An unusual cause of HV prolongation

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Introduction
Hyperkalemia is among the most common electrolyte imbalances encountered in clinical practice, affecting up to 8% of hospitalized patients in the United States. 1 The most common causes are renal disease (encountered in 75% of patients with severe hyperkalemia) and administration of medications that elevate serum potassium levels (found in 67% of patients), including drugs often employed in cardiovascular disease such as angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers and aldosterone blocking agents. Often the origin of hyperkalemia is multifactorial and the specific cause is never defined. Hyperkalemia is known to have significant effects on electrical impulse generation and propagation in the heart, with conduction disturbances that may occur at various locations in the specialized conduction system.

Case Presentation
We present the case of a 55-year-old male with a history of diabetes, hypertension, and nonischemic dilated cardiomyopathy, status post–cardiac resynchronization therapy plus defibrillator, who was referred to the electrophysiology laboratory for ventricular tachycardia ablation after experiencing recurrent implantable cardioverter-defibrillator shocks despite multiple prior ablation procedures. The patient’s drug regimen included valsartan 80 mg twice daily, spironolactone 25 mg once daily, sotalol 80 mg twice daily, mexiletine 150 mg twice daily, and bumetanide 1 mg once daily. He had chronic renal insufficiency with a baseline creatinine concentration of 1.7 mg/dL. Biventricular pacing was discontinued for the procedure, and the device was programmed to VVI backup pacing at 40 beats per minute. On catheter placement, a prolonged HV interval was noted (HV 119 ms) in contrast to an HV interval of 75 ms in the previous procedure (7 months prior); the AH interval was 99 ms. PR prolongation (292 ms) and a wide QRS (225 ms) with an atypical right bundle branch block morphology and right superior axis were evident in the surface electrocardiogram (Figures 1 and 2). The serum potassium concentration (K) was noted to be 6.8 mEq/L. After treatment with insulin, glucose and intravenous loop diuretics, potassium levels were normalized (K 4.4 mEq/L), and the HV interval decreased in parallel, to 78 ms, with concomitant reductions in PR and QRS widths (221 ms and 183 ms, respectively) (Figures 1 and 2). Hyperkalemia was attributed to the patient mistakenly discontinuing loop diuretics (instead of mexiletine as instructed) while remaining on valsartan and spironolactone in the days leading up to the procedure. The procedure was resumed immediately after the correction of hyperkalemia, with induction of ventricular tachycardia that was successfully ablated from the endocardium targeting the basal lateral wall of the left ventricle.

Discussion
Although hyperkalemia has long been known to induce conduction disturbances at several levels, 2 few documented cases of acute reversible HV prolongation secondary to hyperkalemia are found in the literature. 3 Extracellular potassium levels play a key role in the normal electrophysiological function of myocytes, as the transmembrane potassium gradient is the main determinant of resting membrane potential. A biphasic response to hyperkalemia has been described 4 whereby mild hyperkalemia (K < 6–7 mEq/L) lowers resting membrane potential (makes it less negative) more so than threshold potential, diminishing the difference between the two and leading to increased excitability and enhanced conduction. 5 Additionally, action potential duration is reduced because of increased potassium conductance during phases 2 and 3 of the action potential. These phenomena may manifest in the surface electrocardiogram as shortening of the PR, QRS, and QT intervals. 4 With increasing extracellular potassium levels (K > 7–8 mEq/L), as resting membrane potential progressively becomes less negative, the percentage of available sodium channels is reduced, leading to a decrease in the inward sodium current during phase 0 of the action potential and a reduction in conduction velocity; which is evidenced by lengthening P
waves and prolongation of the PR and QRS intervals. As the specialized conduction tissue is least sensitive to the effects of hyperkalemia; impaired conduction at this level (His bundle) should be expected to appear only at higher extracellular potassium levels.

- Prior to an electrophysiological procedure, attention should be paid to the patient’s drug regimen. An effort should be made to adequately relay instructions on any changes to previous medications required for the procedure, such as discontinuation of antiarrhythmic drugs.

- It is indispensable to carefully review the results of the patient’s blood work, including basic blood chemistry as well as complete blood count, before the initiation of an electrophysiological procedure.

- Hyperkalemia can induce reversible infra-Hisian conduction delay. In patients with underlying conduction disease, HV prolongation may actually occur with lower potassium levels.

In our patient, infra-Hisian conduction disease was already present at baseline (HV 75 ms 6 months prior, HV 78 ms after correction of hyperkalemia) and may have been partly influenced by the long-term use of mexiletine (which the patient had already been taking at the time of the previous procedure). Nonetheless, the decrease in the HV interval from 119 to 78 ms on normalization of potassium levels suggest additional infra-Hisian conduction delay attributable to hyperkalemia. Additionally, the patient’s AH interval under hyperkalemia also appears to be longer than under normokalemia (99 ms vs 69 ms). The AH interval duration is characterized by marked variability due to variations in autonomic state. In this particular case, the first recordings (left in Figure 2, K 6.8 mEq/L, AH 99 ms) were obtained under deeper sedation, with the later recordings (right in Figure 2, K 4.4 mEq/L, AH 69 ms) obtained under more-superficial sedation. These variations in the depth of sedation likely account for any variations in AH interval.

Conclusion
We describe a case of reversible HV prolongation secondary to moderate hyperkalemia in a patient with underlying infra-Hisian conduction disease. In patients with diseased con-

![Figure 1](image-url)

**Figure 1** Surface electrocardiogram with serum potassium levels at 6.8 mEq/L (top) and at 4.4 mEq/L (bottom). Note the prolongation of the PR (292 ms) and QRS (225 ms) intervals during hyperkalemia compared with normokalemia (PR 221 ms, QRS 183 ms). QRS in both cases has an atypical right bundle branch block morphology with a right superior axis.
duction tissue, further decreases in impulse conduction may become apparent at potassium levels not normally associated with HV prolongation.

**References**

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![Figure 2](image-url) Intracardiac tracings with serum potassium levels (K) at 6.8 mEq/L (left) and 4.4 mEq/L (right). From top to bottom: surface electrocardiogram leads, His bundle recordings. Note the prolongation of the HV interval (119 ms), which decreases (78 ms) on correction of hyperkalemia. His bundle electrocardiogram is singled out with a red arrow: QRS onset (marked by the vertical red line) is earliest in V2.