Introduction: Atrial standstill (AS) is characterized by the loss of electric and mechanical activity. Moreover, AS has been reported in families with autosomal dominant Brugada syndrome with the alpha subunit of the cardiac Na+ channel (SCN5A) mutations. We present a case that three-dimensional (3D) mapping system showed partial left atrial AS and recovery of the electric activity using isoproterenol infusion.

Methods: A 41-year-old female was referred to our cardiology department with paroxysmal palpitation and short of breathness. She denied any syncope, dizziness, and chest pain. As a past medical history, she experienced cardiogenic stroke due to paroxysmal atrial fibrillation (AF), revealed by insertable cardiac monitoring system. She took apixaban 10mg/day. Her father had advanced atrioventricular block, underwent pacemaker implantation and her son was diagnosed as Brugada syndrome. They also had SCN5A mutations.

Result: An electrocardiogram showed atrial bigeminy at a rate of 67 beat per minutes and was otherwise normal. A chest X-ray showed cardiomegaly without pulmonary congestion. The echocardiogram revealed normal ventricular systolic function and no left atrial dilation. She underwent catheter ablation for AF using the Abbott Ensite Precision Cardiac Mapping SystemTM with AdvisorTM HD Grid Mapping Catheter, Sensor Enabled. During the procedure, no electrical activity was recorded in the almost whole left atrium, whereas right atrium was normal. After bilateral pulmonary vein isolations, it recovered by isoproterenol infusion to check the dormant conductions and dissipated rapidly with time. Isoproterenol also improved mechanical activity of left atrium and auricular appendage in postoperative transesophageal echocardiography.

Conclusion: This is the first case report about transformation of atrial viability; unique response to isoproterenol infusion in AS, visualized by 3D mapping system. Isoproterenol has been shown to be effective in electrical and mechanical recovery of AS by increasing sodium current due to beta-receptor activation. We provide AS with SCN5A mutations is essentially a matter of sodium channel dysfunction.