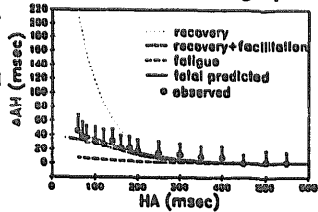


FUNCTIONAL ANALYSIS OF RATE-DEPENDENT PROPERTIES OF AV NODE IN ANESTHETIZED DOGS.

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Three AV nodal (AVN) properties contribute to rate-dependent changes in AV conduction: AVN recovery (Rec), facilitation (Fac) and fatigue (Fat). In order to determine whether these parameters can completely account for rate-dependence we quantified and related each property to steady state AH intervals at different atrial rates in 8 autonomically-blocked dogs. **Results:** AH interval of single atrial impulses was a monoexponential function of preceding HA (Trec, 66±1.9 msec). Premature stimuli (A2) caused a leftward shift of the Rec curve of a subsequent A3 (Fac, max shift=115±27 msec). Fat (independent of Rec or Fac) increased AH during prolonged atrial stimulation by a maximum of 10.9±1.9 msec. Magnitudes of Fac and Fat were exponential functions of AA and HA intervals, respectively. Mathematical modelling was performed to identify the contribution of each process to changes in AH interval over baseline (ΔAH) occurring as a result of increases in cardiac rate (graph).

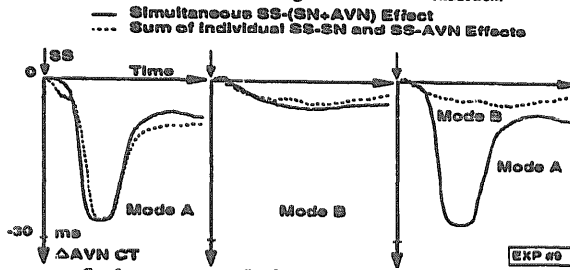
As HA interval decreased at the faster rates, both fatigue and recovery components tended to increase AH interval, while Fac tended to improve conduction. Close agreement between observed values of ΔAH and total predicted changes was observed.



We conclude that Rec, Fac, and Fat are demonstrable in vivo and that each has an important and changing role in determining rate-dependence of the AV node.

MECHANISMS OF SYMPATHETIC CONTROL OF ATRIOVENTRICULAR NODAL CONDUCTION Todor Mazgalev, Ph.D., Leonard S. Dreifus, M.D., F.A.C.C., Rohn Price, B.S., Eric L. Michelson, M.D., F.A.C.C. Lankenau Medical Research Center and Hahnemann University, Philadelphia, PA

Changes in atrioventricular nodal conduction time (ΔAVN CT) after sympathetic stimulation (SS) result from interaction of opposing mechanisms mediated by the release of catecholamines in sinus node (SN) and AVN. This study examined the ΔAVN CT as a sum of the individual effects of SS in SN and AVN. Surface bipolar electrograms were recorded from the crista terminalis, interatrial septum and bundle of His in 12 rabbit heart atrial-AVN superfused preparations. The SN cycle length, intraatrial and AVN CT were measured before (control) and after local postganglionic SS in SN, AVN or simultaneously (SN + AVN) during spontaneous rhythm (mode A) or programmed feedback pacing (mode B) during spontaneous rhythm (mode A) or programmed feedback pacing (mode B) which mimicked SS-induced tachycardia but excluded changes in intraatrial conduction.



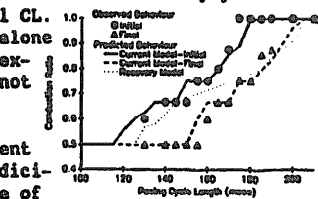
Results: ΔAVN CT after simultaneous SS-(SN + AVN) correlated with the sum of the individual SS-SN and SS-AVN effects in modes A (r=0.98±0.01) and B (r=0.88±0.04) (fig 1,2). However, the sum of the individual effects in mode B did not correlate with the simultaneous SS-(SN + AVN) effect in mode A (r=0.39±0.08; fig 3). Notably, this was due to SS-SN induced shift of the inputs to AVN which existed only in A and caused pronounced shortening of AVN CT. **Conclusions:** 1. ΔAVN CT is a sum of the individual effects of SS-SN and SS-AVN 2. Changes in intraatrial conduction due to sympathetic-induced shift in AVN input activation limit interpretation of the results of programmed pacing in the evaluation of the sympathetic control of AVN.

MECHANISM OF DYNAMIC CHANGES IN WENCKEBACH-TYPE AV BLOCK.

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Recovery (Rec) properties of AV node (AVN) conduction have been used to explain Wenckebach-type AV block, ignoring the potential contributions of other rate-dependent properties [AVN fatigue (Fat) and facilitation (Fac)]. We developed a mathematical model of AVN conduction, which incorporates quantitative indices of Rec, Fac, and Fat, to predict Wenckebach periodicity during atrial pacing at multiple cycle lengths (CL). These predictions were compared to Wenckebach patterns observed experimentally in 5 autonomically-blocked dogs. **Results:** In each experiment, Wenckebach conduction ratio (CR, defined as # of V. impulses/# A. impulses) decreased as pacing was continued at the same rate. Mean CR observed initially and after 5 minutes (final) of pacing (CL 180 msec) were 0.9±0.1 and 0.7±0.1 respectively (p<0.05). Corresponding predicted CR's were 0.9±0.1 and 0.7±0.1, (NS vs observed values). As shown in graph, the shift of CR vs CL to the right with time was accurately predicted by mathematical model at all CL.

Results of model using Rec alone consistently deviated from experimental results and did not account for time-dependent changes in CR.



In conclusion, time-dependent changes in Wenckebach periodicity occur independent of rate of pacing. These changes can be accounted for by considering the interdependence of multiple rate-dependent properties, especially the contribution of AV nodal fatigue.

LONG TERM ELECTROPHYSIOLOGICAL CHANGES AFTER PARTIAL ATRIOVENTRICULAR NODAL ABLATION IN THE DOG

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It would be desirable to produce modification of atrioventricular nodal (AVN) function rather than total ablation to maintain A-V synchrony in patients with refractory AVN reentrant tachycardia. The electrophysiology of AVN modification induced by radiofrequency energy (RFE) or a sham procedure was studied in 9 dogs. The 4 dogs that received RFE had A-H prolongation >70% of control, while the 5 sham treated controls showed no acute or chronic electrophysiological changes. The pacing cycle length was 500 msec.

	TIME AFTER RFE	A-H	AVNFRP*	AVNWP†
RFE	30 min.	217±26 [‡]	125±12	139±11
GROUP	1 week	215±27	93±8	108±16
	2 months	165±5	87±2	104±21
CONTROL	30 min.	100±1	103±4	103±3
GROUP	1 week	100±5	98±2	98±4
	2 months	106±4	102±4	98±7

*Values are % of Baseline ± SEM
*FRP = Functional Refractory Period
†WP = Wenckebach Point

After ablation, the A-H interval remained prolonged but the AVNFRP and AVNWP returned to baseline, indicating a dissociation between AVN conduction and refractoriness. In all the dogs that received RFE there was fibrosis of the approaches to the AV node and the region of the AV node itself. **Conclusion:** Chronic modification of AVN conduction without concomitant changes in refractoriness can be induced by RFE delivered proximally to the AV node.