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# Electrode ingrowths revealed during cadaveric dissection of a 5y old implanted pacemaker — the case report

MARCIN KUNIEWICZ<sup>1,2</sup>, JERZY ANDRZEJ WALOCHA<sup>1</sup>, KINGA BUDNICKA<sup>1</sup>,  
MAŁGORZATA MAZUR<sup>1</sup>, EWA WALOCHA<sup>3</sup>, MICHAŁ JURCZYK<sup>4</sup>

<sup>1</sup>Anatomy Department, Jagiellonian University Medical College, Kraków, Poland

<sup>2</sup>Department of Electrophysiology, John Paul II Hospital, Institute of Cardiology  
Jagiellonian University Medical College, Krakow, Poland

<sup>3</sup>Department of Clinical Nursing, Institute of Nursing and Obstetrics, Jagiellonian University Medical College  
Kraków, Poland

<sup>4</sup>Department of Pathophysiology, Jagiellonian University Medical College, Kraków, Poland

**Corresponding author:** Marcin Kuniewicz, MD, PhD

Department of Electrophysiology, John Paul II Hospital, Institute of Cardiology  
Jagiellonian University Medical College

ul. Piłsudskiego 80, 31-202 Kraków, Poland

Phone: +48 12 614 23 81; E-mail: marcin.kuniewicz@uj.edu.pl

**Abstract:** Increasing numbers of implanted cardiovascular electronic devices, results in a need for lead extractions, which has increased to an annual volume of over 10,000 worldwide. We present a cadaveric dissection body with a single chamber pacemaker implanted 5y before death.

**Key words:** pacing, TLE, transvenous lead extraction, electrode ingrowths.

## Introduction

There has been an increased amount of cardiovascular implantable electronic devices (CIEDs), resulting in a rise of lead extractions worldwide. Currently, there have been more than 10,000 lead extractions occurring annually [1]. Although these procedures

are typically uneventful, they are associated with a consistent rate of significant procedure-related complications and mortality [2]. The National Cardiovascular Data Registry for transvenous lead extraction reports possible cardiac complications to be around 2.3% [3].

The pacemaker implantation procedure most commonly requires the left cephalic or left subclavian access, through which the electrode is placed in the right ventricle via the superior vena cava, right atrium, and tricuspid valve. The pacemaker can be placed beneath the skin, below the clavicle medially to the anterior axillary line. Reasons and indications for lead extraction were published in Heart Rhythm Society (HRS) guidelines [4].

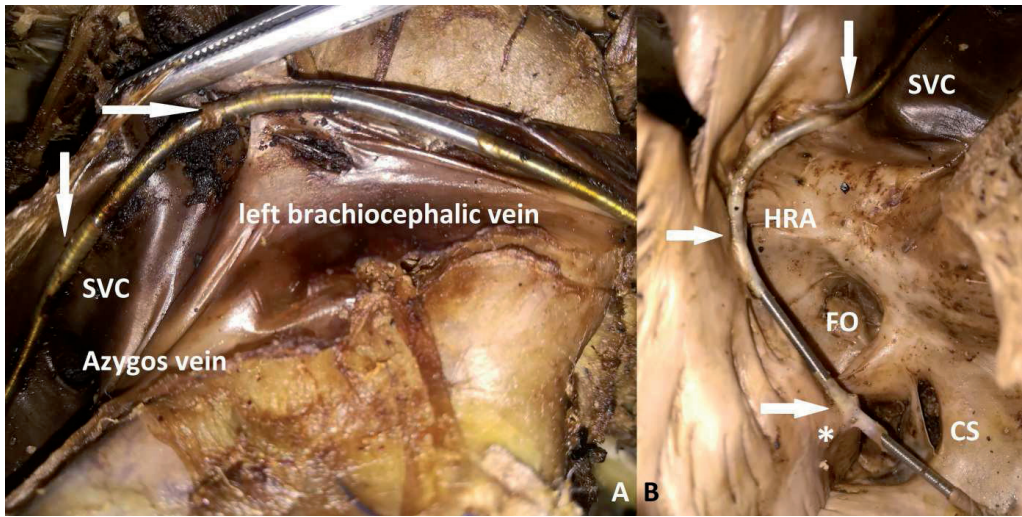
Traction on densely adherent lead or using tools to cut through a dense adhesion may result in vein wall damage. We have not found such a case of a dissected electrode in literature during cadaveric dissection.

### Case report

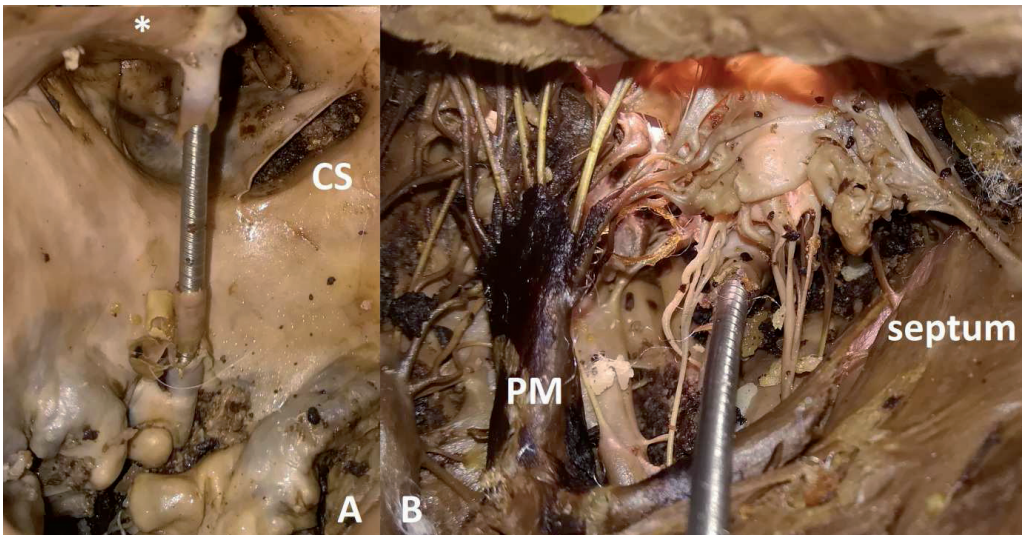
During a routine dissection of a 10% formalin-fixed cadaver of an 87-year-old male, with a well known medical history involving an implanted pacemaker, we uncovered the whole course of an electrode from the pacemaker pocket up to the right ventricle. The single chamber pacemaker Vitatron G20– was inserted because of atrioventricular block t.3 in the course of bradycardia during permanent atrial fibrillation. Venous access was made by cephalic vein dissection in the deltopectoral groove.

Anatomic dissection started with sternotomy. During the early stages of preparation, there were difficulties with the opening of the chest because of a previous cardio surgery operation (coronary artery bypass graft CABG Ao to RCA and Dg2).

After uncovering the electrode, we found epithelial lining throughout the course of the electrode and an ingrowth in the beginning of the superior vena cava (SVC), on the right aspect, close to the left Brachiocephalic vein access (Fig. 1A), in the end of the SVC just before the entrance to the right atrium (Fig. 1B) and in the cavotricuspid isthmus — over Todaro Ligament (Fig. 1B). The length of ingrowth was also impressive — high right atrium — beginning of SVC — 5 cm, the superior aspect of SVC– brachiocephalic connection — 2 cm, ligament of Todaro 1 cm, tricuspid annulus 0.5 cm. After the electrode trespassed the tricuspid annulus, impressive tissue was found layering over the silicone cuff (Fig. 2A, 2B), between the septal and posterior cusp of the tricuspid valve. From the described position, the electrode was directed toward the interventricular septum.



**Fig. 1.** **A** — an uncovered course of the electrode via brachiocephalic vein and superior vena cava (SVC); **B** — entrance through SVC to the right atrium, HRA — high right atrium, FO — foramen ovale, CS — coronary sinus, with white arrows marked places of ingrowths into heart endothelium, \* Todaro tendon.



**Fig. 2.** **A** — view of the electrode from right atrium; \* Todaro ligament, CS — coronary sinus; **B** — view from the right ventricle, PM — papillary muscle.

## Discussion

The ingrowths present within the silicone electrode occur because of so-called tribological phenomena [5], but not as described, lead to lead, but between the lead surface and epithelium. Such findings may be similar to wound healing described by Modarai, who analyzed native resolutions of thromboses in venous systems [6]. The same kind of process was described after the Micra pacemaker autopsy [7]. Biotribology deals with interacting surfaces in motion in biological system. Wear, friction and lubrication influence not only function of lead but also native tissue. Tribology phenomena appears on leads bend in heart cavities which contact with native tissues as well as other leads [8]. Systematic friction in the most curved parts of the electrode with the highest force of contact creates local endothelial damage, inflammation and clot formation; therefore, repair processes were observed. Lead cannot damage blood at the same time suitable lubrication must be preserved what is often impossible in biological system. Tribological phenomena may also appears between elements of lead, inside it. Lead implantation results in endothelial damage what is worsen by continous friction and irritation by the electrode. These processes are more common in heart cavities than in vein because of continuous movement what is confirmed by higher frequency of ingrowths in heart compared to vessels. Lead diameter and route of implantation affect initiation of ingrowths formation. In the same time inflammation also promote fibrosis. Patients with pacemakers have significant higher levels of proinflammatory cytokines. The levels do not result in systemic symptoms but may result in to local inflammatory. Vascular endothelium function is worsen by pacemaker implantation. It is exacerbated by atrial fibrillation and hypertension [9, 10]. There are hypothesis that fibrosis has protective function by lowering risk of thrombosis. Encapsulation also provide the fixation of electrode and prevent from moving. Nethertheless fibrosis caused serious danger during lead extraction or disturbing electrical properties of lead. Fibrousus capsule contain fibrousus tissue, collagen, white blood cells and endothelium, particularly external layer.

The adhesion of the electrode over the tricuspid valve, between the septal and posterior cusps, was observed and described through echocardiography imaging as a form of Lead dependent Tricuspid dysfunction (LDTD) [11, 12]. Ingrowths may cause regurgitation as well as stenosis. This lead impingement on the leaflets leads to abnormal coaptation of the tricuspid valve with progressive leaflet dysfunction, functional tricuspid regurgitation, and finally, right ventricle asynchrony [13, 14]. After years almost 80% of canine intraventricular leads results in valular impairment cause by fibrosis ingrowths.

Because it was 5 years after implantation, the size and depth of the ingrowth were significant. Simple traction cannot be removed after long-term electrode implantation,

due to possible complications involving venous laceration, tricuspid valve damage or cardiac tamponade. Fibrosis covering may enlarge enough to cause vena cava superior stenosis but also vena cava inferior stenosis.

### Clinical implication

Transvenous lead extraction has various complications. It may cause cardiac tamponade, vascular injury or tricuspid valve regurgitation. Vascular damage is mainly caused because of traction on densely adherent lead or using tools to cut through a dense adhesion.

The anatomical section of the patient with the implantable device has a tremendous cognitive value for the risk of transvenous electrode removal procedures.

These pictures may emphasize the significance for better planning and should be used to influence operators to perform in a much safer manner during lead extraction procedures.

### Conflict of interest

None declared.

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