**Aim 1: Identify triggered variants of pAF.** *Intracardiac and extracardiac electrical features will classify pAF into triggered versus re-entrant variants.* Population level data will be leveraged to identify clinical subphenotypes of pAF, incorporating ECG and echocardiography markers, arrhythmia burden, recurrence, and time-varying components of potential risk-factors. These subphenotypes will be compared against intracardiac findings, including EGM and EAM data, to validate triggered arrhythmia.

**Aim 2: Determine the role of autonomic mechanisms in triggered pAF.** *Triggered pAF will be associated with increased electrical and neurohormonal biomarkers of vagolysis.*  
Neuropsychological markers of stress will be obtained through clinical interview prior to PVI, and will be clinically phenotyped (**Aim 1**). Coronary sinus levels of NPY, Gal, S100B and arrhythmia thresholds will be obtained before and after PVI, and at the time of any additional physiological testing (catecholamine infusion, vagal nerve stimulation).

**Aim 3: Evaluate the role of cardiovagal receptor genotypes in the risk of vagally-triggered arrhythmias.** *We hypothesize that novel variants exist that may explain the risk of vagally-triggered arrhythmias.* A subset of patients identified with both decreased rest and reactivity cardiovagal outflow and increased CV mortality have been identified. Genetic analysis will be performed to discover novel mechanisms and biomarkers of arrhythmia risk. Identified variants will be validated in a separate cohort to evaluate the arrhythmia risk and electrical phenotypes identified in **Aim 1** and **2**.