artificial intelligence (AI) to a standard 12 lead ECG using a convolutional neural network.

**Objective:** This exemplary case demonstrates the potential use of the algorithm to identify aortic stenosis one decade before clinical detection.

**Methods:** N/a

**Results:** Case Description: An 81-year-old man with a history of hypertension and a long-standing heart murmur presented to the hospital with one month of worsening dyspnea on exertion and a recent syncopal event. He had been previously in satisfactory health.

Physical exam demonstrated significant carotid upstroke delay, loud (3/6) late-peaking systolic ejection murmur at the right upper sternal boarder which extended into the second heart sound with radiation to the carotid arteries. Lungs had rales at the bases bilaterally. An ECG showed sinus tachycardia with a first-degree AV block, left anterior fascicular block, lateral T-wave inversions with lateral ST-depressions, meeting criteria for left ventricular hypertrophy (Figure 1E). Compared to prior ECGs, worsening ST-T-wave abnormalities and left ventricular hypertrophy were noted (Figure 1). Review of prior ECGs with the AI ECG algorithm for aortic stenosis revealed an increased probability of clinically relevant valve disease since 2010 with increasing likelihood of disease each subsequent ECG (Figure 2).

Echocardiography identified critical, calcific aortic valve stenosis with a mean systolic gradient of 78 mmHg and valve area of 0.56 cm² with a left ventricular ejection fraction of 51% (Figure 2F). NT-proBNP was 7086 pg/mL. Moderate coronary disease identified on angiography. The patient underwent transcatheter aortic valve replacement and PCI with good outcome.

**Conclusion:** This case demonstrates the potential of the AI ECG to detect clinically relevant valve disease years before clinical presentation. Though this patient had regular contact with the healthcare system, his historical murmur did not trigger advanced imaging. Though multiple ECGs were obtained over years, all changes were assumed to be benign and minimal (Figure 1). Use of the AI ECG algorithm may have expedited identification of this valve disease nearly a decade prior to his critical presentation (Figure 2).

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**HALF-NORMAL SALINE IRRIGATION AS A METHOD FOR PHRENIC NERVE DISPLACEMENT DURING EPICARDIAL ABLATION OF VENTRICULAR TACHYCARDIA**

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**Background:** Phrenic nerve displacement can be necessary during epicardial catheter ablations. High impedance irrigants can be used to direct radiofrequency (RF) energy toward myocardium.

**Objective:** We present the novel use of half-normal saline (HNS) within the pericardial space to displace the phrenic nerve during ventricular tachycardia (VT) ablation.

**Methods:** N/A

**Results:** A 39 year-old male with non-ischemic cardiomyopathy and idiopathic VT previously underwent an endocardial VT ablation five years ago targeting the left ventricular (LV) anterolateral papillary muscle, and again one year ago targeting a similar VT felt to be from the anterolateral LV. A cardiac MRI showed thinning and akinesis of the basal to mid anterolateral, lateral, and inferolateral segments, with delayed gadolinium enhancement. A mid-myocardial stripe was present. LV ejection fraction was 35%. An implantable cardioverter-defibrillator was implanted, after which he experienced a shock for VT. He was started on sotalol and referred to our institution for repeat ablation. Under general anesthesia, anterior epicardial access and vascular access were obtained. An endocardial LV voltage map showed no scar. Epicardial voltage mapping showed anterolateral and inferolateral scar. A dense cluster of late potentials (LPs) was present within a deceleration zone identified by isochronal late activation mapping. VT was induced, and the cluster of LPs became mid-diastolic. Attempts to entrap were unsuccessful. Substrate homogenization was performed. However, additional LPs persisted directly under the course of the phrenic nerve identified by pace mapping, and VT remained inducible. HNS was infused to create a pericardial effusion and displace the phrenic nerve; high output pacing was performed until loss of phrenic nerve capture, at which point ablation was performed to abolish the remaining LPs. The pericardial effusion was drained and phrenic capture re-confirmed. VT was thereafter non-inducible on isoproterenol, and the patient has remained arrhythmia-free.

**Conclusion:** Intrapericardial fluid infusion can be used to displace the phrenic nerve. Low ionic irrigants can preferentially direct RF to myocardium. To our knowledge, this is the first reported case of HNS use for epicardial phrenic nerve displacement.