

Quiz

A 70-year old woman presented to the emergency department with fatigue and presyncopal episodes commencing two days ago. The ECG recording revealed 2:1 atrioventricular block with periods of 3:2 Wenckebach conduction. The intracardiac electrograms during spontaneous sinus rhythm and atrial pacing are demonstrated below.

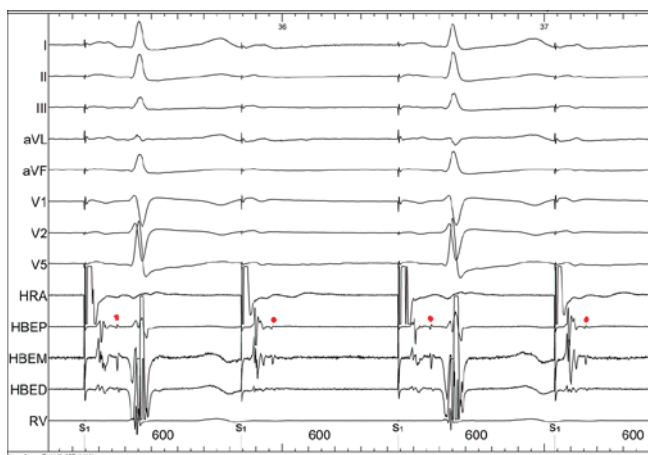


Figure 1. Atrial pacing. His bundle recording is marked with a red dot.



Figure 2. Recording during sinus rhythm. His bundle recording is marked with a red dot.

What is your diagnosis?

Answer to the quiz

Usually, Wenckebach conduction implies that the site of the disturbance is above the His bundle, namely in the atrioventricular node. In most circumstances this kind of conduction abnormality does not mandate the implantation of a permanent pacemaker. However in this specific case, we can see that during atrial pacing which reproduces 2:1 block, the conduction is halted below the

His bundle as the recording of both A (atrial) and H (His) electrograms suggest (figure 1). During sinus rhythm, Wenckebach pattern is apparent and the second beat is conducted with aberrancy and a prolonged HV interval (figure 2). The third atrial beat is blocked below His bundle. In this example we have an infra-Hisian Wenckebach conduction pattern which obliges the need of a permanent pacemaker.

Cardiology News /Recent Literature Review

Antonis S. Manolis, MD, Ector Anninos, MD, Spyridon Koulouris, MD

The ACC Annual Congress is slated for April 3-5, 2011 in New Orleans

The HRS Annual Meeting is scheduled for May 4-7, 2011 in San Francisco

Euro-PCR will take place in Paris on May 17-20, 2011

The ESC Annual Congress is slated for 27-31/8/2011 in Paris

The TCT Annual Conference will be held in San Francisco 7-11/11/2011

The AHA Annual Scientific Sessions are scheduled for 12-16/11/2011 in Orlando

Athens Cardiology Update 2012 is slated for April 5-7, 2012

Local Atrial Inflammation Present in Paroxysmal AF

Atrial biopsies were obtained from 70 patients (age 60 ± 12 years, 49 males) undergoing radiofrequency catheter ablation for AF and 10 patients with Wolff-Parkinson-White syndrome, all undergoing trans-septal puncture. Biopsies were obtained by washing the dilator and needle used for trans-septal puncture with 20 mL sterile phosphate-buffered saline, and formalin fixed specimen were examined by immunohistochemistry for the presence of intracytoplasmic C-reactive protein. C-reactive protein was revealed in isolated atrial cardiomyocytes in 11 (73%) of 15 patients with paroxysmal AF as compared with 2 (25%) of 8 patients with persistent AF ($P=0.02$). This technique for obtaining biopsy specimen was safe and feasible. In this study, local atrial inflammation as assessed by C-reactive protein present in atrial myocytes, is more likely involved in paroxysmal rather than in persistent AF (Narducci ML et al, *Europace*. 2011 Mar 29. [Epub ahead of print])

Are all statins the same? Focus on the efficacy and tolerability of pitavastatin

Pitavastatin is the latest addition to the statin family which undergoes minimal metabolism by cytochrome P450 (CYP) enzymes and, thus, has a low propensity for interactions with drugs metabolized by CYP enzymes. It displays potent and consistent beneficial effects on lipids and has a favourable side effect profile with increases in plasma CK levels in <5% of patients and the incidence of myopathy or rhabdomyolysis being extremely low. Interestingly, in addition to its lowering effect on total and LDL cholesterol and triglycerides (21-32%, 30-45%, and 10-30%, respectively), it also exhibits some potential for increasing HDL cholesterol (mean increase 3-10%). In comparison to atorvastatin, pitavastatin showed similar efficacy in lipid-lowering ability with a trend towards HDL increase. Moreover, it shows beneficial effects regarding the cardiovascular risk independently of its effect on lipid levels. In the JAPAN-ACS study, it was non-inferior to atorvastatin at reducing plaque volume in patients with ACS undergoing PCI. It also exerts pleiotropic effects as revealed by improvements in markers of inflammation, oxidative stress, and renal function that have been demonstrated in a number of small studies (da Silva PM. *Am J Cardiovasc Drugs* 2011;11:93-107).

Radiofrequency catheter ablation of ventricular tachycardia in arrhythmogenic right ventricular dysplasia using non-contact electroanatomical mapping: single-center experience with follow-up up to median of 30 months

The efficacy of radiofrequency ablation (RFA) of ventricular tachycardia (VT) was evaluated in 15 patients (44 ± 15 years) with arrhythmogenic right ventricular dysplasia (ARVD), using non-contact electro-anatomic mapping. Twenty-two of the 25 inducible VTs mapped, were successfully ablated. In 13 out of 15 patients, all the clinical and inducible VTs were ablated. All patients remained asymptomatic at 25 ± 16 months (2-52 months) although antiarrhythmic medications were discontinued after 6 months. Two patients had recurrence of non-clinical VT on follow-up. In conclusion, a majority of induced VT in patients with ARVD can be successfully mapped and ablated using the non-contact Ensite Array Mapping system with good long-term VT-free outcome. Ablation can be a useful adjunct to AICD implantation in such patients (Nair M et al, *J Interv Card Electrophysiol*. 2011 Mar 25. [Epub ahead of print])

Intracoronary levosimendan prevents myocardial ischemic damages and activates survival signaling

through ATP-sensitive potassium channel and nitric oxide

The cardiac effects of different doses of intracoronary levosimendan on ischemia/reperfusion injuries, and the involvement of K(ATP) channels and nitric oxide (NO) were examined in 56 pigs. In 21 pigs, 1.5, 5 & 12 µg/min levosimendan was infused over 15 min into the coronary artery at the onset of 1 h reperfusion following 2-h ischemia and the effects on cardiac function, infarcted area, and on apoptosis/autophagy were examined. The activation of Akt and extracellular receptor kinase (ERK) was also analyzed. The findings were compared with those obtained in 14 pigs where the highest dose levosimendan was infused after glibenclamide and l-nitro-arginine methyl ester (l-NAME). Intracoronary levosimendan caused a dose dependent increase of segmental shortening, dP/dt(max) and cardiac output from values measured at the end of ischemia. Notably, these beneficial effects were still evident albeit somewhat attenuated at the end of reperfusion. When doses of 5 and 12 µg min⁻¹ levosimendan were used, a reduction of infarcted area to about 69% and 67% of area at risk was observed, and was significantly different from that of about 79% measured in control animals. In addition, the inhibition of apoptosis and activation of autophagy and a dose-related increase of the level of phosphorylation of ERK and Akt were observed. These responses were completely prevented by glibenclamide and significantly reduced by l-NAME. In conclusion intracoronary levosimendan seems to reduce cell death induced by ischemia/reperfusion in a dose-dependent manner and activates survival signaling through K(ATP) channel opening and NO, in an animal model of cardiac ischemia/reperfusion (Caimmi PP et al. *Eur J Cardiothorac Surg*. 2011 ;39:e59-67. Epub 2011 Jan 17)

Genetic mechanisms mediating atherosclerosis susceptibility at the chromosome 9p21 locus

Recent genome-wide association studies have demonstrated that common genetic variants in a region of chromosome 9p21 confer risk of coronary artery disease (CAD) and other atherosclerotic conditions. The absolute increase in risk is small (some 20-30% increase in risk of CAD per copy of the deleterious alleles), but the common occurrence of the variants may pose a substantial risk in a general population scale. So far, no association between risk variants and both "classical" and "emerging" atherosclerotic risk factors has been established, indicating that the effect of the 9p21 locus on atherosclerotic risk is mediated via a hitherto unknown pathway which could represent a possible therapeutic target (Cunnington MS, Keavney B. *Curr Atheroscler Rep* 2011;13:193-201)