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Clinical Cardiology [ALERT]

Critical analysis of the latest clinical research in cardiovascular medicine

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ABSTRACT & COMMENTARY

Study Adds Complexity to Post-TAVR Anticoagulation

By Jeffrey Zimmet, MD, PhD

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Dr. Zimmet reports no financial relationships relevant to this field of study.

SYNOPSIS: In this large contemporary, prospective transcatheter aortic valve replacement registry, oral anticoagulation appears to be protective against valve degeneration but is associated with increased mortality. The strongest predictors of mortality at three years were male gender, renal failure, and atrial fibrillation.

SOURCE: Overtchouk P, Guedeney P, Rouanet S, et al. Long-term mortality and early valve dysfunction according to anticoagulation use: The FRANCE-TAVI registry. *J Am Coll Cardiol* 2018; Aug 22. pii: S0735-1097(18)36960-2. doi: 10.1016/j.jacc.2018.08.1045. [Epub ahead of print].

The story of anticoagulation after transcatheter aortic valve replacement (TAVR) starts with the design of the pivotal trials, which prescribed dual antiplatelet therapy (DAPT) for a period of three to six months after deployment. That decision resulted from expert cardiologist opinion at the time (the TAVR valve looks like a giant stent, after all) rather than from patient-level data. Although DAPT remains the standard recommendation for these devices, many patients are treated with single antiplatelet therapy or oral anticoagulation, depending on the characteristics of the patient and the experience of the treatment center.

Although valve thrombosis causing hemodynamically significant early valve failure is rare, several studies in recent years have demonstrated that a significant percentage (as high as 20% in some series) of TAVR valves develop imaging findings of organized thrombus that are seen on 4D-CT or on transesophageal echocardiography (TEE). These are too subtle to be seen on the transthoracic echocardiograms (TTE) that are performed more typically as follow-up in TAVR patients. There is increasing recognition that relatively modest increases in transvalvular gradients seen by standard echo may represent early valve thrombosis. Importantly, treatment of such patients with warfarin

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typically results in resolution of the thrombosis. In addition, patients post-TAVR who are maintained on oral anticoagulants demonstrate a very low rate of these thrombotic imaging findings.

FRANCE-TAVI is a large prospective, nationwide registry that contains data on TAVR patients from 48 centers in France that were collected between 2013 and 2015. Overtchouk et al sought to determine whether the type of antithrombotic therapy used post-TAVR affects long-term mortality. Further, they looked for increases in transaortic gradient by TTE as a marker of bioprosthetic valve dysfunction (BVD), defined as an increase in prosthetic gradient of at least 10 mmHg between baseline and follow-up, or a new prosthetic gradient ≥ 20 mmHg on follow-up.

Of the nearly 13,000 patients included in the registry, 11,469 survived to discharge with known antithrombotic treatment and were analyzed for mortality. A total of 2,555 patients had the necessary data from at least two echocardiographic evaluations, and the authors assessed this group for BVD.

In a multivariable regression analysis, chronic renal failure, male gender, and history of atrial fibrillation were the weightiest independent mortality correlates. Anticoagulation exposure at discharge was one of several additional independent correlates of mortality, which also included such factors as nonfemoral access, moderate-to-severe prosthetic regurgitation, and small valve size. In the analysis of valve deterioration, nonfemoral TAVR access and anticoagulation at discharge were found to be protective, while high body mass index, prior TAVR, and chronic renal failure were predictive of BVD. Neither aspirin nor clopidogrel were independently associated with either mortality or BVD.

The authors concluded that male gender, renal failure, and atrial fibrillation affected mortality in the post-TAVR population the most. Anticoagulation decreased the risk of valve dysfunction; nonetheless, it remained independently predictive of mortality.

■ COMMENTARY

Although DAPT remains the standard recommendation, studies to date have not shown any advantage to DAPT vs. single antiplatelet therapy, usually with aspirin alone, while the rate of bleeding episodes increases. In keeping with this, the results of the Overtchouk et al study suggest no mortality advantage to either aspirin or clopidogrel and no protective effect of these medications regarding BVD. The most recent American Heart Association/American College of Cardiology guidelines (2017) assign a IIb indication to prescribing up to six months of clopidogrel in addition to aspirin after TAVR.

Oral anticoagulation has been protective against subclinical valve thrombosis in prior small trials using advanced imaging (4D-CT and TEE) post-TAVR. As expected, FRANCE-TAVI confirms a reduction in BVD with anticoagulation at discharge. Interestingly, the results also suggest that anticoagulation increases mortality independently of atrial fibrillation. Here, the most recent guideline update has gotten ahead of the data, suggesting that it is reasonable to prescribe a vitamin K antagonist for at least three months after TAVR in patients at low bleeding risk. There are a lack of data on the direct oral anticoagulants (DOACs) in this field, with several ongoing trials investigating these agents in post-TAVR patients. One of these trials, GALILEO, ended prematurely in October when investigators found that low-dose rivaroxaban plus aspirin was associated with increased rates of death or a first thromboembolic event (11.4% vs. 8.8%), all-cause mortality (6.8% vs. 3.3%), and bleeding (4.2% vs. 2.4%) compared with DAPT. Full results of that study remain unpublished.

Despite its status as standard therapy, DAPT appears to offer few concrete benefits post-TAVR while conferring higher bleeding risks. We should maintain a low threshold for paring this down to single antiplatelet therapy in patients with significant bleeding risks. Oral anticoagulation, including vitamin K antagonists and DOACs, reduces the risk of subclinical valve thrombosis. However, data available to date suggest this does not improve outcomes. The Overtchouk et al study suggests an association with increased mortality. Until more data are available, oral anticoagulants should be limited to patients with a concrete indication such as atrial fibrillation. ■

ABSTRACT & COMMENTARY

Detection of Prosthetic Heart Valve Endocarditis

By Michael H. Crawford, MD, Editor

SYNOPSIS: A multicenter, retrospective, observational study of patients undergoing ^{18}F-fluorodeoxyglucose PET/CT for prosthetic valve endocarditis or other indications showed that if certain obvious confounders are excluded, this imaging technique offers a high degree of accuracy for diagnosing prosthetic valve endocarditis, especially if performed early in the course of the disease.

SOURCES: Swart LE, Gomes A, Scholtens AM, et al. Improving the diagnostic performance of ^{18}F-fluorodeoxyglucose positron-emission tomography/computed tomography in prosthetic heart valve endocarditis. *Circulation* 2018;138:1412-1427.

Bittencourt MS, Blankstein R. More evidence supporting fluorodeoxyglucose position emission tomography for diagnosing prosthetic valve infective endocarditis. *Circulation* 2018;138:1428-1430.

The use of ^{18}F-fluorodeoxyglucose (FDG) PET/CT imaging has shown promise for detecting prosthetic valve endocarditis (PVE), but several confounders such as the use of surgical adhesives or glue have reduced specificity. The use of quantitative parameters in addition to visual evaluation can overcome some of these limitations. The European Association of Nuclear Medicine Research Ltd (EARL) has proposed standardized calibration and reconstruction methods so that the results are interpreted in a uniform fashion between medical imaging centers.

The purpose of the Swart et al study was to evaluate the diagnostic performance of FDG PET/CT for identifying PVE in a cohort of patients in whom PVE was suspected compared to prosthetic valve patients undergoing FDG PET/CT for other reasons, using the EARL criteria. This retrospective, observational study took place across six centers in the Netherlands and included all patients who received a prosthetic valve and underwent FDG PET/CT between 2010 and 2016 and had at least a one-year follow-up. If there were multiple tests conducted for a patient with suspected PVE, only the first was included. The final diagnosis of PVE was made by expert consensus after one year of follow-up. There were 160 prosthetic valve patients who underwent FDG PET/CT for suspicion of PVE and 77 prosthetic valve patients with other indications for FDG PET/CT.

After one year of follow-up, 80 patients were diagnosed with PVE. Visual assessment for PVE was confounded significantly by the use of surgical adhesives or glue and low C-reactive protein (CRP) values due to prolonged antibiotic use, but not recent prosthetic valve implantation. When patients with significant confounders were excluded, the sensitivity and specificity for diagnosing PVE were 91% and 95%, respectively. When the quantitative EARL criteria were used in those without confounders, sensitivity and specificity were 100% and 91%, respectively. The authors concluded that both visual and quantitative FDG PET/CT imaging offers high diagnostic accuracy for PVE if conducted early and the use of surgical adhesives is considered.

■ COMMENTARY

PVE is notoriously difficult to diagnose. The standard transesophageal echo (TEE) and blood cultures approach has reduced sensitivity for diagnosing PVE compared to such usage in native valve endocarditis. However, the stakes are higher, as late diagnosis often leads to poor outcomes. The European Society of Cardiology (ESC) 2015 guidelines concluded that there were enough data on FDG PET/CT to include its use more than three months after valve replacement for suspected PVE.

The Swart et al study expands the database on FDG PET/CT use in suspected PVE, particularly regarding the effect of confounders that could reduce the accuracy of the test. First, they did not find that scans performed in the first three months after valve implantation were compromised by false-positive results. Thus, the authors could not support the ESC guidelines prohibition against FDG PET/CT use in the first three months after implantation. Other, more recent studies also support this conclusion. Second, Swart et al found that if FDG PET/CT was performed late in the course of PVE, when inflammation has quieted down (CRP < 40 mg/dL), that false-negative studies occurred more often. However, early use of FDG PET/CT in PVE cases with only valve vegetations and no annular inflammation can be negative since FDG PET/CT only identifies inflammation. Thus, Swart et al suggested that FDG PET/CT be used early in potential PVE cases, especially if the TEE is negative for vegetations. Third, the use of surgical adhesives for valve replacement created false-positive results. This knowledge is important for accurate interpretation of the scans. Fourth, Swart et al found that strict adherence to at least 24 hours of a low-carbohydrate diet and a 12-hour fast before the

test to reduce myocardial uptake of FDG enhanced the interpretation of the scans. Finally, the authors showed that a quantitative approach comparing standardized uptake values at the valve with those of the blood pool in the descending aorta were highly sensitive and specific when patients with obvious confounders were eliminated. This new quantitative information using standardized protocols at all institutions is promising, but requires further evaluation. Nevertheless, it has been widely adopted in Europe.

There were limitations to this study. The most obvious is its retrospective, observational design; however, it is unlikely anyone will conduct a randomized, controlled trial that includes these critically ill patients. There probably was some selection bias in who underwent a PET/CT. Also, not all patients were suitable for adhering to a low-carbohydrate diet and a 12-hour fast. Even though myocardial uptake of FDG was not always suppressed, Swart et al did not find this to be a significant issue with scan interpretation. Strengths of the study included the use of two blinded readers and the inclusion of a non-PVE control group. Further, interobserver agreement was very high, but these were highly experienced nuclear medicine physicians. Considering the caveats, when PVE is suspected and the initial TEE is negative, it is reasonable to proceed to FDG PET/CT before the blood culture results return. PVE is a condition with high morbidity and mortality. Early diagnosis could alter the course of the disease. This should be proven in prospective outcome studies, but such investigations will be difficult to conduct. ■

ABSTRACT & COMMENTARY

Mitral Annulus Disjunction and Ventricular Arrhythmias

By Michael H. Crawford, MD, Editor

SYNOPSIS: A patient cohort with mitral annular disjunction (MAD) identified by echo was characterized clinically and by MRI. Ventricular arrhythmias were common in MAD patients and related to the degree of MAD and papillary muscle fibrosis by MRI but not the presence of mitral valve prolapse.

SOURCES: Dejgaard LA, Skjølsvik ET, Lie ØH, et.al. The mitral annulus disjunction arrhythmic syndrome. *J Am Coll Cardiol* 2018;72: 1600-1609.

Basso C, Perazzolo Marra M. Mitral annulus disjunction: Emerging role of myocardial mechanical stretch in arrhythmogenesis. *J Am Coll Cardiol* 2018;72:1610-1612.

Displacement of the left ventricular (LV) basal wall from the mitral annulus or mitral annulus disjunction (MAD) is associated with mitral valve prolapse (MVP), papillary muscle fibrosis, and ventricular arrhythmias. However, these interrelationships are poorly understood. Dejgaard et al sought to clinically characterize patients with MAD and describe MAD anatomy by echocardiography and cardiac MRI.

In two hospitals in Norway, patients with MAD were identified in the echo lab and recruited into the study, which included a new echo, cardiac MRI, ECG, and clinical assessment. Patients with aborted cardiac arrest underwent a full evaluation for etiologies. Patients with a history of ventricular arrhythmias underwent ECG monitoring, exercise testing, or telemetry strips analysis. Of 122 patients identified during screening, protocol echo or MRI revealed the presence of MAD in 116 patients. A history of palpitation was common (71%), and 12% had experienced severe arrhythmic events. MAD longitudinal distance averaged 3 mm by MRI (range, 0-7.9 mm). Circumferential MAD averaged 150° (range, 30° to 240°). Also, MAD was confined to the annulus of the posterior leaflet.

MVP was present in 78% of MAD patients. A 24-hour ECG was obtained for 70% of all patients. Arrhythmia frequency was not different between those with and without MVP. An evaluation did not reveal another etiology for any of the 10 patients with cardiac arrest. In a multivariate analysis, longitudinal MAD distance and papillary muscle fibrosis by late gadolinium enhancement on MRI were associated with ventricular arrhythmias. Patients with severe arrhythmic events more frequently exhibited papillary muscle fibrosis than those without such events (36% vs. 9%; $P = 0.03$). The authors concluded that MAD, but not MVP, was associated with ventricular arrhythmic events.

■ COMMENTARY

Although known about for more than 30 years, MAD generally has been thought of as a subtype of MVP that increases the risk of ventricular arrhythmias. This study and others have shown that MAD can exist without MVP and carries a high risk of serious ventricular arrhythmias (31% in this select series). Paradoxically, this study shows when MVP is associated with MAD, arrhythmic risk is lower (7%). MAD leads to an abnormal curling motion of the basal posterior wall,

which may result in increased wall stress and subsequent ventricular arrhythmias. If the individual also has MVP, this may reduce the wall stress, especially if there is mitral regurgitation, and decrease the incidence of VA. In support of this stress hypothesis, VAs also were associated with the presence of papillary muscle fibrosis on MRI. Further, some have hypothesized that MAD may be the initial lesion, and abnormal stress on the mitral apparatus leads to degeneration of the mitral valve apparatus. However, since there are many causes of MVP, this hypothesis (if true) would only explain a subgroup of MVP patients.

The major limitations to this study were the cross-sectional design with clinical events collected retrospectively and the selection bias of patients in whom an echo was ordered. Thus, the results may not apply to a general population. Also, ambulatory ECGs were not performed for all patients, nor were MRIs. In addition, without follow-up information, it is difficult to estimate the clinical impact of these findings.

Several clinical issues arise from this study. If one incidentally finds MAD in a patient imaged for nonarrhythmic reasons, should the clinician order an ambulatory ECG? If a patient is undergoing surgical mitral valve repair, should the surgeon correct the MAD? How should MAD-associated ventricular arrhythmias be treated: with drugs, ablation, or ICD? Since the MAD distance can vary along the mitral annulus and range from zero to several millimeters in the same patient, should all patients with MVP and no MAD detected undergo an MRI? Can some degree of MAD be a benign normal variant? These and other questions will await further studies of this interesting but potentially lethal condition. ■

ABSTRACT & COMMENTARY

Early Identification of Cardiac Amyloidosis in Carpal Tunnel Surgery Patients

By Van Selby, MD

Assistant Professor of Medicine, University of California, San Francisco Cardiology Division, Advanced Heart Failure Section

Dr. Selby reports no financial relationships relevant to this field of study.

SYNOPSIS: In a cohort of older patients undergoing carpal tunnel release surgery, an analysis of tenosynovial tissue revealed amyloid deposits in 10% of patients. This development may facilitate early diagnosis of cardiac amyloidosis.

SOURCE: Sperry BW, Reyes BA, Ikram A, et al. Tenosynovial and cardiac amyloidosis in patients undergoing carpal tunnel release. *J Am Coll Cardiol* 2018;72:2040-2050.

Amyloidosis encompasses a group of disorders caused by protein misfolding and subsequent aggregation, leading to organ dysfunction. Light chain (AL) and transthyretin (ATTR) amyloid are the two amyloid types that affect the heart most commonly. New therapies are improving outcomes for patients with both ATTR and AL cardiac amyloidosis. However, early diagnosis and prompt initiation of treatment are crucial. Amyloidosis is a systemic disease, often involving noncardiac sites, too. Previous studies have revealed that amyloid, particularly ATTR, frequently deposits in the tenosynovium and transverse carpal tunnel ligaments, leading to carpal tunnel syndrome (CTS). Patients often present with CTS five to 10 years before the diagnosis of cardiac amyloidosis. Patients undergoing carpal tunnel release surgery may present an opportunity to diagnose cardiac amyloidosis earlier in the disease course.

Sperry et al studied men ≥ 50 years of age and women ≥ 60 years of age who underwent carpal tunnel release surgery. Patients with known amyloidosis or CTS thought to be secondary to another condition were excluded. At the time of surgery, surgeons excised a small sample of the tenosynovium, which was evaluated for amyloid deposits using hematoxylin and eosin and Congo red staining. When amyloid was identified, mass spectrometry was used to determine the amyloid type (i.e., AL vs. ATTR). All patients with amyloid identified subsequently underwent thorough evaluation for cardiac involvement, including labs, echocardiography, and nuclear imaging.

Of the 98 patients enrolled, investigators found amyloid deposits in the tenosynovium in 10 patients. The authors found ATTR in seven patients and AL amyloid in two patients. Among the 10 patients with amyloid, two were diagnosed with cardiac amyloidosis and one with amyloid polyneuropathy, allowing early initiation of treatment. Patients with amyloid deposits were older (mean age, 72.5 years) and all had been diagnosed with bilateral CTS. The authors concluded

that tenosynovial biopsy is a low-risk procedure that may lead to early diagnosis of amyloidosis in those with CTS.

■ COMMENTARY

Cardiac amyloidosis has been considered a rare disease with no effective treatments. Because of this, many clinicians do not routinely consider amyloidosis in their evaluation of patients presenting with heart failure. However, recent data suggest that cardiac amyloidosis, particularly ATTR amyloid, is much more prevalent than previously thought. With new treatments available for cardiac amyloidosis, identifying these patients has taken on new importance. Currently available treatments for cardiac amyloidosis work by suppressing the production of new amyloid fibrils or preventing those fibrils from depositing in tissues rather than addressing the amyloid that has deposited in the heart or other organs already. Therefore, earlier identification of amyloidosis and earlier initiation of therapy will prevent accumulation of amyloid fibrils in the first place and improve patient outcomes.

Sperry et al have built on previous studies that identified amyloid deposits in the soft tissues leading to CTS as well as spinal stenosis and biceps tendon rupture. They found that 10.2% of older patients with idiopathic CTS who are referred for surgery exhibit evidence of amyloid deposition, particularly ATTR. Given the frequency of CTS, this represents a large number of patients with potential cardiac amyloidosis. The authors also found that routine evaluation for amyloid led to early initiation of therapy in three patients.

This was a cross-sectional cohort study with important limitations. While the authors identified amyloid in the carpal tunnel specimens of many patients, their study design cannot determine how many of those patients will go on to develop clinically significant cardiac involvement. In the seven patients with amyloid identified on tenosynovial biopsy but without evidence of cardiac amyloid, the appropriate management is unclear. There are no data to support starting amyloid therapies in these patients. Perhaps serial monitoring for development of cardiac disease would be the best approach. Longer-term follow-up of this study cohort is planned and will clarify what proportion of these patients with amyloid will develop cardiac involvement.

Despite these limitations, the findings from Sperry et al carry implications for two different groups of providers: hand surgeons and cardiovascular providers. Hand surgeons should consider developing an algorithm to determine when to send carpal tunnel tissue for pathologic evaluation. Ideally, cardiology providers at larger institutions will work with hand surgeons to develop these protocols and follow the patients in whom amyloid is identified. The other crucial lesson for cardiologists is to remember the link between CTS and cardiac amyloidosis.

When evaluating a patient with heart failure (particularly heart failure with preserved ejection fraction), a history of bilateral CTS, particularly in the five to 10 years before onset of cardiac symptoms, should be a strong clue to screen for cardiac amyloidosis. Once providers start inquiring about CTS, they will be surprised how often CTS appears in many patients' medical histories. A substantial proportion of these patients will, in fact, receive a cardiac amyloidosis diagnosis. With effective treatment for cardiac amyloidosis now available, the cardiovascular community should embrace this opportunity to facilitate early, rapid identification of patients with cardiac amyloidosis. ■

ABSTRACT & COMMENTARY

A Review of Updated Guidelines Regarding Bradycardia and Cardiac Conduction Delay

By Joshua D. Moss, MD

Associate Professor of Clinical Medicine, Cardiac Electrophysiology, Division of Cardiology, University of California, San Francisco

Dr. Moss reports he is a consultant for Biosense Webster and Abbott.

SYNOPSIS: The American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society have established updated guidelines on the evaluation and management of patients with bradycardia and conduction delays. Many key elements remain largely unchanged from prior guideline recommendations on pacemakers published in 2008 and 2012, but there also are important new definitions, recommendations, and areas of emphasis.

SOURCES: Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay. *Circulation* 2018. Available at: https://bit.ly/2RKVDrM. Accessed Nov. 9, 2018.

Slotwiner DJ, Raitt MH, Del-Carpio Munoz F, et al. Impact of physiologic pacing versus right ventricular pacing among patients with left ventricular ejection fraction greater than 35%: A systematic review for the 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay. *Circulation* 2018. Available at: https://bit.ly/2QtCKcl. Accessed Nov. 9, 2018.

For patients with sinus node dysfunction (including sinus bradycardia, sinus pauses, and chronotropic incompetence), particularly with nocturnal bradycardia, evaluation should include consideration of screening for sleep apnea. Treatment of sleep apnea can offer cardiovascular benefits beyond reduction in nocturnal bradycardia events. Permanent pacing is not required if nocturnal bradycardia is the only manifestation of sinus node dysfunction. Importantly (and consistent with prior guideline recommendations), observation alone is appropriate in the setting of sinus node dysfunction without any associated symptoms.

The newly emphasized corollary is the absence of any defined minimum heart rate or pause duration that should prompt pacemaker implant (even in the absence of symptoms). A pacemaker for minimally symptomatic patients with chronic heart rates < 40 bpm while awake is no longer a Class IIb recommendation. Appropriate monitoring to establish correlation between bradycardia and symptoms is critical, including via implantable cardiac monitors if symptoms are infrequent (> 30 days between symptoms).

Atrioventricular (AV) block

As in the past, permanent pacemakers are not recommended for patients with AV block due to a reversible and nonrecurrent cause (such as Lyme carditis).

Permanent pacemakers also may result in harm to asymptomatic patients with first-degree AV block, second-degree Mobitz type I (Wenckebach) AV block, or 2:1 AV block believed to be at the level of the AV node. However, a permanent pacemaker is recommended now, regardless of symptoms for patients with acquired second-degree Mobitz type II AV block, complete (third-degree) AV block, and high-grade AV block (defined as ≥ 2 consecutive P waves at a constant physiologic rate that do not conduct to the ventricles).

Previously, for asymptomatic patients with these more dangerous types of AV block, a permanent pacemaker was considered a Class IIa recommendation rather than this new Class I recommendation.

Conduction disorders with 1:1 AV conduction

More emphasis is placed on evaluating patients with intact AV nodal conduction but evidence of infranodal conduction disease, particularly left bundle branch block (LBBB). Newly diagnosed LBBB should prompt evaluation for structural heart disease, starting with an echocardiogram, given the strong association between LBBB and left ventricular systolic dysfunction.

Ambulatory monitoring for AV block also is recommended for symptomatic patients with LBBB. An electrophysiology study is reasonable if there are intermittent symptoms suggestive of bradycardia, such as lightheadedness or syncope, that cannot be captured on monitoring.

Physiologic pacing

The new guidelines speak more to the increasingly appreciated risks of heart failure and atrial fibrillation associated with chronic right ventricular (RV) pacing. This applies particularly to those patients who do not already suffer from impaired left ventricular function (ejection fraction, ≤ 35%) and would already qualify for cardiac resynchronization therapy (CRT) via biventricular pacing.

For patients expected to require ventricular pacing > 40% of the time because of AV block and who have a left ventricular ejection fraction between 36% and 50%, pacing methods that maintain more physiologic ventricular activation are now a Class IIa recommendation based on recent randomized studies.

Such pacing methods include CRT and His bundle pacing (included for the first time in published guidelines). That technique, described for temporary pacing as far back as the 1960s and with permanent pacemaker leads almost 20 years ago, has grown in popularity and garnered research interest in the electrophysiology community recently. A pacing lead is implanted directly into or near the bundle of His using specialized lead delivery tools, depolarizing the ventricles via the His-Purkinje system.

The resultant paced QRS complex is narrow and sometimes indistinguishable from the native conducted QRS complex, with an isoelectric segment between the pacing artifact and QRS complex that can be mistaken for lack of pacemaker capture. More research is required before the technique is adopted for more indications, but studies thus far suggest a lower likelihood of deleterious effects compared with traditional RV pacing. ■

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CME/CE QUESTIONS

1. Mitral annulus disjunction has been associated with:

a. mitral valve prolapse.

b. ventricular arrhythmias.

c. papillary muscle fibrosis.

d. All of the above

2. Under newly published guidelines, a permanent pacemaker is indicated if:

a. there are overnight heart rates in the 30s with pauses up to four seconds.

b. there is asymptomatic Mobitz type I block (Wenckebach).

c. there is asymptomatic Mobitz type II block.

d. a patient is minimally symptomatic, with a chronic heart rate < 40 bpm.

3. The results of a recent study suggest that appropriate antithrombic therapy for early post-TAVR patients without atrial fibrillation should be:

a. single antiplatelet therapy (e.g., aspirin).

b. dual antiplatelet therapy.

c. warfarin.

d. warfarin plus single antiplatelet therapy.

4. FDG PET/CT imaging for the detection of prosthetic valve endocarditis is less accurate if:

a. performed early in the course of the disease.

b. surgical adhesives were used for implantation.

c. performed within three months of valve implantation.

d. the patient maintained a low carbohydrate diet for > 24 hours.

5. Patients with cardiac amyloid may present with a prior history of:

a. bilateral carpal tunnel syndrome.

b. biceps tendon rupture.

c. spinal stenosis.

d. All of the above

CME/CE OBJECTIVES

Upon completion of this educational activity, participants should be able to:

- discuss the most current information related to cardiac illness and the treatment of cardiac disease;
- explain the advantages and disadvantages, as well as possible complications, of interventions to treat cardiac illness;
- discuss the advantages, disadvantages, and cost-effectiveness of new and traditional diagnostic tests in the treatment of cardiac illness; and
- discuss current data regarding outpatient care of cardiac patients.

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