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Pacemaker-induced cardiomyopathy in the sheep: RVA but not RVOT pacing results in a heart failure cellular phenotype.

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Abstract

Chronic RV apical pacing can have adverse effects on LV function and up to 10% of patients develop Pacemaker-induced Cardiomyopathy. The pathophysiology of this is incompletely understood, although previous work has shown that altered ventricular activation patterns can cause abnormal calcium handling and apoptosis. The aim of this work was to determine whether physiological-rate RV apical pacing could cause a cellular heart failure phenotype and if this could be prevented by pacing from the RV outflow tract (RVOT).

Experiments were performed in adult female Welsh Mountain sheep, in accordance with national regulations and local ethical review. Under general anaesthetic and fluoroscopic screening, transvenous pacing leads (Medtronic Novus 4076) were implanted via the right internal jugular vein and attached to a generator positioned in a cervical pocket. After 1 week to recover from surgery, pacing was commenced according to the experimental model.

Background

Right ventricular apical (RVA) pacing can be detrimental to cardiac health. Although most apparent with pre-existing heart failure, chronic pacing can also cause heart failure in patients with previously normal ventricular function.

Pacemaker-Induced Cardiomyopathy (PiCM) affects up to 10% of patients with high RV pacing burdens within 1 year of implant. This causes deterioration of left ventricular (LV) function and may be associated with symptoms of heart failure. These changes are largely irreversible by cardiac resynchronisation therapy.

Methods

RV pacing Model

Leads were positioned in the right atrial appendage and either RV apex or RVOT. These were connected to a Medtronic Sensia dual chamber pacemaker.

Heart Failure Model

A single lead was positioned at the RV apex. This was connected to a Medtronic Consulta implantable defibrillator.

Fluoroscopic RAO projections demonstrating RVA (left) and RVOT (right) lead positions. Digital orientation was confirmed using LAO projections.

Results

3 months of physiological rate RV apical pacing resulted in a heart failure cellular phenotype, characterized by calcium transient abnormalities and T-tubule disruption. These features were not observed with RVOT pacing.

These findings occurred before clinical or echocardiographic features of heart failure and may therefore represent the initial stages of Pacemaker-induced Cardiomyopathy.

Conclusions

1. Yu et al. (2009), NEJM 361:2123-34

References

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