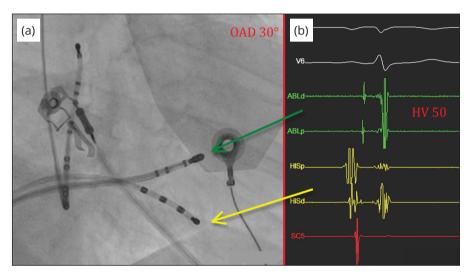
# What is the Diagnosis?

### **CASE PRESENTATION**

The image and tracings were obtained during the para-Hisian stimulation maneuver in an electrophysiological study. The question is: is there any retrograde nodal or extranodal conduction behavior?



**Figure 1.** (a) Radiological image of catheter placement during the procedure; (b) Electrograms corresponding to the catheters identified on the left.

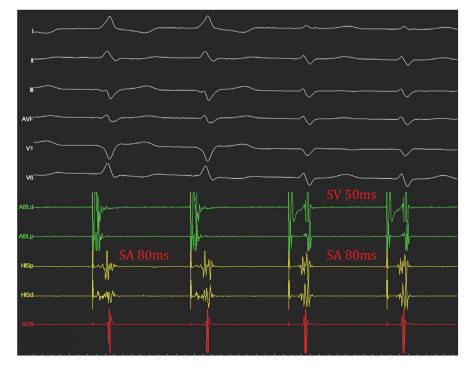


Figure 2. Tracings obtained during catheter stimulation at HIS bundle position.



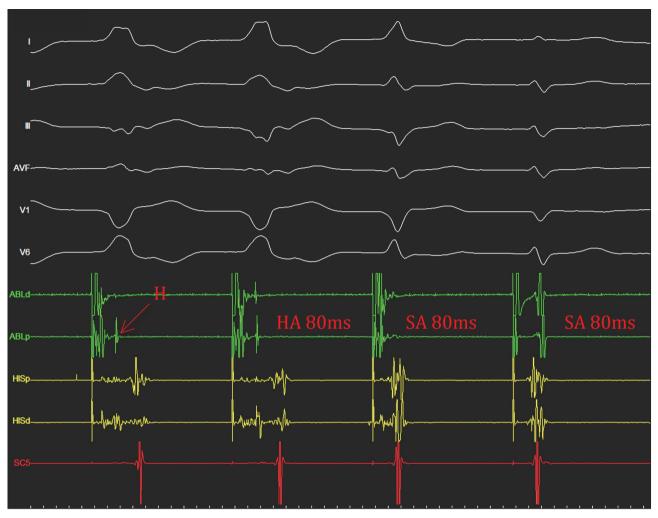


Figura 3. Three electrocardiographic patterns captured during catheter stimulation at the HIS bundle position

#### **ANSWER**

Figure 2 shows the para-Hisian stimulation generating two morphologies of QRS complexes. The first two complexes are widened, and the next two are narrow with a morphology similar to sinus complexes and an S-V interval similar to the base HV interval. In both circumstances, there is retrograde ventriculoatrial conduction, with a fixed S-A interval of 80 ms. The persistence of the same S-A interval during a para-Hisian stimulation inducing different patterns of QRS complexes, either narrow or wide, at first leads to the definition of an extranodal conduction behavior, i.e., retrograde conduction dependent on an accessory pathway. We must keep in mind, however, that the two narrow complexes demonstrated in Fig. 2 were generated by a selective His bundle capture, characterizing not a "para-Hisian" stimulation but a Hisian stimulation in fact, as it is well characterized by the observation of a delay between the stimulation artifact and the QRS complex equivalent to the HV interval (50 ms). In other words, there is no fusion complex generated by ventricular activation resulting from the direct His bundle capture added to the capture of the adjacent ventricular myocardium. Therefore, the whole process of retrograde conduction in this circumstance occurs through the standard conduction system. The two initial broader complexes, in turn, appear to be fusion complexes generated by local myocardial capture and ventricular activation induced by the direct His bundle capture; thus, there is direct but nonselective stimulation of the His bundle. In both situations, retrograde conduction occurs through the standard system. This proof is shown in Fig. 3, where there is, in fact, a loss of the direct His bundle capture. It can be noted that the first two complexes are substantially extended, with a longer

S-A interval. A retrograde His potential is now identified, noting that the H-A interval is precisely 80 ms, with the S-A interval being prolonged by the S-H interval. That is, in this circumstance, the His bundle is indirectly activated after ventricular depolarization. Hence, we have a typical retrograde nodal conduction.

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