, tobacco use, stress, infection, pollution, nutritional intake: what do these things have in common? They are all modifiable risk factors that can affect one's likelihood of being part of the twenty-five percent that will die from heart disease tomorrow, next week or next year. Choosing a healthy lifestyle pattern, including a healthy diet, can decrease those risks by up to 80 percent.

"Healthy", however, remains a vague word and is especially ambiguous in today's diet and nutrition industry culture. The American Heart Association (AHA) released its "2021 Dietary Guidance to Improve Cardiovascular Health: A Scientific Statement from the American Heart Association", outlining a heart healthy diet. The AHA recommends the following dietary guidelines (1):

1). Adjust energy intake and expen -diture to achieve and maintain a healthy body weight.

2). Eat plenty of fruits and vegetables, choose a wide variety.

3). Choose foods made mostly with whole grains rather than refined grains.

4). Choose healthy sources of protein: mostly plants, fish and sea food, low fat/fat-free dairy. If meat or poultry is desired, choose lean cuts and avoid processed meats.

5). Use liquid plant oils rather than tropical oils, animal fats, or partially hydrogenated oils.

6). Choose minimally processed foods instead of ultra-processed foods.

7). Minimize intake of beverages and foods with added sugars.

8). Choose to prepare foods with little or no salt.

9). If you do not drink alcohol, do not start; if you choose to drink alcohol, limit intake.

10). Adhere to above guidance regardless of where food is prepared or consumed.

To the trained eye, these recommendations appear standard, but to the general public these recommendations can be overwhelming and difficult to follow—especially in today's "right here, right now" world flooded with calorically dense, sugar-and fat-filled foods with animal meat being the main protein source. Even more confusing than the "right here, right now" world that we live in is the desire to fulfill the AHA's number one recommendation of maintaining a 'healthy body weight". For an adult who is overweight or obese, obtaining a healthy body weight not only takes time, but it takes sacrifice and discipline as well. Enter the diet and fitness industry with their slew of fad diets, ultra-processed protein shakes, and low-calorie food products.

While plant-based proteins such as pulses (beans, lentils, peas), ancient grains (quinoa, farro, chia, etc.), nutritional yeast, tofu and tempeh are heart healthy and recommended as the major source of protein intake by the American Heart Association, many mainstream vegetarian and vegan diet trends are not always concentrated around these whole food sources. With plant-based diets now trending, access to meat substitutes is at an all-time high. Unfortunately, a large majority of plant-based meat alternatives are high in fat, saturated fats, and sodium, are low in fiber, and are considered ultra-processed (4). Research shows that plant-based diets are beneficial for heart health, resulting in an overall mortality reduction and positive changes in lipid profiles; however, these benefits are negated by processed foods in diets that do not focus on whole food plant-based sources (3).

VERY LOW CARBOHYDRATE DIETS

Carbohydrate restricted diets such as the Atkins diet and Keto diet have increased in popularity, specifically for weight loss. By restricting carbohydrate intake, the body decreases insulin production and therefore increases lipid catabolism (use of fat for energy) resulting in weight loss. In the short term, such diets have shown promise in weight management. Low carbohydrate diets can decrease blood pressure readings, decrease blood glucose and HbA1C levels, and may have some cardioprotective effects (rat studies have shown that ketones reduce fatty acid production and oxygen consumption and increase cardiac contractility). While these outcomes may seem promising, other studies have shown an increased risk of atrial fibrillation, QT prolongation, cardiac chamber enlargement, and inflammatory markers (4). The positive effects on HbA1C levels are minimized if the patient goes off the ketogenic diet.

Problems surrounding low carbohydrate diets include excessive fat (especially saturated fat) intake, as well as reduced consumption of whole grains, which provide vital B vitamins and fiber intake. Low carbohydrate diets are also restrictive and hard for most people to manage long term, so that any positives

that may result from being on the diet are not sustained. Overall, long-term results show an increase in all-cause mortality, insulin resistance, cardiovascular disease risks, nutritional deficiencies, cancers of the colon, kidney disease and bone loss in patients on a ketogenic diet (3).

INTERMITTENT FASTING

Intermittent fasting is a diet focused on restricting the time frame of calorie consumption. Most intermittent fasting protocols focus on alternate-day fasting or various fasting intervals in a 24 hour period. This approach to nutritional intake has shown an increase in weight loss between study subjects when looking at equal caloric intake of one meal versus three meals, as well as positive effects on lipid profiles, blood pressure and blood glucose levels (although conclusions surrounding utilizing intermittent fasting in diabetes remains inconclusive) (4). Interestingly, rodent studies have shown enhanced weight loss in mice despite no caloric restriction. While an intermittent diet pattern may appear to have its benefits, the true possibilities lie in the sustainability of the diet and high-quality intake of foods whenever oral consumption occurs. Most human studies have focused on young to middle aged adults and should not be generalized to the older population (3).

WHAT DO THE EXPERTS RECOMMEND?

While popular diets promoted by industry may not be the answer to cardiovascular protection or treatment, there are several diets out there that are effective. The American Heart Association encourages these diet patterns:

- whole food plant-based diet
- the American Heart Association (AHA) diet
- the Mediterranean diet
- the Dietary Approaches to Stop Hypertension (DASH) diet.

Each of these diets is high in fruits and vegetables, with some encouraging up to five servings a day of fruits and five servings a day of vegetables. These diets also encourage a normal to liberalized intake of grains, focusing on whole grain consumption opposed to refined grains, while limiting dairy intake to low/non-fat dairy and limiting animal meat to low fat or minimal intake altogether. Fish intake is also a common recommendation in these heart-healthy diets, providing healthy fat intake and a lean protein source. Dietary patterns that focus on these foods give rise to better adherence and lead to lower sodium intake, limited saturated fat intake, increased intake of fiber, and adequate intake of micronutrients such as calcium, potassium, and magnesium (2).

Forbes Health recently surveyed board-certified physicians, registered dietitians, and certified food scientists on the "healthiest" diets for 2024. Parameters surrounding diet "health" included diet sustainability, effect on safe and healthy weight loss, promotion of heart health, promotion of general health for diabetics, and the diet's ability to be implemented by all socioeconomic classes. The top two diets in this survey of experts were the Mediterranean and DASH diets, showing these diets' application to the general public for overall health and prevention of chronic disease.

THE MEDITERRANEAN AND DASH DIETS

The Mediterranean diet specifically targets high intake of fruits, vegetables, whole grains, nuts, legumes, and olive oil, low to moderate intake of yogurt and cheese (daily), fish and poultry (a few times a week) and wine, with consumption of red meat and added sugars being limited to a few times a week. The DASH or "Dietary Approach to Stop Hypertension" diet specifically consists of daily consumption of 6-8 servings of grains, 6 oz. or less of meat, poultry, or fish, 4-5 servings of fruits and vegetables, 2-3 servings of low fat or fat-free dairy and fats or oils with 4-5 servings of nuts weekly and less than 5 servings of sweets weekly. Additionally, sodium intake should be less than 2300 mg daily with an ultimate goal of less than 1500mg daily for optimum blood pressure control. Both the Mediterranean and DASH diets recommend against consuming processed foods, and both encourage fresh whole foods as much as possible. Research shows the Mediterranean diet and DASH diet favorably affect lipid profiles, decrease insulin resistance, and increase overall cardiovascular health (2).

Changing eating habits takes time and effort. Making sustainable changes that provide nourishment and health in the long-term should be the ultimate goal, while also considering quality of life surrounding nutritional intake. Health professionals must help guide patients down the correct road, one that has minimal detours and is paved with evidence-based nutritional recommendations. Maybe this can move the mark and take that one in four to one in five. Maybe by encouraging our patients to make sustainable changes that are suitable for all will result in impact for entire families, and we can move the mark to one in six. It is time for the medical community to ditch the fad diet gimmicks and talk to our patients about strategic ways to procure, prepare and consume whole foods rich in nutrients, flavor and health.

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Endovascular Structural Heart Interventions: TAVR & Beyond

by Matt Parker, PT, MBA
Vice President Cardiovascular Services BSA Health System

Prior to 2002, surgical aortic valve replacement was the treatment of choice for patients who suffered from severe aortic stenosis. For those considered inoperable due to frailty and the inherent risk of the surgical procedure, options for treatment were limited. After pioneering techniques for balloon aortic valvuloplasty (BAV) in the mid-80's, the French physician Alain Cribier, MD, FACC, FESC, began to develop ideas for treatment options that would supersede the unsatisfyingly impermanent answer that BAV provided for inoperable severe AS patients (1). After considerable development through the 1990's and with invaluable research on transcatheter aortic valve replacement (TAVR) development by Dr. Henning Andersen (a fascinating history beyond the scope of this article), Dr. Cribier was the first cardiologist to percutaneously implant an aortic valve in an inoperable patient (2). On April 16, 2002, Dr. Cribier and his team performed the first-in-human TAVR in Rouen, France, and the future of treating AS was forever changed.

In March of 2005, the first TAVR was implanted in the United States (3). By 2007 the PARTNER (Placement of Aortic Transcatheter Valves) and CoreValve/ Evolut trials of balloon-expandable and self-expanding valves, respectively, were underway. With more than 9,600 patients in the studies, the results demonstrated superiority to medical treatment for AS in this patient cohort. The FDA recognized the efficacy of the treatment and greenlighted the device for use in inoperable patients in 2011, and CMS followed with coverage conditions in 2012 (2). The National Coverage Determination (NCD) from Medicare laid out specific indications for coverage. This was meaningful to providers and facilities because a significant portion of the candidates were expected to be Medicare beneficiaries.

SPECIFIC REQUIREMENTS INCLUDED:

- The patient must have an FDA-approved indication for the specific device,

- The patient must be under the pre- and post-operative care of a comprehensive heart team, to include a cardiac surgeon and an interventional cardiologist, as well as advanced patient practitioners, nurses, research personnel, administrators, and other professionals,

- The patient must be evaluated face-to-face independently by a cardiac surgeon and interventional cardiologist,

- The TAVR must be performed in a

hospital that provides on-site cardiac surgery and interventional cardiology, post-procedure intensive-care facilities with personnel skilled in open heart valve procedures, and appropriate surgical volume requirements, and

- The center must participate in an audited registry, such as the STS/ACC TVT Registry.

Perhaps the greatest benefit of the NCD was the widespread adoption of the "Heart Team" approach to patient care. Its not that this approach was necessarily novel, but the NCD did enforce the adoption of this approach across all facilities that joined the growing wave of active sites. At the national level, this led to cross-society cooperation (including the American College of Cardiology and the Society of Thoracic Surgeons) in the release of guidelines for TAVR in 2011. This led to the 2012 publication of the multi-society Expert Consensus Document on Transcatheter Aortic Valve Replacement that included contributions from the Society for Thoracic Surgery, the American College of Cardiology Foundation, the Society for Cardiovascular Angiography & Interventions, and the American Association for Thoracic Surgery (2).

After the CMS decision, high-risk surgical patients were approved by the FDA in 2012, and, four years later, intermediate-risk patients were added to the list. After the results of the PARTNER-3 trial were released, the FDA deemed low-risk surgical patients to be appropriate for the procedure (4). Locally, on November 11, 2019, a team comprised of Joaquin Martinez, MD, Kade Carthel, MD, and Don Robertson, MD, were joined by a talented multi-disciplinary team to implant the first TAVRs in Amarillo at BSA Hospital.

As the risk stratification expansion has increased the number of candidates for treatment, so have the number of facilities providing TAVR as an option. In 2013, only 252 TAVR programs were operating in the United States. By the second quarter of 2023, that number had ballooned to 831. In that same time, the per annum procedure count grew 11-fold, from 8,946 to 98,504 implants. Volume per site varies considerably, but 60% of TAVR facilities are doing between 50 and 200 cases annually, and just 27% are performing 50 or fewer. As the collective skillset developed and the patient risk stratification expanded to lower-risk patients, the percentage of cases utilizing the femoral approach rose to 96% in 2020. Prior to 2015 it had been 87%, with remaining patients receiving access from more direct vascular paths with less tortuosity and distance, such as transapical or transcarotid approaches. Importantly, as technical expertise has increased, the mortality data has steadily improved. In 2012, one year after the FDA approval for inoperable AS patients, the 1-year mortality rate was 26.4%. High-risk patients were added in 2012 and, by the time intermediate-risk patients were approved, the 1-year mortality rate had decreased to 15.3%. As of 2020 (the last year of 1-year mortality data from the STS/ACC TVT Registry Database), the rate had decreased to 11.2%. In-hospital mortality was 5.7% in 2012 but has held below 1.5% since 2018, with the 2022 rate being 1.2%. Given the calcific nature of this disease and the invasive penetration of large bore catheters and devices, embolic stroke is an inherent concern and risk. This metric has also demonstrated a steady decline over time, with in-hospital stroke rate dropping from 2.2% in 2012 to 1.4% in 2022 (5).

Per the Advisory Board, we can expect to see continued growth in the cardiovascular space over the next 10 years, both in outpatient and inpatient procedures, including TAVR. Despite headwinds like a dwindling workforce, unfavorable reimbursement, competing TAVR programs, and evolving site-of-care delivery models, the outlook for TAVR and other structural heart procedures is still strong. Endovascular valves are expected to increase 115% over the next 10 years, and left atrial appendage occlusion implants could go up as much as 65% in the same time span (6).

Structural heart procedure growth won't be driven by TAVR alone. Other endovascular structural heart techniques include edge-to-edge repair for mitral regurgitation, implants for left atrial appendage occlusion, and occlusion devices for patent ductus arteriosus, ventricular and atrial septal defects, and patent foramen ovale. Most recently, on February 1, 2024, the FDA approved the Edwards EVOQUE tricuspid valve for endovascular delivery. These minimally invasive treatments, and the others that are in various stages of development, will drive the growth of structural heart endovascular interventions and innovations over the next decade. Its an exciting time to be in the world of cardiology.

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Sudden Cardiac Death: A Review

by Jithin Kurian (MS4), Rajesh Nambiar, MD, MBA, FACC, FSCAI

"Analysis of death is not for the sake of becoming fearful but to appreciate this precious lifetime." - Dalai Lama.

Sudden Cardiac Death, or SCD, is the unexpected sudden death of a person due to a heart condition within one hour of the onset of symptoms. The rapid nature of SCD makes it one of the most intimidating causes of mortality in human pathology. Approximately half of all coronary heart disease-related deaths are due to SCD (1). This review article focuses on several research articles with the aim of providing education about screening, treatment, long-term management and prevention of SCD. With a better understanding of the pathogenesis and development of care, the incidence of SCD can be appropriately lowered.

BACKGROUND:

SCD is rapid death from a heart condition within one hour of symptom onset. It is most often due to congenital heart disease in the population between ages 0 to 13 and undetected coronary artery disease in the population over 35 years of age. Other less common causes include valvular heart diseases (especially aortic stenosis), cardiomyopathies, and genetic channelopathies. A common means of SCD is a ventricular arrhythmia (VA) such as ventricular tachycardia (VTach) and ventricular fibrillation (VFib). The risk of developing SCD peaks within the first month of a myocardial infarction (MI), but it is present in low-risk patients as well. Most SCDs are diagnosed by a post-mortem examination showing structural changes such as coronary artery disease (CAD), valvular aortic stenosis, or hypertrophic cardiomyopathy (HCM). The underlying heart disease can be either mechanical or arrhythmogenic in nature. Arrhythmogenic issues are non-structural by definition and include channelopathies such as long or short QT syndrome,

Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia (CPVT). An important diagnostic tool for patients with non-structural risk factors is genetic and family testing to prevent SCD.

EPIDEMIOLOGY:

Posthumous examination is unfortunately the most accurate way to diagnose SCD. In the United States, SCD is a leading cause of death and accounts for 15-20% of all mortalities (2). Every year, about 0.1% of the population in the United State and Europe will experience a cardiac arrest event that takes place outside of a hospital setting. The current survival rate for out-of-the-hospital cardiac arrest is 10% (2). An interesting fact about the epidemiology of SCD is that it shows a circadian pattern (with peaks in the early morning and late afternoon) and that Mondays are the days with the highest rate of mortality (3). Data from other parts of the world are limited but the incidence of coronary artery disease in the population correlates with the incidence of SCD. The classic risk factors for CAD [previous MI, diabetes, hypertension (HTN), smoking and body mass index (BMI)] also apply here. Some other predictors for the risk of SCD include low maximum oxygen uptake and ST segment depression or QRS prolongation during exercise.

CLINICAL FEATURES TO LOOK FOR:

It is important that patients at risk of SCD be evaluated preemptively, starting with history (including family history) and physical exam. Important clues on history includes prior history of exertional dyspnea, chest pain, hypertension and history of illicit drug use. Significant family medical history includes premature death before the age of 50, genetic cardiac diseases and familial history of epilepsy. On the physical exam, features to note include tachycardia, hypertension, cardiac murmurs and an elevated jugular venous pressure (JVP).

Current guidelines (1) state that a patient at high risk for SCD should undergo non-invasive diagnostic testing (ECG, stress test, and coronary artery calcium scoring). On ECG, common abnormalities to look for include microvolt T-wave alternans, QRS fragmentation, and prolonged QRS duration. Invasive exams such as coronary angiography and electrophysiology studies may be performed as indicated. Lastly, genetic testing has become an important tool to look for hypertrophic cardiomyopathy and channelopathies.

As mentioned earlier, a majority of cardiac arrests occur outside of the hospital or clinical setting. Typically, sudden cardiac arrest occurs without any preliminary symptoms or signs. The patient suddenly develops pulselessness, loss of consciousness due to reduced cerebral blood flow, and apnea. CPR should be initialed while a bystander calls 911. At this point, the next step before moving the patient to a hospital is to attach an

PANHANDLE HEALTH

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automated external defibrillator (AED) along with an EKG for monitoring purposes. Persons who work in populated areas should familiarize themselves with the operation and use of the AED ahead of time, before the crisis occurs. Using an on-site AED will save precious minutes before the ambulance and EMTs can arrive.

Time is of the essence in the resuscitation setting, since survival decreases from 25% to 0% within 10 minutes of an acute cardiac arrest without the institution of CPR (1). For the cases that take place outside of the hospital, CPR should be initiated immediately. Even in the cases of proper initiation, resuscitation efficiency decreases over time. In fact, the survival probability decreases by ten times after 20 minutes of CPR, in comparison to the survival probability prior to starting resuscitation (1). For cases within the hospital, defibrillation is usually the first-line treatment.

DIAGNOSIS AND ASSESSMENT:

If the patient survives resuscitation, either in the field or in the emergency department, a full cardiac workup can then be done at the hospital. This should include serial EKGs and cardiac biomarkers, as well as echocardiogram to look for signs of structural heart disease. If indicated, exercise stress testing, coronary angiography, and/or endomyocardial biopsy can be performed. Non-cardiac testing includes CBC, serum electrolytes, and urine toxicology screening.

If the patient does not survive resuscitation, an autopsy and post-mortem genetic testing is vital to differentiate between cardiovascular causes and non-cardiac causes of sudden death such as a subarachnoid hemorrhage or pulmonary embolism.

MANAGEMENT:

SCD is a precipitous process, so the main goal is to prevent it before it occurs. It is important to treat underlying cardiovascular conditions such as heart failure and hypertension.

The current mainstay treatment for SCD is the implantable cardioverter-defibrillator (ICD), which is important in both primary and secondary prevention of ventricular arrhythmias. Primary prevention is for high-risk patients, while secondary prevention is for patients who have history of SCD. ICDs are only recommended 40 days post-MI or 90 days after a revascularization procedure. Transvenous ICDs (T-ICDs) are the most commonly used form, followed by subcutaneous ICDs (S-ICDs). External ICDs are still being validated and are not commonly used for SCD yet.

In terms of medications for SCD, beta blockers and amiodarone have shown the greatest success in the prevention. Beta-blockers decrease mortality of patients with SCD and ventricular arrhythmias, while intravenous amiodarone has efficacy against ventricular arrhythmias alone. Catheter ablation can also be used to prevent VA from recurring. This procedure has higher efficacy in patients without structural heart disease as opposed to patient with structural issues.

FUTURE DIRECTIONS:

One of the main goals of out-of-hospital cardiac resuscitation is to regain spontaneous circulation. VA-ECMO, or venoarterial extracorporeal membrane oxygenation, has been used during SCA recovery. While it is not a direct treatment, it allows for a bridge until the return of heart function. According to a study by Napp et al (4), the overall 30-day survival after VA-ECMO was 12.5%, and 3 out of the 5 survivors had a promising neurological outcome. Another method being explored for care of SCA is percutaneous coronary intervention (PCI) performed for patients undergoing CPR. The CPR can be done mechanically or through ECMO. Combining PCI and CPR has shown to increase favorable outcomes as opposed to either one alone (4).

CONCLUSION:

SCD remains a major cause of mortality throughout the world. It is important to know the risk factors for a future SCA and to implement ways to prevent it. Diagnosis is most often done with genetic testing and imaging. In terms of treatment, the goal is to treat the underlying cause or to use methods such as T-ICDs, beta-blockers or VA-ECMO. While it is primarily a cardiac issue, it is an important problem to be recognized by all healthcare professionals.

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Complex Coronary Interventions: The Role of Circulatory Support Using the Impella® Device

by Paresh Rawal, MD

Ischemic heart disease has varied presentations, ranging from chest pain, shortness of breath, cardiac arrhythmias, congestive heart failure and, occasionally, no symptoms at all. Clinically, terms like new-onset angina, unstable angina, ischemic cardiomyopathy, severe multivessel or three-vessel coronary artery disease, left main disease, or "widow maker" LAD disease are used to convey similar findings.

Once the patient has been given the diagnosis of ischemic heart disease, often one of the 3 options are presented for treatment with the goal of getting the patient back to his/her pre-sickness functional status as soon as possible. Those 3 options are (1) medical therapy, (2) percutaneous coronary intervention (angioplasty, stent or atherectomy) and (3) coronary artery bypass surgery. If the patient also has valvular heart disease, then there are additional considerations to see if the patient needs valve repair or replacement together with coronary artery bypass surgery (generally quoted as "open heart surgery").

The option of angioplasty and stents often is very palatable because of the short (same-day or overnight) stay in the hospital and return to full activities within 2 to 3 days. Open heart surgery, on the other hand, entails 5 to 10 days in the hospital, followed by 6 to 12 weeks of cardiac rehabilitation before resumption of full activities. Despite these risks, studies have shown that patients with diabetes and multivessel coronary artery disease do better with coronary artery bypass surgery than with PCI. In addition, in spite of improved surgical experience, the complications of major sternal wound infection, renal failure, early graft closure, perioperative stroke, excessive bleeding, and mortality after surgery continue to be real. These complications, most of which stem from the need to use the external pump and membrane oxygenator, may be quite debilitating and potentially fatal.

Many advances in the last several years have made percutaneous coronary interventions more accessible for high-risk patient populations. Some of these advances include smaller size of the catheters or the sheath that is placed during the PCI, radial artery access techniques, precautionary measures to prevent trauma to the kidneys and judicious use of anticoagulant and antithrombotic medications. One such advance that has taken PCI technique to higher level is the use of an external percutaneous left ventricular assist device called Impella®. Special mention deserves to be made regarding a **"heart team approach"** where a comprehensive clinical team made up of cardiologist, cardiothoracic surgeon, general hospital physician, patient and family members get together to learn and discuss about the best options available for the patient.

CASE REPORT

Here I would like to give you a narration of a treatment that one of my patients successfully underwent at local hospital in Amarillo.

This 75-year-old gentleman presented with a history of hypertension, extensive smoking history, prior aortobifemoral bypass surgery 25 years ago, 2 vessel coronary artery bypass surgery 20 years ago, 3 subsequent coronary artery stent placements, stage IV chronic kidney disease, type 2 diabetes, and symptomatic right carotid stenosis. He presented at one of the local hospitals with debilitating chest pain (to the point that he could barely go for his daily activities like walking, going out to his car or putting out the garbage). Diagnostic cardiac catheterization showed left main disease extending to the origin of the left anterior descending and circumflex arteries (70 to 90% disease within a heavily calcific plaque) as well as tandem 80 to 90% lesions in a large dominant right coronary artery. His left ventricular systolic function was low normal level with ejection fraction of about 50%, and all of his previous bypass grafts were occluded. Patient was then referred to a tertiary care facility in the Dallas area, where he underwent evaluation for coronary artery bypass surgery.

In Dallas, his surgeons estimated that he had a 50% risk of requiring hemodialysis after the surgery and at least a 10% risk of death. Because of his previous coronary artery bypass surgery and aortobifemoral bypass, his vascular complication risk was felt to be extremely high as well. The patient was adamant about not wanting hemodialysis and, as he stated, "he would rather die than be stuck to a hemodialysis machine for the rest of his life". He decided to think about his options and return to Amarillo, admittedly not sure what he would do if he developed severe chest pain again, which is what happened.

This is when I saw him. He had significant ST-T wave changes on the electrocardiogram, a creatinine of 1.93 mg/dL and an estimated GFR of 34. I went over the management options with the patient and his wife and reorganized his medical therapy. He stabilized and was able to go home. He returned to the clinic with the previous outside angiogram films. As mentioned above, he had every risk factor in the book for increased morbidity or mortality for coronary bypass surgery. At this point, we discussed potentially undergoing coronary interventions on a staged procedure, meaning

that we would take care of one vessel (the right coronary artery) first. We would wait for 2 to 4 weeks to allow the renal function to recover and then proceed with high-risk coronary intervention to the left main coronary artery lesion with a bifurcation stent to the left anterior descending and the circumflex coronary arteries with Impella® support. It was a do or die moment for the patient, because his symptoms had become so frequent that he had a feeling of impending doom. Finally, he decided to proceed with the staged PCI approach that I had recommended.

Numerous studies have indicated that a 12-hour IV hydration prior to any contrast-requiring procedure reduces the chance of acute tubular necrosis and renal failure, so the patient was admitted to the hospital for intravenous hydration before the procedure. He then underwent successful right coronary intervention with 2 drug-eluting stents. He was monitored overnight in the hospital, was started on Plavix and aspirin and was discharged home next day without any significant deterioration of renal function.

We waited for about 4 weeks to make sure the renal function remained stable. He then returned for a high-risk PCI. Because he had previous aortobifemoral bypass graft, an access site was carefully chosen into the distal common femoral artery/right proximal SFA, so as not to disturb the right limb of the bypass graft. Because of his left main coronary artery disease, any attempt to perform a balloon angioplasty (temporarily occluding the blood flow through the left main coronary artery), even for a few seconds would risk sudden cardiogenic shock or a malignant ventricular arrhythmia, resulting in sudden death on the table. That is where the Impella® support played a major role. Through the distal right common femoral artery, a 14 French sheath (4.66 mm in size) was placed. A single access technique was used so that there was no need for a second arterial line. Under fluoroscopy, the Impella CP® device was advanced into the left ventricular cavity. Through the same sheath a second access was performed and a 6 French sheath was introduced, through which a specialized guide catheter was then advanced to the left main coronary artery. After about 2 hours of complex maneuvering, requiring intravascular ultrasound and high-pressure balloon angioplasty of heavily calcific coronary plaque within the left main, left anterior descending and left circumflex coronary arteries, 3 drug-eluting stents were eventually placed, recreating a Y shaped bifurcation and reconstituting a fully patent intravascular lumen of the left main, LAD and circumflex coronary arteries. Less than 50 cc contrast was used. At the end of the procedure, the sheath including the Impella® device was removed and a specialized percutaneous suture called Perclose® was applied with 2 simultaneous crisscross sutures, immediately controlling the access site without any evidence for bleeding or hematoma. The distal pedal pulses were monitored and remained palpable. The patient continued IV hydration for additional 12 hours. He was monitored overnight in the hospital and discharged home next day. 2 years later he continues to be fully functional without any episodes of angina and stable renal function. He is an extremely happy patient who avoided

being on hemodialysis and had successful outcome with immediate return to normal activities.

IMPELLA® SUPPORT FOR COMPLEX PCI: AN IMPORTANT INNOVATION

From the 1980s to early 2010's, high risk patients undergoing coronary interventions or coronary artery bypass surgery would undergo placement of intra-aortic balloon pump (IABP) through the femoral arteries. In addition, high-risk patients with unrelenting chest pain or in cardiogenic shock would often require the IABP, which would add about 1 L/min of extra cardiac output to the patient's already sick cardiovascular system. It did not help all patients but that was the best thing available at the time. Many patients did very poorly. This has changed dramatically after the advent of Impella® mechanical assist devices. Impella® devices are percutaneous (i.e., they can be performed in the cath lab) and they can enhance cardiac by up to 3 L/min. This equates to a mean arterial pressure of 65 mmHg. Maintaining a mean pressure of 65 mmHg means that there is adequate brain perfusion and adequate kidney perfusion during and after the high-risk procedures. Numerous trials and articles have provided positive evidence in support of the Impella® devices. High-risk coronary interventions, such as in our patient, can be more safely and effectively performed, returning our patients to their normal lifestyle. Many cardiogenic shock patients have been able to be bridged with the Impella® device for a few days and then successfully weaned and discharged home, or else stabilized for subsequent coronary artery bypass surgery.

As a physician, nothing gives me more pleasure than seeing patients who recover after successful complex coronary interventions, able to go home with vital organ function intact and to resume their normal lifestyle. Impella® percutaneous mechanical assist devices have truly changed the game for high-risk PCI's. We should be proud that the hospitals in Amarillo can provide such a high-level service.

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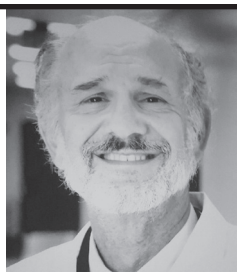
The History of Cardiac Catherization

by Rouzbeh K. Kordestani, MD, MPH and Steve Urban, MD, MACP



Cardiovascular disease-related deaths are the chief cause of mortality and mortality in the modern age. In the year 2000, global data showed that more than 14 million individuals died of cardiovascular related diseases. In the United States, more recent data (2021) shows that almost 700,000 deaths were directly related to heart disease.

With such a tremendous mortality related to one category of disease, it is no wonder that there is and always has been such a focus on its underlying physiology. Many thousands of publications have been dedicated to heart disease, and it has been the focus of millions of hours of intense study and billions of dollars in research. The Centers for Disease Control (CDC) estimates the impact of cardiovascular disease (research included) to be $216 billion a year, with an additional $147 billion a year due to lost productivity.

EARLY FOCUS ON PHYSIOLOGY OF THE HEART

As the numbers demonstrate, cardiovascular disease and its treatment are mainstays of our health industry. This is not a new focus. Early physicians and physiologists, beginning with William Harvey (De Motu Cordis, 1628) studied the circulatory system, but they were hampered by the lack of precise instrumentation. In 1711, Stephen Hales, an English clergyman, reported the first accurate blood pressure readings using a series of brass pipes inserted into the veins and arteries of horses. Hales was an interesting scientist. He spent his entire adult life as a vicar in the small English town of Teddington but, on the side, was an amateur scientist, studying respiration in plants, developing a device to collect gasses for study, and doing extensive research into animal physiology. He was elected to the Royal Societies in London, Paris, and Bologna and became world famous for his investigations. Not everyone agreed with his techniques, however; the poet Thomas Twining wrote:

> Green Teddington's serene retreat
> For philosophic studies meet,
> Where the good pastor Stephen Hales
> Weighed moisture in a pair of scales,
> To lingering death put mares and dogs,
> And stripped the skins from living frogs,
> Nature he loved, her works intent
> To search and sometimes to torment.

Hales' early success was furthered by Claude Bernard and Gustav Magnus in the 19th century. Magnus was a chemist at the University of Berlin, while Bernard was professor of physiology at the Sorbonne in Paris. They were both proponents of careful and precise measurements in the research lab. Bernard in particular was an opponent of "vitalism" (the idea that living things did not obey the natural laws of the inanimate world) and essentially established physiology as a proper field for academic study. He and his followers did many experiments trying to ascertain the mysteries of the vascular system. In 1844, Bernard, using a horse model, accurately measured cardiac output by gauging the temperature differences in the various parts of the animal heart by "retrograde advancement" (catherization) of the jugular and carotid vessels. (A refined version of this thermodilution method is still used to measure cardiac output today). Bernard's experiments also showed that heart catheterization could be completed without sacrificing the study animal. At the time, however, such invasive measurements on human subjects were strictly forbidden.

In the early 1900's, Unger, Bleichroder, and Loeb began experimenting on the administration of cardiac drugs through catheters either directly or indirectly in the vicinity of the heart. Since human studies were prohibited, they chose to use themselves as subjects. They showed that accessing the central system was possible through the basilic and cephalic veins. Unfortunately, in one of these experiments, Bleichroder himself suffered significant chest pain; it is likely that the catheter used may have actually injured his heart.

Unaware of these contemporary experiments, Werner Forssmann, a 24-year-old graduate of the University of Berlin, became fascinated with the idea of cardiac catheterization. Without access to any human subjects, he decided to experiment on himself. With the help of one of his surgical nurses, he used a urology catheter and advanced it through his cephalic vein into his own right atrium. He confirmed the catheter placement by X-ray. He repeated this study several times with no adverse cardiac sequalae. Unfortunately, in 1919 when he published his data (1), he did not receive the recognition he expected. Instead, the medical community was shocked and dismayed. Because of his experiments, his medical license and his medical privileges were temporarily suspended.

CARDIAC CATHERIZATION COMES OF AGE

Other European physiologists continued to expand on the promise of cardiac catheterization after Dr. Forsmann. However, in 1941, two physicians, Andre Cournand and D.W. Richards, at New York's Bellevue Hospital, began intense investigations (2). They first confirmed Forsmann's experiments, adding asep-

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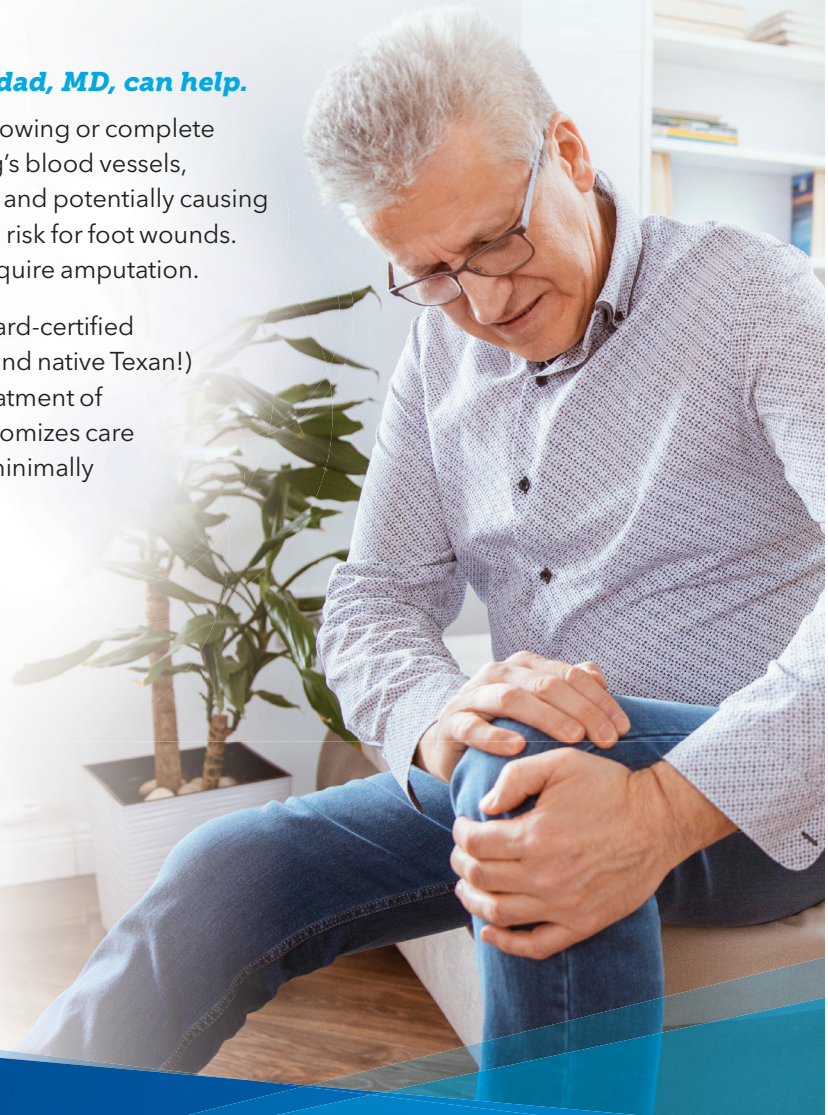

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tic techniques as a corollary. They then began developing in situ catheter modifications to allow the catheters to be placed for extended periods of time. They reasoned that the catheter would be most useful if used not only for monitoring but also for drug administration. With a dedicated staff and a university-based laboratory, they began enrolling patients and completing a series of catheterization studies (3,4). Once their initial trials met with success, they enrolled their first cohort of 260 patients, with pressure measurements and the calculation of cardiac output. They also showed that monitoring could be done for periods of 24-48 hours with few complications. Further studies at Bellevue hospital allowed Cournand to define and catalogue the pulmonary vascular system. Eventually, using their precise measurements, cardiac physiology in humans became much more clearly understood.

In 1958, the work of Forssman, Cournand, and Richards was recognized with the Nobel Prize for medicine. Despite the priority of his work, Forsmann's achievement was downplayed, since in 1932 he chose to join the Nazi party and was dedicated to its goals until the end of World War II.

CORONARY ANGIOGRAPHY

Modern heart catheterization involves imaging and dilation of the coronary arteries, not just measurement of pressures and functional parameters. The first halting steps toward coronary imaging were taken by Charles Dotter, a radiologist at the University of Oregon; his early animal studies (in the mid-1950s) involved transient occlusion of the aorta (!!!) with retrograde injection of dye into the coronary arteries. The first (inadvertent) coronary angiogram in human beings was performed by Mason Sones at the Cleveland Clinic. While attempting to do an aortogram, the catheter slipped into the right coronary artery and a huge bolus of IV contrast was injected directly into the RCA. The patient went into asystole but was able to be resuscitated. In spite of this terrifying complication, Sones went on to study coronary arteriography in animals. After developing better catheters and imaging techniques, Sones and his colleagues began to image the coronary arteries in human beings.

Another pioneer in the field was Melvin Judkins, who studied both with Dotter and Sones and who developed catheters that were properly shaped to selectively catheterize the individual coronary arteries. Judkins later returned to the University of Oregan, which joined the Cleveland Clinic as an important center for early cardiac imaging. The role of coronary angiography took a quantum leap forward when it became possible to do something about the results. The first coronary artery bypass graft (CABG) surgery was performed by the Argentinian Rene Favoloro, working with Sones' group at the Cleveland Clinic, in 1967. By the mid-1980s, over 300,000 coronary angiograms were performed in the U.S. per year, and CABG had become one of the most commonly performed surgeries in the land.

PERCUTANEOUS CORONARY INTERVENTION (PCI)

Originally, heart caths and coronary angiograms were just diagnostic techniques—that is to say, if you found one (or more) severely narrowed coronaries, the only thing to do was to call the surgeon for a CABG. Even medical management was rudimentary in those days—just nitrates, blood pressure control and life-style modifications.

This changed in 1977 when Andreas Gruntzig at the University of Zurich developed an inflatable balloon-tipped catheter that could dilate narrowed coronary arteries without the need for open surgery (5). The era of percutaneous coronary intervention (PCI) had begun, but a significant problem with the balloon technique soon became apparent. Not surprisingly, the process of crushing the atherosclerotic plaque into the wall of the coronary artery led to damage of the inner lining of the artery (the endothelium), and the damaged artery often rapidly re-stenosed. A solution to this came a decade later (in 1989) when Julio Palmaz and Richard Schatz (Palmaz at the University of Texas at San Antonio, Schatz at Brooke Army Meical Center, also in San Antonio) developed the first balloon-expandable stents (5). Now, with the use of balloon angioplasty and the most recent of many generations of expandable stents, many cases of coronary stenosis are treatable with percutaneous (i.e., non-operative) means alone.

One more innovation to mention here came about in 1980, when Marcus DeWood and his colleagues in Spokane, WA performed the first coronary angiograms in patients with an acute heart attack (myocardial infarction)(6). At this time, the medical community was shocked that anyone could be so reckless as to do a heart cath in the midst of a heart attack, but these (and other) investigators demonstrated that, in 70% of cases with ST segment elevation, the culprit coronary artery was not just narrowed (as was thought to be the case at the time), but completely occluded with a clot. Although DeWood had only very crude ways to disrupt the clot, this insight led to a complete transformation in the way that ST-elevation MIs are managed—initially with clot-busting drugs (thrombolytics) and then with catheter-based angioplasty and stenting. Nowadays, an acute heart attack is often managed aggressively—a race against the clock to get the artery re-opened in time (preferably 90 minutes or less) to save the threatened heart muscle cells. Our understanding of the role of thrombosis in the setting of a ST segment elevation MI came about only through insights discovered in the heart cath lab.

CONCLUSION

Over 1 million cardiac catherizations are done in the United States annually. In light of the impact that cardiovascular disease has on the population at large, it is hard to think of a world without this technology that we now consider routine. However, the development of cardiac

catherization was dependent on the dedication of a multitude of individuals who did not surrender their goals and decided to further the field of science even at the risk of losing their livelihood, their careers or their own lives.

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Sinus Node Dysfunction Following Cardioversion for Atrial Flutter/Fibrillation: A Case Report and History of Cardioversion

by Basak Basbayraktar, MD
2nd year IM resident, TTUSOM (Amarillo)

ABSTRACT

Severe junctional bradycardia (JB) due to sinus node dysfunction (SND) is a rare complication following cardioversion for atrial flutter/fibrillation (AF). We present a case of a 73-year-old male with recently diagnosed AF who developed junctional bradycardia and SND following cardioversion. While cardioversion effectively restores sinus rhythm, it may precipitate bradycardia and SND, posing challenges in patient management as it did for our patient, who required pacemaker placement due to persistent bradycardia. This report presents the case and explores the historical context, prevalence, and incidence of post-cardioversion complications.

BRIEF HISTORY OF CARDIOVERSION

The historical evolution of cardioversion underscores its significance in arrhythmia management. Cardioversion can be traced back to the 18th century, when French physician Peter Christian Abilgaard conducted pioneering experiments using electrical charges to revive animals from lifelessness (1). Abilgaard's landmark 1775 experiment, involving the application of electrical charges to a deceased chicken, laid the groundwork for modern cardioversion techniques. Inspired by Abilgaard's work, Benjamin Franklin's experiments with electricity further fueled the exploration of cardiac interventions, with less avian involvement. Franklin's endeavors, albeit daring, contributed to the broader understanding of electrical principles in medicine.

The true advent of cardioversion as a therapeutic modality emerged in the mid-20th century, propelled by the seminal work of Dr. Bernard Lown and his associates at Brigham and Women's Hospital in Boston, MA. Lown's experiments with direct-current (DC) cardioversion on postoperative patients marked a paradigm shift in cardiac care. His landmark 1963 publication validated the efficacy of synchronized DC shocks in restoring normal sinus rhythm, laying the foundation for contemporary cardioversion techniques (2).

Electrical cardioversion, pioneered by Lown and colleagues, has become a mainstay in AF treatment. Synchronized DC discharge terminates arrhythmias, restoring normal sinus rhythm. Biphasic waveform devices offer advantages over monophasic models, including reduced energy requirements and tissue damage.

CASE PRESENTATION:

Sinus node dysfunction (SND), characterized by tachycardia-bradycardia syndrome, commonly complicates AF management, affecting up to one in five patients with AF. AF-induced remodeling, including atrial fibrosis and altered gene expression, contributes to the pathogenesis of SND. While effective in restoring sinus rhythm, DC cardioversion may precipitate SND, necessitating pacemaker placement in select cases.

Our patient, a 73-year-old male with a history of recently diagnosed AF, developed with symptomatic bradycardia following cardioversion. The junctional bradycardia persisted despite medical therapy. Telemetry revealed sinus pauses lasting 5 to 6 seconds, and the patient required an isoproterenol drip to maintain heart rate. Despite interventions, intermittent bradycardia persisted, necessitating permanent pacemaker implantation, which led to improvement of the heart rate and resolution of his symptoms.

DISCUSSION:

SND complicating cardioversion for AF poses diagnostic and therapeutic challenges. While the pathophysiology remains unclear, atrial remodeling and autonomic dysregulation are implicated. Management often involves temporary pacing and pharmacotherapy, before definitive pacemaker placement. Our patient responded well to isoproterenol drip but ultimately required permanent pacing.

The etiology of junctional bradycardia is multifactorial and involves complex interactions between procedural effects and underlying atrial pathology. SND affects up to 20% of patients with AF. Junctional bradycardia, characterized by abnormal electrical conduction within the atrioventricular junction, is a common manifestation of SND post-cardioversion. Studies suggest that a significant proportion of patients undergoing cardioversion experience usually transient or rarely persistent SND and junctional bradycardia. During cardioversion, the energy can affect discrete lesions in the atrial tissue, leading to inflammation, edema, and subsequent fibrosis. These changes disrupt normal atrial conduction pathways, impairing impulse propagation and predisposing to bradycardia. Moreover, the proximity of critical cardiac structures, i.e., the sinus node and atrioventricular node, increases the risk of conduction disturbances. Inflammation-mediated changes in autonomic tone and sympathetic activity further contribute to the development of junctional bradycardia.

CONCLUSION:

The historical evolution of cardioversion underscores its pivotal role in arrhythmia management. From Abilgard's experimental endeavors to modern techniques pioneered by Lown and colleagues, cardioversion has revolutionized cardiac care. However, complications such as SND and junctional bradycardia underscore the importance of vigilant monitoring and individualized patient care.

This case underscores the importance of considering SND as a potential complication following cardioversion for AF and highlights further efforts that are needed to optimize cardioversion strategies and minimize adverse outcomes in patients with AF.

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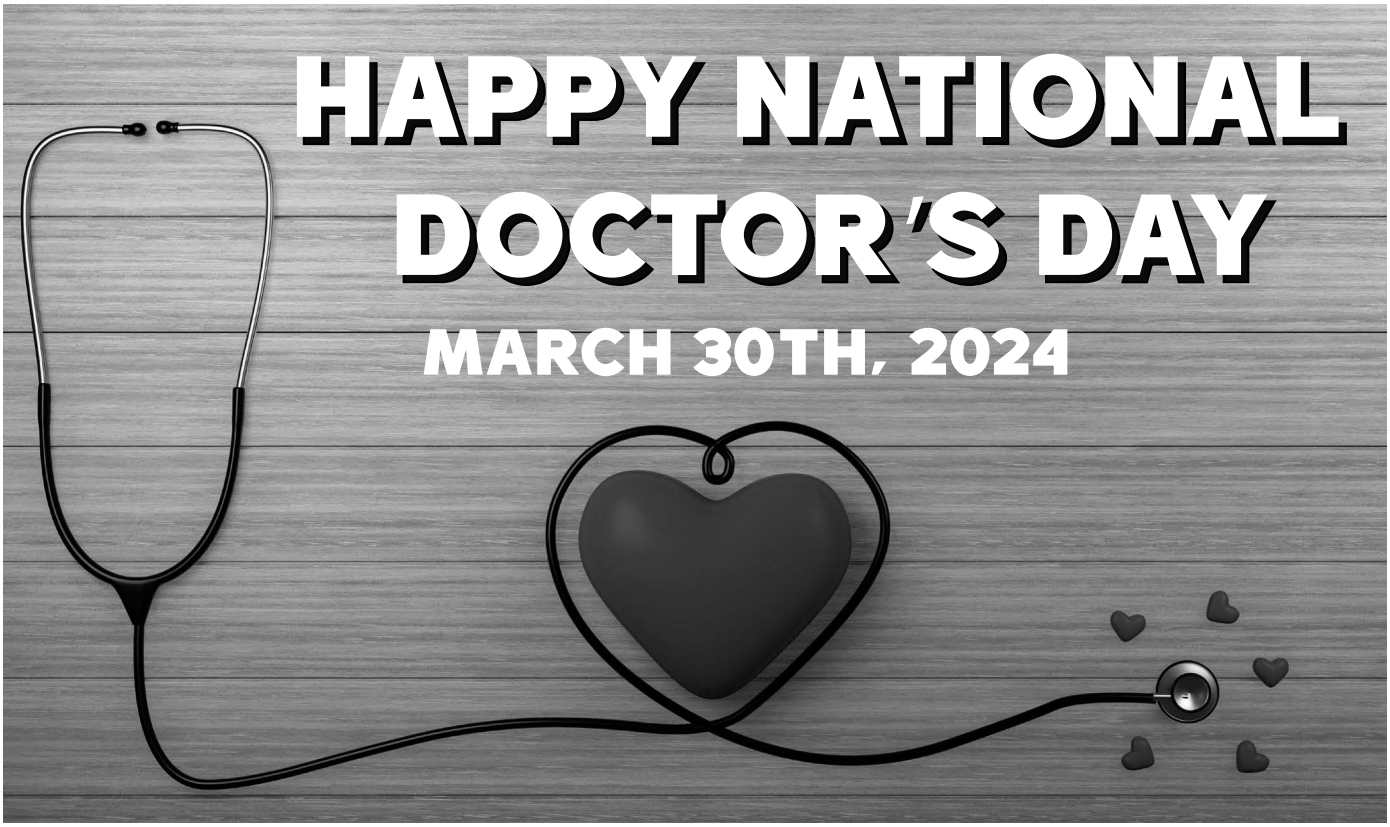

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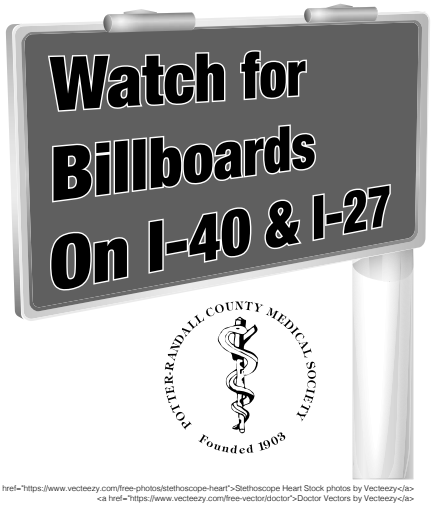

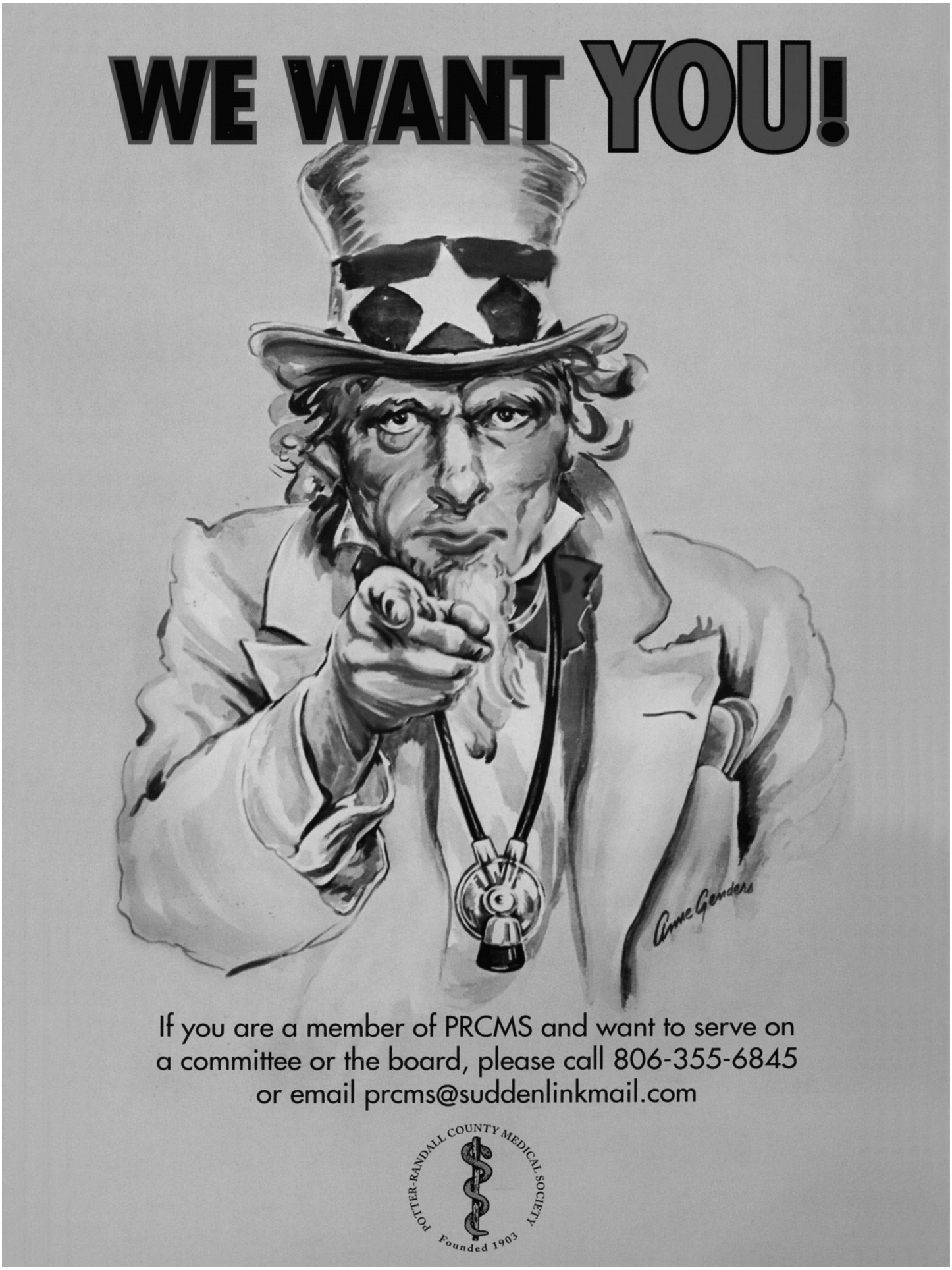
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