# The mechanism of bidirectional VT: A simple "ping pong" in the His–Purkinje system

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Both supraventricular or ventricular mechanisms of bidirectional VT (BVT) have been proposed in the past, involving either focal or reentrant mechanisms.(**Levy 1989**) A supraventricular mechanism with alternating LAFB and LPFB(**Rosenbaum 1969**)was largely excluded with the advent of intracardiac recordings, which failed to show a His bundle potential preceding the alternating QRS complexes during BVT. (**Cohen 1973; Morris 1973**) Postulated ventricular mechanisms have included a single focus in the proximal His bundle or bundle branches with alternating left fascicular block, or single or double foci in the distal HPS. In the single focus case, there is no obvious explanation for why fascicular or BBB should alternate during BVT. Usually, concealed retrograde conduction perpetuates block in the fascicle/bundle branch that initially develops conduction block. Even if a conduction gap prevents concealed retrograde conduction from perpetuating block, both fascicles/bundle branches would have to exhibit the same conduction gap phenomena, which seems highly unlikely. In the double foci case, if neither focus is protected by entrance block, the faster focus should overdrive the slower focus, producing monomorphic VT rather than BVT. If both foci are protected by entrance block, then they would have to have identical cycle lengths, phase-shifted by exactly $180^{o}$ to produce a constant cycle length during BVT, which also is improbable. If only one focus is protected by entrance block, then it could induce a second focus to fire at a fixed coupling interval, but the coupling interval would have to be exactly half of the first focus s cycle length to produce a constant cycle length during BVT.

In contrast to these complicated mechanisms, reciprocating bigeminy solves the puzzle of alternating QRS morphology by a simple "ping pong" mechanism in which DAD-induced triggered activity develops at different heart rate thresholds

in different regions of the HPS or ventricles, consistent with known cellular properties of DAD-induced triggered activity.(**Braunwald 1980; Ferrier 1973, Garfinkel 1992**) To produce a constant (i.e. nonalternating) cycle length during BVT requires only that the coupling intervals of the triggered beats be similar at the two sites.

Although Baher et al. modeled the bigeminy to be due to triggered activity from DADs, the same results are predicted for any mechanism inducing ventricular bigeminy (including automaticity or reentry) at more than one location in the ventricles. In addition, there is no strict requirement for the two bigeminal foci to be located in the distal His Purkinje System (HPS) in opposite fascicles or ventricles. **(Baher 2011)** For example, two reciprocating triggered foci located in the same ventricle, ( **Leenhardt 1995**), or at sites in the endocardium and epicardium, could also produce BVT by this mechanism, (**Nam 2005**) although the QRS axis and morphology changes would be different. However, in humans, the most common BVT pattern during digitalis toxicity and CVPT is RBB block with alternating right and left axis deviation, consistent with reciprocating ectopic foci located in the distal left anterior and posterior fascicles of the left bundle. In mouse, due to its smaller heart size, the more common pattern may be foci located on opposite sides of the interventricular septum.

Finally, Baher et al showed that if the increased heart rate during BVT induces a third bigeminal focus in the HPS, the interactions between the three foci can produce irregular activation patterns resulting in polymorphic VT **(Baher 2011)**. (Figure 1) Since the model is deterministic, the irregularity may be due to chaos, which is a common scenario observed with coupled oscillators. **(Hilborn1994)**

As heart rate progressively accelerates, Baher et al. speculate that additional regions develop DADs, making the conversion to VF progressively more likely.

In summary, these authors conjecture that the full spectrum of arrhythmias described electrocardiographically in acquired and familial conditions associated with BVT can be accounted for based on the known properties of DAD-triggered arrhythmias, as follows:

i. **Ventricular bigeminy,** when a single site in the HPS or ventricular myocardium develops a single DAD-triggered beat following each sinus beat;

ii. **BVT**, when a second site develops ventricular bigeminy, and reciprocally activates the first site by the ping-pong mechanism described above;

iii. **Polymorphic VT**, when 3 or more sites concurrently develop bigeminy. Note that these first 3 mechanisms specifically require bigeminy, i.e. that DADs are capable of triggering only a single PVC following each sinus or paced beat, so that the subsequent beat has to arise from a different location (thereby altering the QRS morophology/axis).

iv. **Monomorphic VT**, when bigeminy progresses to repetitive DADs which generate a run of triggered activity, such that the site with most rapid rate of the triggered activity overdrives the other slower sites, producing a monomorphic QRS complex.

v. **Degeneration to VF**, when any of these VT forms results in wavebreak and initiation of reentry, which is then likely to be maintained by a mixture of reentry and DAD-triggered focal activations.

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