TITLE: REAL TIME SIMULATION OF CARDIAC ELECTROPHYSIOLOGY

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Monitoring the electrocardiogram (ECG) is a routine, standard practice during anesthesia to determine cardiac rhythm and to assess any electorcardiographic evidence of myocardial ischemia These interpretations may now be automated by advanced ECG monitors which can detect and classify arrhythmias and can display and measure ST segment abnormalities. We developed a simulator of cardiac electophysiology which can produce in real time appropriate rhythms and waveforms of the ECG for use in testing automated rhythm and ST analysis systems or for teaching about ECG interpretation and the physiologic substrates of arrhythmias. The physiologic behavior of electrically distinct areas of the heart is appropriately simulated and the rhythms "emerge" naturally from the interaction of basic cycle times, refractory periods, and topology of the distinct "nodes" and "pathways." The system differs from prior simulations in that: (1) Rhythms are generated in real time[1] and (2) they are generated de novo, not from a library of "examples."

The simulation model (Figure 1) consists of two types of units: Automaticity units (white boxes) which can, if not refractory, trigger themselves with an intrinsic basic cycle time (BCT) or be triggered by impulses from other nodes. Automaticity units are connected to each other via "Conduction units" (grey rectangles) which have no intrinsic triggering, but which contain conduction delays. Conduction units can conduct antegrade and/or retrograde with separate conduction delays and refractory periods. The conduction unit between AV1 and AV2 incorporates "decremental conduction" with a conduction delay dependent on the prior inter-beat interval.Special automaticity units allow for semi-random triggered or "parasystolic" ectopic beats and atrial fibrillation.

The network models only cardiac **rhythm** and outputs a different stylized "spike" for atrial, ventricular, or atrial or ventricular ectopic beats. An augmented version will have the actual ECG waveform generated by the atrial and ventricular automaticity units producing endocardial (ENDO) and epicardial (EPI) action potentials. When all units' components are combined, standard ECG leads can be constructed from the x, y, and z coordinate system. Alteration of parameters in the equations generating the action potentials allows manipulation of the QRS, ST, and T waves[2] (Figure 2).

The rhythm system was implemented as a sequential process in C on a Macintosh IIci, updating every 17 milliseconds (1/60 second).

The system naturally demonstrates capture by distal pacemakers, 1°, 2° (Type I and II), & 3° AV Block (when AV refractory periods and conduction delays change), bundle branch blocks, re-entrant tachycardias, etc. The network is too small to directly demonstrate atrial or ventricular fibrillation. The Atr Fib node will generate appropriate stimuli, but a random number generator must inscribe the firbrillation waveforms. Figure 3 shows example tracings from the rhythm network.

Since rhythms emerge from the interaction of simple units with different electrophysiologic parameters, it may be useful for training concerning the electrophysiology of complex arrhythmias or of anti-arrhythmic drugs.

FIGURE 1

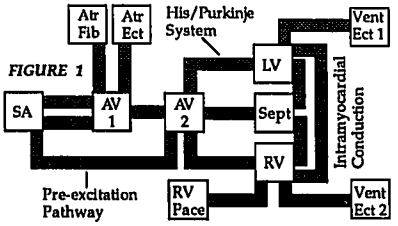


FIGURE 2

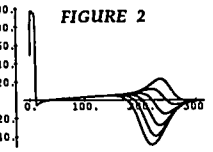
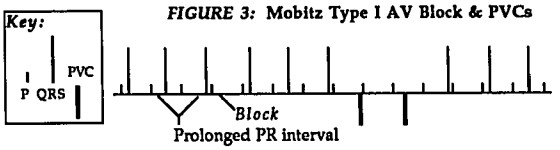


FIGURE 3: **Mobitz Type I AV Block & PVCs**



References: 1. Comp Biomed Res 19:237-253, 1986
2. Eur Heart J 8:409-416, 1987

A1070

Title: INTRALUMINAL GAUGE PRESSURE AFFECTS TRACHEAL TUBE IGNITION BY CO_2 LASER

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As part of a long-term investigation of laser-induced tracheal tube fires and their causes, a physical model was developed to describe the effect of tube intraluminal gauge pressure (IP) on ignition risk. This study measured times to ignition for plain polyvinylcholoride (PVC) tracheal tubes exposed to a CO_2 laser beam.

A 10-W CO_2 laser beam was focused for up to 60 s at normal incidence on tube segments (8 mm ID) in room air through which flowed 40% O_2 with nitrogen at IP of 0.13-4 pascal (Pa) (low) or 196-490 Pa (high). Five trials were conducted at each IP. Low IP was calculated by the formula:

$$IP = (32\mu l \cdot V_i)/(d^2),$$

where μ is gas viscosity, l is the distance from the open end of the tube, V_i is intraluminal gas velocity, and d is the diameter of the tube. For low IP, it was varied by strictly controlling gas flow rate. High IP was obtained with a water column PEEP valve and monitored with a pressure transducer. Total exposure time was measured on a digital timer that was triggered by the laser footswitch.

As IP increased above atmospheric pressure up to 4 Pa, ignition time decreased significantly (Table). However, when IP was increased to ≥ 196 Pa, ignition time increased beyond 60 s in all tubes tested.

These different effects over the two IP ranges are attributed to different controlling factors. At low IP, increasing velocity through a perforation in the tube wall increases O_2 concentration adjacent to the heated region of the tube, which increases risk of ignition. At high IP, gas velocity through the perforation is high enough that convective heat loss is significant. Heat generated in the tube wall is removed and risk of ignition decreases. Under the conditions tested, maintaining an IP pressure of ≥ 196 Pa (2 cm H_2O) significantly decreases the risk of a PVC endotracheal tube fire.

TABLE. Intraluminal Gauge Pressure and Ignition Time (mean ± SD) of Endotracheal Tubes

	Intraluminal Pressure (pascal)	Ignition Time (s)
Low	0.125	14.1 ± 2.0
	0.249	12.3 ± 1.9
	0.374	11.3 ± 2.1
	0.624	9.1 ± 0.5
	0.873	9.6 ± 2.5
	1.372	8.7 ± 1.1
	1.622	6.5 ± 1.8
	1.871	8.4 ± 1.1
	2.495	6.1 ± 0.8
	3.119	6.8 ± 1.6
	3.742	5.5 ± 1.3
High	196.00 (2 cm H_2O)	no ignition (> 60)
	392.00 (4 cm H_2O)	no ignition (> 60)
	490.00 (5 cm H_2O)	no ignition (> 60)