Seven Years of Left Ventricle Pacing Due to Mal-positioning Through Patent Foramen Ovale.

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Citation

Abstract
Pacing in the right ventricular apex is typically seen on the surface ECG as a Left Bundle Branch Block (LBBB) pattern, hence presence of a Right Bundle Branch Block (RBBB) pattern should raise a suspicion for atypical conduction pattern or lead mal-position. Unintentional mal-positioning of the pacemaker lead in different locations, including left ventricle has been reported. This may be due to unintentional inter-atrial septal puncture or through a congenital defect such as patent foramen ovale (PFO), atrial or ventricular septal defects. Cannulation of the sub-clavian artery is another possibility. The exact incidence of a lead mal-position is not known but is rare. Patients may remain asymptomatic with normal lead function but are at an increased risk for thromboembolic events. We report a case of lead mal-positioning in the left ventricle through a PFO which was incidentally discovered years later.

CASE REPORT
An 89 year old female with past medical history significant for hypertension, gout, obesity, chronic kidney disease stage 5, coronary artery disease, status post permanent pacemaker implantation, was admitted for worsening lower extremity edema, orthopnea and easy fatiguability without overt paroxysmal nocturnal dyspnea. The symptoms had been going on for a few weeks. Her medications included two diuretics - furosemide and metolazone, ACE inhibitor, Aspirin, carvedilol and allopurinol. The pacemaker was implanted at an outside facility seven years prior to this presentation secondary to a diagnosis of sick sinus syndrome. She was followed on an outpatient basis. The pacing thresholds, lead impedance and sensing were within normal limits.

Initial EKG on presentation showed a RBBB pattern (Fig. 1). Chest radiography demonstrated a posterior displacement of the ventricular lead, suggestive of placement in the LV rather than the RV (Fig. 2a and 2b). A transthoracic echocardiogram (TTE) demonstrated an ejection fraction of 55-60% with impaired LV relaxation and reversal of mitral E-A wave ratio. There was mild to moderate tricuspid regurgitation with a right ventricular systolic pressure of 51 mm Hg with an estimated RA pressure of 10 mm Hg. The ventricular pacing lead was noted to cross the inter-atrial septum with implantation into the left ventricle (Fig. 3). A CT scan of the chest also showed the ventricular pacing lead within the LV (Fig. 4). The patient had had no documented thromboembolic events.

Cardiology consultation recommended consideration of long term anti-coagulation with warfarin as these patients are considered high risk of having a thromboembolic event. After consideration the patient declined long term anticoagulation. She felt that due to her advanced age and lack of any symptoms, the added risk of anticoagulation was too high. The patient was treated for her congestive heart failure and discharged home after an uneventful hospitalization.

Figure 1
Fig. 1 – RBBB pattern EKG
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**Figure 2**
Fig. 2(a) PA view of chest X Ray

**Figure 3**
Fig. 2(b) Lateral view of CXR - Arrow pointing to the posterior displacement of the pacemaker wire.

**Figure 4**
Fig. 3 – Four chamber view of ECHO showing patent foramen ovale and the pacer wire passing through it into the left atrium and left ventricle.

**Figure 5**
Fig. 4 – CT Chest – Arrow pointing towards the diffraction from the pacemaker wire in the left ventricle.

**DISCUSSION**
A ventricular pacing lead can be unintentionally malpositioned into the left ventricle through 1) perforation of inter-atrial or inter-ventricular septum, 2) patent foramen ovale, sinus venosus defect, primum or secundum ASD, 3) puncture of subclavian artery instead of subclavian vein, 4)
through a VSD or 5) through intentional or unintentional pacing of a branch of the coronary sinus. The incidence is not well-known. The most feared complication is a cerebrovascular event because of foreign body in the systemic circulation. Patients may remain asymptomatic and the malposition is often discovered incidentally as in our patient.

The expected findings on surface EKG of right ventricular pacing is a LBBB pattern. This is due to the earlier stimulation of the right ventricular muscle however a RBBB can be seen (as per Okmen et al- (3) in 8.3% patients out of a cohort of 300 consecutive patients which fell to 4.3% after modifying the EKG locations to one interspace lower than standard positions – (Klein manoeuvre). Explanation for a RBBB include- a) retrograde right to left bundle activation, b) preferential stimulation of parts of the anatomic right septal surface which is behaving electrically as left ventricle, c) right ventricular delay because of severe conduction disease. If RBBB is seen after pacemaker placement immediate evaluation should be undertaken with a lateral view chest X-ray or fluoroscopy. If an X-ray is non-diagnostic, an echocardiogram may be necessary. A CT scan of the chest can also confirm lead positioning. Immediate repositioning of the lead should be carried out if confirmed at the time of or just after the procedure. As in our patient, when the malpositioned lead is noted during long term follow up, anti-coagulation (preferably with warfarin) or lead removal are generally recommended. Antiplatelet therapy and warfarin are considerations. The data with warfarin has far better results in both primary and secondary prevention of thromboembolic events. In regards to thrombus formation, a case has been reported (Bohm et al where two thrombi were found on surgical extraction of the lead which were not detected on TEE- (4).

Surgical extraction should be considered in patients with recurrent embolic events or if cardiac surgery is being considered for other indications. A diagnosis of left ventricular lead malposition requires a high index of suspicion if the surface EKG shows RBBB pattern. It should also be considered in the differentials if a patient with pacemaker or defibrillator presents with an unexplained neurovascular event.

References
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