Verapamil-sensitive fascicular ventricle tachycardia is the most common form of idiopathic left ventricle tachycardia (ILVT). It was first recognized as an electrocardiographic entity by Zipes et al, and further elaborated by others. Reentry involving the left His-Purkinje system (HPS) was thought to be the underlying mechanism. Ablation directed at the earliest retrograde presystolic potential (PP) or diastolic potential (DP) was proved feasible and effective. Functionally, the main branch of left HPS is insulated from the adjacent myocardium and exhibits isolated conduction during sinus rhythm (SR), but in vivo study in human beings is very limited. Meanwhile, whether the isolated conduction also exists during idiopathic left ventricle tachycardia (ILVT) is not clearly defined. The current study aimed to delineate the activation sequence and gross anatomy of left HPS during SR and ILVT.

Methods

Study Population
The study involved 25 consecutive patients with ILVT. During SR, left HPS exhibited antegrade activation sequence, and its surrounding myocardium depolarized after HPS in an apical to base direction. During ILVT, the earliest retrograde presystolic potentials were mainly located at the middle portion of left posterior fascicle (0.5±0.1 [95% confidence interval, 0.46–0.58] of its full length) with an average of 29.5±6.0 mm (19.8–41.5) away from the His position. Left posterior fascicle was depolarized from the earliest retrograde presystolic potentials via 2 opposite wavefronts with significantly shorter activation time than that during SR (15.1±2.1 versus 30.0±3.2 ms; P<0.001). The left anterior fascicle was depolarized after left posterior fascicle with an antegrade activation sequence and comparable activation time with that during SR (21.9±2.9 versus 22.0±4.1 ms; P=0.932). The depolarization of ventricle septum also occurred after HPS in an apical to base direction.

Conclusions
During SR, isolated conduction within the HPS is demonstrated by documenting the reverse activation sequence with its surrounding myocardium. During ILVT, the earliest retrograde presystolic potentials were usually recorded at the middle segment of left posterior fascicle, and the isolated conduction within the HPS remained.

Key Words: His-purkenje system ■ isolated conduction ■ ventricular tachycardia
and ablation. Bipolar intracardiac electrograms were filtered (30–500 Hz) and obtained via electrophysiology system (Labsystem Pro, Bard Electrophysiology, Lowell, MA).

Mapping was initially performed during SR, and the Left HPS and LV were reconstructed by mapping the antegrade PPs and ventricular potentials, respectively, where the PPs with the largest amplitude were tagged as main branch of HPS (Figure 1).

The common branch from the His potentials recording position to the bifurcation of fascicles was defined as common trunk of left bundle branch (LBB). The left anterior fascicle (LAF) and left posterior fascicle (LPF), or possibly left middle fascicle (LMF), were defined from the bifurcation to their distal ends (fusion of PPs with the local ventricular potentials).

The activation time of HPS and LV myocardium was calculated by using the onset of QRS on lead V1 as the reference (Figure 1), and their activation sequence was described as antegrade (from the ventricle base to the ventricle apex) or retrograde (from the ventricle apex to the ventricle base) way. The length of LBB, LAF, and LPF was measured via the built-in software of Distance measurement. LV breakthrough site was defined as the myocardial site with the earliest local activation time. The activation sequence was color coded, with the red being the earliest and the purple being the latest, and color between the adjacent sites was interpolated.

To characterize the anatomy of fascicle system, the angle formed by LAF and LPF was calculated according to Cosine theory: \( \cos C = (a^2 + b^2 - c^2) / 2ab \) (here, \( a = \text{LAF length}, \ b = \text{LPF length}, \ c = \text{the length between the distal ends of LAF and LPF} \)). From this, the patients were classified into 3 subgroups according to the cumulative percentage of frequency distribution: small angle group, with the angle degree pertaining to the lower 20% of whole group; middle angle group, with the angle degree pertaining to the middle 60% of whole group; large angle group, with the angle degree pertaining to the upper 20% of whole group.

After mapping in SR, ILVT was reinduced, and activation mapping was performed via the Remap process based on the geometry obtained beforehand. Especially, mapping along the predefined LBB, LAF, and LPF, and possibly LMF was performed to define the earliest

**Figure 1.** Mapping during sinus rhythm. Middle, Activation maps of left His-Purkinje system (HPS; middle, top) and left ventricle (LV) myocardia (middle, bottom), on both sides are surface ECG V1 (black tracing), coronary sinus (blue tracing), and mapping catheter (black tracing) recordings documented along the common trunk of left bundle branch (B1 and B2), left anterior fascicle (LAF; A1 to A4), left posterior fascicle (LPF; P1 to P4), and the myocardial sites adjacent to the distal ends of fascicles (A5 and P5; screen speed of 200 mm/s). By mapping the antegrade presystolic potentials (PP), the gross anatomy and activation sequence of left HPS were reconstructed and tagged as yellow points (middle, top), by mapping the LV potentials, the activation sequence of LV myocardia was revealed (middle, bottom).

The activation time of left HPS and its surrounding myocardium at each position was indicated before the PPs and after the LV potentials, respectively, on the mapping channels (numbers in red, negative indicated earlier than V1, positive indicated later than V1). The left HPS exhibited antegrade activation sequence (middle, top), its surrounding myocardia exhibited retrograde activation sequence, and the LV breakthrough site was adjacent to the distal end of LAF (middle, bottom; position A5). The activation sequence was color coded with the red being the earliest and the purple being the latest, color settings between the adjacent sites were interpolated.

**Figure 2.** Mapping during idiopathic left ventricle tachycardia (ILVT). Continued from the patient in Figure 1, and figure arrangement is same. Activation sequence of left His-Purkinje system (HPS) and left ventricle (LV) myocardia were calculated according to the protocols used during sinus rhythm. Middle, Top. The earliest retrograde presystolic potential (P2) was localized at the middle segment of left posterior fascicle (LPF) with 38 ms earlier than the QRS on V1, and it centrifugally propagated toward the proximal LPF (–P1[−29 ms]→B2[−12 ms]→B1[−2 ms]), and toward the distal LPF (–P3[−29 ms]→P4[−30 ms]), respectively. It also spread across the bifurcation and activated the left anterior fascicle (LAF) antegraderly: –A1[−21 ms]→A2[−14 ms]→A3[−9 ms]→A4[−2 ms]. The LV breakthrough site was adjacent to the distal end of LPF (position P5). Ventricule septum depolarized retrogradely (middle, bottom). Activation sequence of septum along the LPF: →P5[−3 ms]→P4[−3 ms]→P3(5 ms)→P2(10 ms)→P1(15 ms)→B2(21 ms)→B1(29 ms). Activation sequence of septum along the LAF: →A5(2 ms)→A4(5 ms)→A3(9 ms)→A2(14 ms)→A1(18 ms)→B2(21 ms)→B1(29 ms). This indicates that the activations from the myocardial breakthrough site failed to invade distal end of LAF, and the isolated conduction within the left HPS remained during ILVT.
3 divisions conforming to LAF, LMF, and LPF in 2 patients (Figure 4). The LAF was shorter in length than LPF in 23 of the 25 patients, and this resulted in significant difference (23.4±2.8 versus 30.0±3.1 mm; P<0.001). The angle formed by LAF and LPF averaged 57.1°±14.3° (32.4–82.4°). According to the predefined classifications, 4 (16%) patients pertained to small angle group with angle degree <43.1°, 15 (60.0%) patients pertained to middle angle group with angle degree between 43.1° to 71.1°, and 6 (24.0%) patients (including the 2 patients with 3 divisions) pertained to large angle group with angle degree >71.1° (Figure 3).

Both LAF and LPF exhibited antegrade activation sequence. Although the activation time of LAF was significantly shorter than that of LPF (19.9±3.9 versus 30.0±3.1 ms; P<0.001), their conduction velocity is only slightly different (1.1±0.3 versus 1.0±0.2 mm/ms; P=0.027).

During ventricle depolarization, 23 patients (92.0%) presented single breakthrough site, which was in proximity to the distal end of LAF; 2 patients (8.0%) presented 2 simultaneous breakthrough sites, and they were adjacent to the distal ends of LAF and LPF, respectively. During SR, LV septum was depolarized retrogradely in all patients, and the averaged activation time of LV was 53.0±7.9 ms.

Mapping During ILVT
After mapping during SR, ILVT could not be induced in 2 patients. Therefore, mapping during ILVT was performed in 23 patients.

The earliest retrograde PPs were located at the predefined ILVT with an average of 15.6±3.9 mm (10–23.1) away from its distal end and 29.5±6.0 mm (19.8–41.5) away from the His potential recording position. The earliest retrograde PP ratio averaged 0.5±0.1 (95% confidence interval, 0.46–0.58). The earliest retrograde PPs preceded the scalar QRS by 36.2±5.2 ms with an averaged PP-ventricle potential interval of 30.9±3.1 ms.

During ILVT, the LPF proximal to the earliest retrograde PP was activated retrogradely, whereas the LPF distal to the earliest retrograde PP was depolarized antegrade, and total activation time of LPF during ILVT was 15.1±2.1 ms, which was significantly shorter than that during SR (P<0.001). The LAF was depolarized after LPF with an antegrade activation sequence, and its total activation time was 21.9±2.9 ms, which was comparable with that during SR (P=0.932; Figure 2).

Depolarization of myocardium occurred after HPS. The breakthrough site of LV was adjacent to the distal end of LPF, and ventricle septum was depolarized retrogradely (from the breakthrough site to the His region; Figure 2). The activation...
time of LV was 71.3±3.8 ms, which was significantly longer than that during SR (P<0.001). The activation time between the distal ends of LAF and LPF was a difference of 15.2±2.9 ms during ILVT, which was significantly longer than that during SR (8.2±3.2 ms; P<0.001).

Ablation and Follow-up
In all 23 patients with inducible VT, single ablation at the earliest retrograde PPs successfully terminated the VT; in 2 patients with noninducible VT, anatomic ablation at the middle segment of LPF was performed. After an average of 15±3 months follow-up, no patients reported recurrence. All patients remained stable throughout the procedure, no complications occurred immediately after the procedure and during follow-up.

Discussion

Major Findings
Through electroanatomical mapping, the main branches of left HPS could be successfully reconstructed. During SR, the HPS exhibited isolated conduction by documenting reverse activation sequence with its surrounding myocardium. During ILVT, the earliest PPs were usually clustered at the middle segment of LPF, and the isolated conduction within the HPS remained.

Anatomy and Depolarization of Left HPS and LV Myocardia
The anatomy of the left HPS has been a subject of debate. Tawara\(^1\) showed that the bundle radiates in a fan-like fashion with 3 major divisions. Myerburg et al\(^12\) suggested a functional interconnection between the fascicles, adding more information to the debate. The study of Kulbertus indicated that the left bundle could be a bifascicular structure.\(^13\) However, those studies were mainly based on the animal or postmortem examination. So far, in vivo data in human heart was very limited. Ouyang et al\(^7\) have used the CARTO system to map the HPS, but only the LPF was mapped. Meanwhile, they did not map the HPS selectively, and its depolarization sequence was not clearly demonstrated.

The current study found, after an average of 13.8 mm continuation of common trunk, the LBB bifurcated into 2 divisions conforming to the LAF and LPF in 23 patients, into 3 divisions conforming to LAF, LMF, and LPF in 2 patients. The LAF was shorter in length than LPF in 23 of the 25 patients, and this resulted in significant difference. The angle formed by LAF and LPF varied with individuals, but the majority pertained to the middle angle group. However, in some patients, the angle was smaller because of anterior-superior distribution of LPF; in some others, the angle was larger because of posterior-inferior distribution of LPF. Knowing the angle variations has some clinical implications, especially when mapping within the conventional region failed to find LPF, mapping at higher or lower level needs to be considered.

During normal depolarization, the main branch of HPS exhibited antegrade activation sequence, and its surrounding myocardium exhibited retrograde activation sequence. The reverse activation sequence of these 2 adjacent structures provides further proof that the main branch of HPS is insulated by connective tissue, and only bridged to the myocardium at its distal end. In this physiology setting, the HPS and surrounding myocardium resemble the antegrade or retrograde limb of a reentry. If this half-open conduit becomes closed, because of abnormal connections at its proximal, a reentry may develop.
breakthrough site to LAF was explained by the shorter time the activations take to propagate from the His to its distal end. Because the conduction velocity of LAF and LPF was only slightly different, we think it is the shorter length of LAF contributing more to the difference.

Depolarization of Fascicle System and Ventricle During ILVT

Available data suggest the mechanism of ILVT is usually reentrant, involving the affected fascicle and connection strand. The connection strand serves as the antegrade limb, the fascicles serve as the retrograde limb, and the earliest retrograde PP is usually recorded at the beginning of retrograde limb. Partially conforming to those findings, the current study found that the earliest retrograde PP was recorded along the predefined LPF and averaged 15 mm away from its distal end. All VTs could be successfully abolished by ablation at the earliest retrograde PP. This indicates not only the reentry confined to the fascicle system, but also the earliest retrograde PP as the crucial site of ILVT. Meanwhile, the evidence suggests LPF proximal to the earliest retrograde PP is part of the reentry, whereas the LPF distal to it seems to be the bystander only. Via calculating the earliest PP ratio, we found the earliest retrograde PPs were clustered at the middle segment of LPF (95% confidence interval, 0.46–0.58 of its full length). Those findings are suggestive of the middle segment of LPF as the ablation target. In clinical work, ILVT may not be inducible after catheter deployment. To save medical resources, it is reasonable to deliver ablation at this area. Like the 2 patients in the current study, ILVT was eliminated via anatomic ablation at the middle segment of LPF only.

Nogami et al. and Ouyang et al. advocated the DP ablation during their studies. But in clinical work, the DPAs may be recorded within a large area, and it requires more catheters and longer procedure time to differentiate the bystander DPs from the culprit DPs. In some others, only very limited DPs were recorded near the proximal HPS, ablation carries a very high risk of LBB block. In contrast, the earliest retrograde PP site is not only far from the proximal HPS (averaged 29.5 mm away from the His potential recording site as found in current study), but also a target more definite and reproducible in terms of mapping and ablation.

During ILVT, depolarization of LPF begins from the earliest retrograde PPs and spreading via 2 opposite wavefronts: the proximal LPF was activated retrogradely, and the distal LPF was activated antegradely. The LAF was depolarized after LPF and exhibited antegrade activation sequence. Myocardium breakthrough site was switched to the distal end of LPF. Similar to the findings during SR, depolarization of surrounding myocardium occurred after HPS and exhibited retrograde activation sequence (blue lines and arrows). This indicates that the activations from the myocardial breakthrough site failed to invade distal end of LAF, and the isolated conduction within the left HPS remained during ILVT.

Figure 6. Schematic demonstrations of activation sequences of left His-Purkinje system (HPS) and left ventricle (LV) myocardia during idiopathic left ventricle tachycardia (ILVT). During ILVT, depolarization of LPF begins from the earliest retrograde presystolic potentials (black star) and spreads along the fascicle via 2 opposite wavefronts: the proximal LPF was activated retrogradely, and the distal LPF was activated antegradely. But the LAF was depolarized after LPF and exhibited antegrade activation sequence (as indicated by red lines and arrows). Myocardium breakthrough site (hot red) was switched to the distal end of LPF. Similar to the findings during sinus rhythm, depolarization of surrounding myocardium occurred after HPS and exhibited retrograde activation sequence (blue lines and arrows). This indicates that the activations from the myocardial breakthrough site failed to invade distal end of LAF, and the isolated conduction within the left HPS remained during ILVT.

composed of specific fibers with discrete conduction ability, activations could propagate with rapid speed. Therefore, the initial stage of ILVT is always seen with the depolarization of conduction system.

During SR, activation time between the distal ends of LAF and LPF is only a difference of 8.2 ms, and both fascicles contribute relatively equal to the depolarization of ventricle. Although during ILVT, the activation time difference reached 15.2 ms, the LV is predominantly depolarized by LPF alone, and this resulted in a longer activation time of LV. This helps explain why QRS duration is longer during ILVT than that during SR.

Implications of Mapping the Fascicle System

Reproducing left HPS is helpful for distinguishing the PPs of main branches from the near or far field PPs, choosing the ablation target. Meanwhile, by delineating the running course of HPS, the spatial relationship of His bundle with the ablation target is demonstrated; unintentional ablation on proximal HPS could be avoided. Moreover, guided by CARTO system, ablation catheter could be roved toward the septum without persistent exposure to x-ray radiation.

Limitations

CARTO maps could be distorted by catheter tenting and inaccurate collection of positions. Those factors could cause an incorrect description of HPS. However, this was minimized by gentle manipulation of mapping catheter and collecting equally distributed positions by experienced operators.
Conclusions
During SR, isolated conduction within the HPS is demonstrated by documenting the reverse activation sequence with its surrounding myocardium. During ILVT, the earliest retrograde PPs were usually recorded at the middle segment of LPF, and the isolated conduction within the HPS remained.

Disclosures
None.

References

CLINICAL PERSPECTIVE
In vivo data of the left His-Purkinje system (HPS) in human beings is very limited. In the current study, by electroanatomical mapping, the main branches of left HPS and their activation sequence were reconstructed. The results provide additional data about the gross anatomy and the isolated conduction property of HPS during sinus rhythm. Meanwhile, mapping during idiopathic left ventricle tachycardia revealed the earliest retrograde presystolic potentials were usually clustered at the middle segment of left posterior fascicle, and the isolated conduction within the HPS remained. This supports not only the concept of a reentry involving the HPS, but also the earliest retrograde presystolic potential as the crucial point of the reentry. Moreover, by defining the earliest retrograde presystolic potentials and its distribution along the left posterior fascicle, the ablation target could be reliably identified.
Isolated Conduction Within the Left His-Purkinje System During Sinus Rhythm and Idiopathic Left Ventricle Tachycardia: Findings From Mapping the Whole Conduction System

De-Yong Long, Jian-Zeng Dong, Cai-Hua Sang, Chen-Xi Jiang, Ri-Bo Tang, Qian Yan, Rong-Hui Yu, Song-Nan Li, Yan Yao, Man Ning, Tao Lin, Mohamed Salim, Xin Du and Chang-Sheng Ma

Circ Arrhythm Electrophysiol. 2013;6:522-527; originally published online May 14, 2013; doi: 10.1161/CIRCEP.113.000293

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