

Calvin J Tan, BS

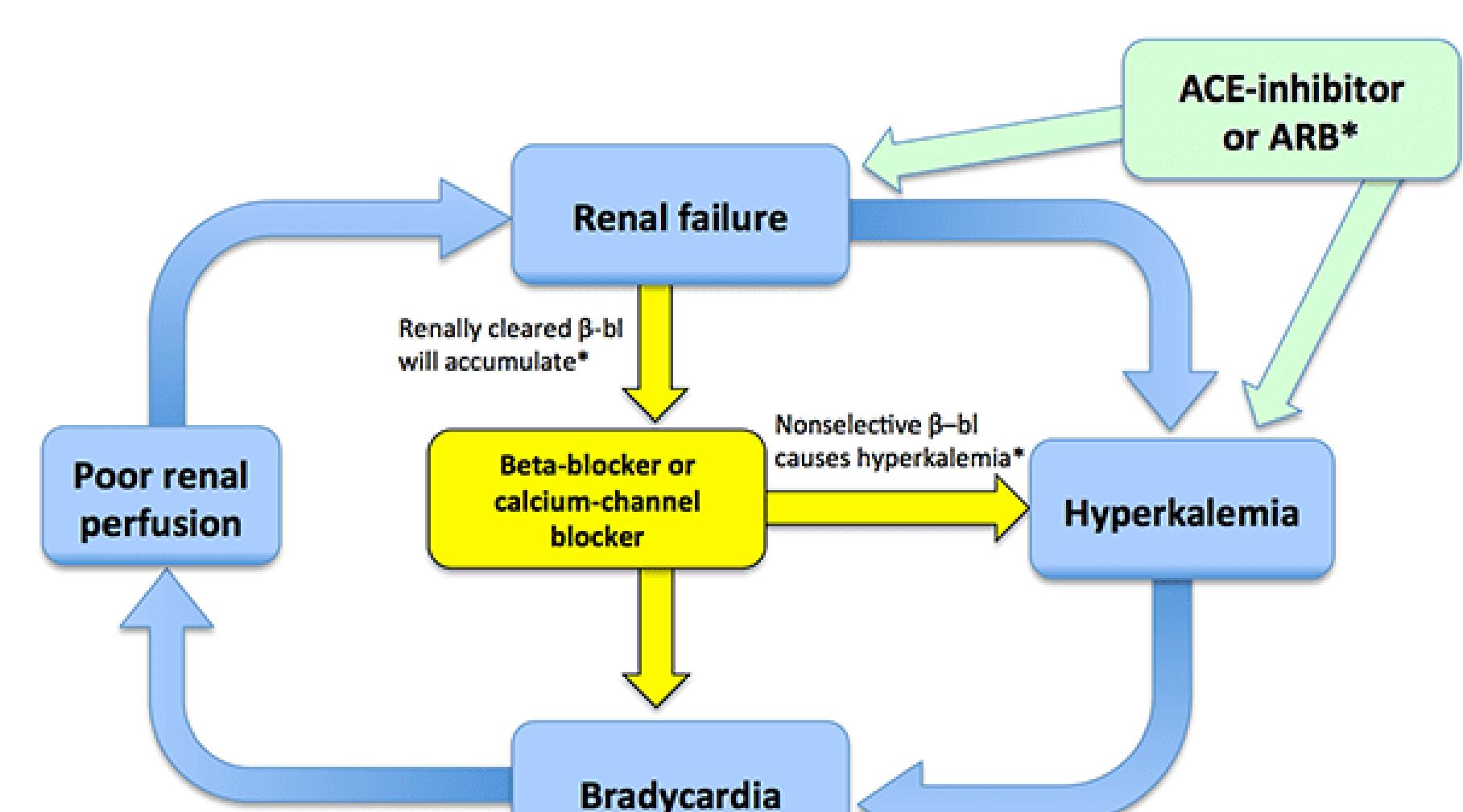
State University of New York – Downstate, College of Medicine; Kings County Hospital Center

## Introduction

BRASH syndrome (bradycardia, renal failure, AV node block, shock, hyperkalemia) is a relatively newly described clinical entity. The term 'BRASH syndrome' appears to originate from a 2016 post by Dr. Josh Farkas on the *EMCrit* blog where he described the pathophysiologic cycle that produces this particular bradycardic state.<sup>1</sup> Patients with BRASH syndrome present with profound bradycardia and AV block that is not proportional to their rate controlling medication use or their (often mild) hyperkalemia alone. Furthermore, these patients' symptoms may be refractory to typical positive chronotropic interventions or overdose antidotes.

## Case Description

An 89-year old woman with history of hypertension, diabetes mellitus, congestive heart failure, chronic kidney disease stage IV, and atrial fibrillation was brought in by EMS for decreased responsiveness and bradycardia (HR 17 bpm). The patient was on multiple medications including Metoprolol, Coumadin, and Furosemide. She was given 2 doses of Atropine by EMS en route and 1 dose during resuscitation without significant effect on the bradycardia; glucagon also failed to correct the heart rate. An external pacer was applied with capture at 72mA and rate set at 70. Initial testing revealed third-degree heart block on EKG, multiple metabolic and electrolyte abnormalities notably Cr 4.38, glucose of 521 and, potassium of 6.6 without characteristic hyperkalemic EKG changes – no QRS widening or peaked T waves – and an eGFR of 9. Before consulting for transvenous cardiac pacing, the patient received a trial of calcium gluconate which increased her heart rate to 105. She was weaned off the external pacer, admitted to the MICU for dialysis, and was subsequently discharged 2 weeks later.



\* Indicates components which are not required, but may contribute in some cases  
Image from *EMCrit* article on BRASH Syndrome by Josh Farkas, MD<sup>1</sup>

## Initial EKG



Ventricular rate: 32 bpm, Atrial rate: 234 bpm, QTC: 440 ms

## Discussion

In this patient, the initial cause of bradycardia is likely multifactorial caused by a mix of cardiac disease, kidney disease, and beta blocking medication. Reduced plasma volume and dehydration are thought to be environmental causes of hyperkalemia that have been suggested as provoking factors in BRASH syndrome.<sup>2-4</sup> In the proposed cycle of BRASH syndrome, a patient with cardiac, renal, and pharmaceutical risk factors falls into a low volume state and is unable to compensate with an increase in heart rate due to pharmaceutical AV-blockade. The resulting bradycardic cardiogenic shock and subsequent decreased kidney perfusion in a patient who already has poor renal function worsens the hyperkalemia. The developing hyperkalemia synergizes with AV-blocking agents causing increasing nodal blockade and further bradycardia even at relatively low potassium levels.<sup>5,6</sup> It is important to note that even with the involvement of cardiac, renal, and pharmacologic causes, the patient's symptoms are caused by a correctable electrolyte derangement.<sup>4</sup> In these patients, following the American Heart Association ACLS bradycardia algorithm (Atropine, Dopamine, Epinephrine, or transvenous pacing) will not correct the underlying cause of the bradycardia, nor will the beta-blocker antidote glucagon. Instead, fixing the fluid status and treating the electrolyte abnormality can lead to dramatic improvement in the patient.

## Conclusion

For cases of profound, initial treatment-refractory bradycardia in patients on rate controlling medication with hyperkalemia, consider BRASH syndrome with treatment centering on correction of the electrolyte abnormality even in the absence of characteristic hyperkalemic EKG findings.

## References

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## Acknowledgements

Dr. Lorenzo Paladino  
Dr. Richard Sinert