LVEF <40%, an analysis of the DAVID trial also found the cutoff of 40% pacing as a predictor of increased HF adverse events.[S6.4.4.1-38] Although there is unlikely a precise value for RV pacing burden where adverse remodeling occurs in all pacemaker patients, a cutoff of at least 40% RV pacing is where increases in left ventricular end-systolic volume index and decreases in LVEF have been demonstrated.[S6.4.4.1-11,S6.4.4.1-13,S6.4.4.1-18,S6.4.4.1-39,S6.4.4.1-40] In patients with an LVEF of >50%, CRT has not been associated with increased exertional capacity or improved QOL compared with RV pacing.[S6.4.4.1-15,S6.4.4.1-16,S6.4.4.1-20,S6.4.4.1-21,S6.4.4.1-36] Although the BLOCK-HF trial demonstrated benefit with CRT compared with RV pacing, the benefit was attributable to improved LV function with CRT rather than worsened LV function with RV paving and algorithms that minimize ventricular pacing were unavailable.[S6.4.4.1-41]

6. His bundle pacing is another promising pacing option because it prevents or mitigates the ventricular dyssynchrony and mechanical adverse remodeling observed with RV pacing.[S6.4.4.1-23] Two small crossover studies showed mixed results in terms of improvement in New York Heart Association class, 6MHW, and LVEF but overall seem to show a reduction in left ventricular end-systolic volume index and improvement in LVEF.[S6.4.4.1-22,S6.4.4.1-25] One nonrandomized study did show a reduction in HF hospitalizations compared to RV pacing in the group pacing >40%.[S6.4.4.1-24] A recent study found that His bundle pacing was associated with a significant decrease in heart failure hospitalizations particularly in those patients with ventricular pacing >20% compared with RV pacing.[S6.4.4.1-42] Although a progressive increase in thresholds was identified in a small number of patients, His bundle pacing has been shown to be feasible in patients after atrioventricular nodal ablation.[S6.4.4.1-42,S6.4.4.1-43] More studies are needed to better characterize His bundle pacing and compare it to RV and CRT pacing in atrioventricular block patients.
7. In patients with permanent AF with no plans to attempt rhythm control, there is no need to pace the atrium and no benefit to sensing the atrial activity. Given that dual chamber systems have a higher peri-operative complication rate as well as a higher, long-term complication rate,[S6.4.4.1-8,S6.4.4.1-26,S6.4.4.1-27] exposing the patient to the risk of an additional lead without any potential benefit does not make clinical sense. In a large Dutch pacemaker registry looking at pacemaker implants from 2003 to 2007, and complications within 2 months of implant, a HR of 3.09 for dual chamber pacemakers compared with single chamber was seen.[S6.4.4.1-27] In contrast to new implants, if the patient already has an existing dual chamber system, and subsequently develops persistent AF, it may be reasonable to use another dual chamber device at the subsequent generator change as an alternative to capping the atrial lead to allow future attempts for rhythm control and because of the sparse data on the safety of MRI in the setting of abandoned leads.[S6.4.4.1-44]

7. CONDUCTION DISORDERS (WITH 1:1 ATRIOVENTRICULAR CONDUCTION)

This section focuses on QRS abnormalities caused by fascicular blocks and bundle branch blocks caused by delayed or blocked conduction within ≥1 of the branches of the His-Purkinje system, which consists of the division of the His bundle into left and right bundle branches, followed by division of the left bundle into anterior and posterior fascicles. The combination of delayed or blocked conduction of the right bundle branch and 1 of the left bundle's fascicles is denoted bifascicular block (which also includes LBBB). Although first-degree atrioventricular block is more accurately a conduction disorder rather than atrioventricular block, for historical reasons full discussion and recommendations on this condition are provided in the section on atrioventricular block.

7.1. Pathophysiology

The normal conduction axis consists of the sinus node, atrial muscle, atrioventricular node, His bundle, bundle branches, fascicles, Purkinje fibers, and ventricular muscle. The pathophysiology involved in conduction disease may be developmental, hereditary/genetic, metabolic, infectious, inflammatory, infiltrative, traumatic, ischemic, malignant, or degenerative. In general, it may be helpful to characterize the process as static or progressive.

7.2. Etiology/Classification

There are a number of possible etiologies for conduction disorders with 1:1 atrioventricular condition that the clinician should consider (Table S3 in Web Supplement).

7.3. Clinical Presentation

Isolated fascicular and bundle branch blocks are rarely associated with symptoms on their own although their presence may be a marker for underlying structural heart disease and cardiac dyssynchrony from LBBB may cause symptoms particularly in the setting of reduced