

# Atrial standstill during escape nodal rhythm with no retrograde conduction – a new possible risk factor for ischemic stroke?

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The authors have nothing to disclose.

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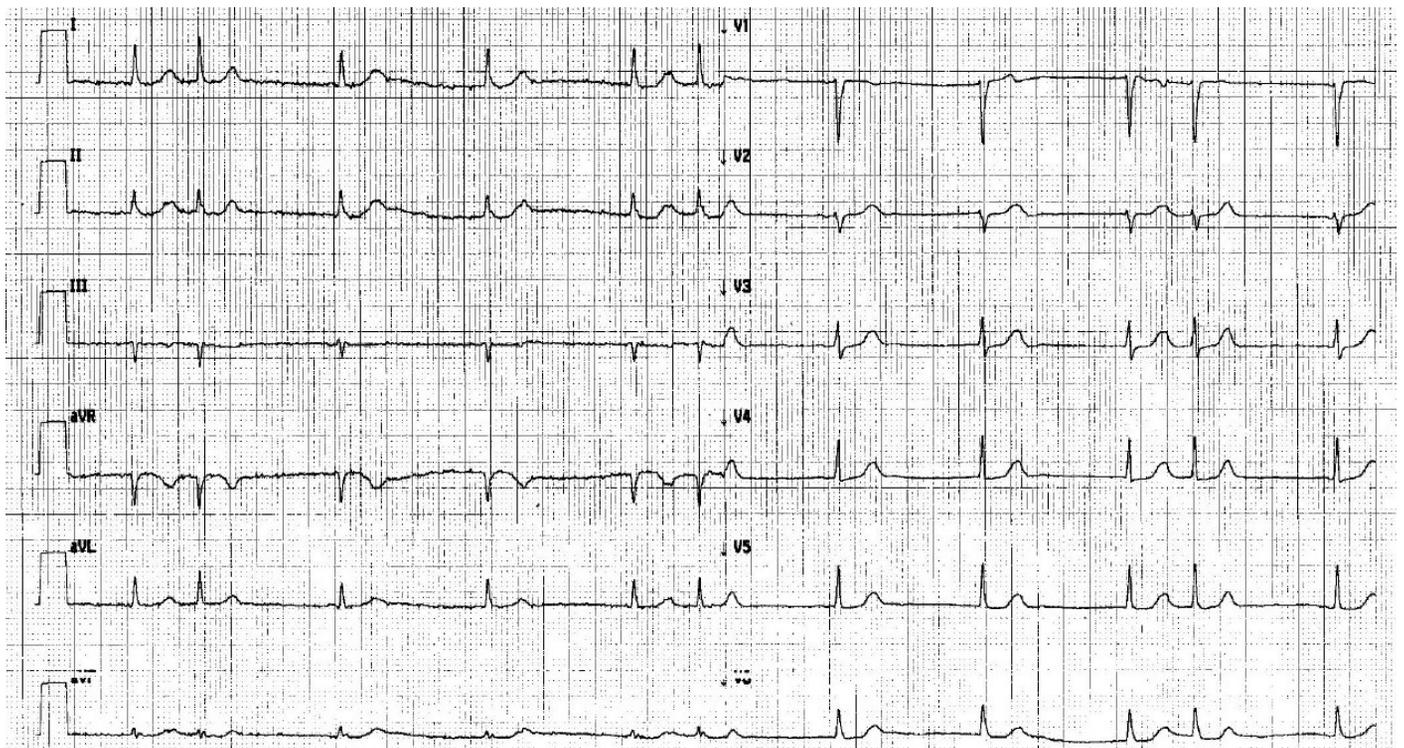
## Abstract

Up to 30% of ischemic strokes are called “cryptogenic”. We would like to present a case of ischemic brain embolism possibly due to mechanical asystole of the atria caused by escape nodal rhythm without retrograde conduction through the atrioventricular (AV) node. Ischemic stroke is a devastating condition of variable etiology. In spite of progress in the diagnostic work-up of stroke, up to 30% of strokes are called “cryptogenic,” which means that no causative factor can be

found. We would like to present a case of possible ischemic brain embolism due to mechanical asystole<sup>[1]</sup> of the atria caused by escape nodal rhythm without retrograde conduction through the AV node.

A 78-year-old woman was admitted to the cardiac intensive care unit due to fatigue and dyspnea. The ECG examination revealed escape nodal rhythm of 45 per minute without retrograde conduction and with sporadic (10/min) atrial extrasystoles (Figure1).

**Figure 1.** Nodal escape rhythm without retrograde conduction with occasional atrial extrasystole.



Blood pressure was 95/50 mmHg. The patient had a history of coronary artery disease, coronary artery bypass graft in the

year 2005 and subsequent percutaneous coronary intervention with drug-eluting stent implantation in the left main coronary

artery in 2010, hypertension and hyperlipidemia. Twelve hours after admission normal sinus rhythm spontaneously returned and the patient was transferred to the general cardiology ward. The X-ray and transthoracic echocardiography were normal. Two hours later the patient developed neurological symptoms – aphasia and partial paraplegia of the right upper limb. Acute stroke was diagnosed, and the patient was sent to a stroke unit in another hospital for thrombolysis. However, a few hours later all neurological symptoms subsided and brain computed tomography showed no abnormalities. Ultrasonography of the carotid artery revealed only one plaque in the right internal carotid artery (ICA) that narrowed its lumen to 60%. Transesophageal echocardiography ruled out the presence of patent foramen ovale or atrial septal defect as a possible cause of stroke. Left atrial appendage function was normal, with flow velocity of 0.84 m/s and without signs of thrombi or echogenic blood. The invasive electrophysiological study showed no retrograde conduction via the AV node.

We speculate that one of the possible causes of ischemic stroke in our patient could be lack of left atrial contraction during escape nodal rhythm in a patient with no retrograde

AV nodal conduction. The non-contracting atrium is a reservoir for thrombus and may be a source of embolic material which can be released upon resumption of sinus rhythm and return of atrial muscle contractility<sup>[2]</sup>. It may be speculated that permanent atrial pacing may be effective for preventing atrial asystole and thrombus formation. However, this has to be documented in a prospective study. Authors' contributions: JB – design, interpretation, drafting article; RP, BZ – data analysis, drafting article; PK – design, interpretation, drafting, revising and approval of article

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