The Bradycardia-Dependent Paroxysmal AV block (B-DPAVB) occurs if a single P wave is delayed and falls on the phase 4 range of conduction block when the cell is no longer at resting membrane potential and is therefore unable to transmit the action potential. From there on the AV block tends to persist and may result in prolonged asystole. An escape beat distal to the blocking zone is necessary to repolarize the site of block and to reestablish a normal resting potential. The escape beat, only can restore conduction if the P wave that follows the escape complex does not again fall in the phase 4 range of conduction block. Without an escape spontaneous conduction does not resume even when the sinus rate speeds up proving that the apparent B-DPAVB is not related to an increased vagal tone. (Rosenbaum 1973; Lee 2009)

A vagal mechanism is excluded by the fact that not only sinus deceleration but also premature atrial complexes followed by compensatory pauses can trigger the block. Phase 4 block has not been documented within the AV node. The B-DPAVB is associated with narrow QRS because it occur within the proximal or distal portion of His-Purkinje system (Oe 2008). In the present case QRS is broad because pre-existent block (LAFB? rS pattern II?). The absence of junctional escape complexes during the prolonged asystolic periods can be best explained by probable concealed His bundle escapes that arose proximal to the intra-His blocking site. (Coumel 1971; Fish 1990)

During phase 4 AV block, only those escape complexes can conduct to the ventricles which occur distal to the blocking zone. Symptomatic paroxysmal AV block localized to the HisPurkinje system is a high-risk condition that warrants implantation of a permanent pacemaker. (Medina-Ravel 1989)

References
