Safety of Electronic Apex Locators and Pulp Testers in Patients With Implanted Cardiac Pacemakers or Cardioverter/Defibrillators

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Abstract

The purpose of this study was to determine if electronic apex locators (EAL) or electric pulp testers (EPT) interfere with the function of implanted cardiac pacemakers (ICP) or cardioverter/defibrillators (ICD). Twenty-seven patients with ICPs or ICDs had continuous electrocardiogram monitoring and device interrogation to detect interferences during the use of two types of EALs and one EPT. No interferences were detected by any ICP or ICD. In six patients, with intermittent pacing, a significant increase in pacing was observed during EAL/EPT stimulation ($p < 0.05$). Examination of RR intervals (a measure of intrinsic heart rate) demonstrated significantly longer RR intervals (slower intrinsic heart rate) during EAL/EPT stimulation ($p < 0.05$). Evaluation of the electrocardiograms for each patient failed to show any abnormalities in pacing during testing. These findings led us to conclude that the increased pacing frequency observed was related to a slower intrinsic heart rate and not electrical interference with the cardiac devices. In conclusion, the two EALs and one EPT used in this study did not interfere with the functioning of any of the cardiac devices tested. *(J Endod 2006;32: 847–852)*

Key Words

Electronic apex locators, electronic pulp testers, implanted cardiac pacemakers, implanted cardioverter/defibrillators

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0099-2399/$0 - see front matter

doi:10.1016/j.joen.2006.03.005

The increasing use of implanted cardiac pacemakers (ICP) and cardioverter/defibrillators (ICD) raises concern regarding electrical interference that might cause device dysfunction or patient harm. In dentistry, the electric pulp tester (EPT) and electronic apex locator (EAL), which are approved by the Food and Drug Administration, are routinely used in providing endodontic treatment. Both of these devices apply an electric current directly to the patient's oral tissues. The American Association of Endodontics estimates that nearly 16 million root canal procedures are performed every year (1). It is likely that patients with ICP/ICDs needing endodontic care will be encountered frequently. The dental profession has become increasingly aware of the possibility of electrical interference in patients with ICP/ICDs. Currently, EALs and EPTs are not recommended for use in patients with ICPs (2, 3). No current recommendations from the manufactures (2, 3) exist for their use in patients with ICDs. However, because of the variety and sophistication of systems available and lack of current research, there is confusion over which dental equipment should be a cause for concern.

The ICP and ICD are battery powered and implanted subcutaneously in either the pectoral or abdominal location. The devices are connected to the heart by electrodes and lead wires through which the cardiac rhythm is monitored and treatment, when indicated, is delivered by sending electrical charges to the heart. The life span of these devices is primarily determined by their battery, which lasts from 3 to 10 yr depending on the specific device and functions.

Pacemakers correct bradycardia or an abnormal heart rate by stimulating (pacing) the heart. The first ICP was implanted in 1958 (4). Pacemakers at this time were nonprogrammable, asynchronous (fixed rate) devices that stimulate (pace) the heart, usually the ventricle (single chamber), continuously at a predetermined (fixed) rate. Since then, ICPs have evolved into multiprogrammable, synchronous (demand) devices that stimulate the heart, ventricle and or atrial (dual chamber), only when needed.

Defibrillators function to correct cardiac arrhythmias. The first ICD was implanted in 1980, and ICD use was approved by the Food and Drug Administration in 1985 (5). Many of the ICDs today have incorporated pacing functions that allow them to treat patients with both bradycardias and tachycardias (6). Tachycardia, depending on the severity, can be treated by antitachycardia pacing, cardioversion pulses, or defibrillating shocks for more seriuous arrhythmias such as ventricular fibrillation (7).

The function of ICDs and most ICPs is to sense the intrinsic cardiac electrical activity and deliver appropriate electrical therapy to the heart when indicated. Electromagnetic interference may interfere with the function of these cardiac devices in several ways. Interpretation of an extraneous electrical signal as a cardiac signal in origin may temporarily inhibit pacing of an ICP (8). With ICDs, the electrical signal may be sensed and result in antitiachycardia pacing or the delivery of an inappropriate defibrillating shock (9). The electrical signal may by interpreted as noise and temporarily cause reversion of an ICP to an asynchronous pacing mode, or the signal may inappropriately reprogram the cardiac device (8). Conducted electrical energy, such as that produced from electrocautery, may travel down the lead wires to the heart and induce ventricular or atrial fibrillation (8).

EALs are used to determine working length within a tooth for endodontic treatment by measuring the electrical impedance between two electrodes using multiple signal frequencies (10). The accuracy of EALs in determining working length has led to a

decrease in the number of radiographs required during root canal treatment (10). EPTs are used to determine vitality of pulpal tissue within teeth. During testing, the strength of the stimulus is increased from 15 to 350 volts, for the device used in this study (2). The value of EPTs lies in their ability to determine pulp vitality since other noninvasive vitality testing (thermal testing) becomes less reliable as people age (11).

The available literature evaluating interference between EAL/EPTs and ICP/ICDs is limited, and conclusions are difficult to draw. There is only one study evaluating the effects of EALs on pacemakers. In 2002, an in vitro study reported that four out of five EALs tested with a single pacemaker showed normal pacing and only one produced an irregular pace recording on an oscilloscope (12).

In 1974, an in vivo study using dogs showed that an EPT caused mode switching in a pacemaker to fix rate pacing (13). That study is commonly cited as a rationale for not using EPTs in patients with ICPs. However, in 1974 ICPs were relatively immature in the evolution of pacemaker sophistication. Further, the type of interference that was reported would not be problematic to patients, since functional pacing continued. Three more recent in vitro studies reported no effects of EPTs on pacemakers (14–16).

Only two studies evaluate the effect of EPT use on ICPs in humans. In 1975, an in vivo study reported no effect in a selected subset of 14 patients using various ICPs (by Medtronic, Cordis, and General Electric) evaluated by electrocardiography (17). None of the ICPs tested are implanted in patients today. In 1991, a group in Germany studied EPT use in 26 patients with ICPs. The report found no interference from EPT use on pacemaker rate or electrocardiogram morphology (18). EALs were not tested, and patients with ICDs were not included.

A 1996 case reported on a patient with a fixed-rate pacemaker requiring root-canal treatment. Under consultation with the patient's cardiologist, an EAL was used. The patient experienced no adverse effects immediately or with follow-up (19). To our knowledge, no other human in vivo data is available on interactions of EALs with ICPs, and no data is available on the effects of any electrical dental device with ICDs.

Currently, manufacturers of EPTs and EALs warn against using these devices in patients with ICPs (2, 3). Such warnings are based on speculation of potential risk of electromagnetic interference (EMI) rather than on scientific evidence. The purpose of the current study was to evaluate possible interactions of EPL/EAT use on ICP/ICDs in adult patients.

Methods

Patient Recruitment

The study protocol was approved by the Oregon Health & Science University Institutional Review Board before enrolling patients. All patients aged 18 to 90 of any race or gender with working ICPs or ICDs were eligible for enrollment. The first five patients were recruited before their scheduled operation for their ICP/ICD generator change in the electrophysiology laboratory, where ready cardiovascular support and physical access to the device were available. The remaining patients were recruited after their regularly scheduled follow-up visit in the cardiology clinic. Patients who would not give written, informed consent were not enrolled. Patients who were pacemaker dependent (an intrinsic heart rate less than 40 beats/min) were excluded from this study. All willing patients signed informed consent before participation.

Each of the first five patients was placed under conscious sedation and local anesthesia per usual protocols. After the existing cardiac device and lead ends had been exposed, but still in the tissue pocket, the telemetry wand was placed over the device in a sterile plastic sleeve for testing. The remainder of the implant operation proceeded under the direction of the supervising electrophysiologist. After these first five patients, all results were reviewed for any occurrence of adverse events by a cardiologist (CSB).

TABLE 1. Specifications of Dental Equipment Tested

	EAL1	EAL2	EPT
Device name	Root ZX	Endo Analyzer	Endo Analyzer
Voltage	80 mV AC	2 V AC	15-350 V AC
Amperes (maximum)	10 uA	10 uA	1.5 mA
Frequency	0.8-2 KHz	0.5-8 KHz	10 KHz
Power supply	7.5 V DC	9.0 V DC	9.0 V DC

EAL1, electronic apex locator #1; EAL2, electronic apex locator #2; EPT, electronic pulp tester.

As no adverse events were noted in the first five patients, additional patients were enrolled during their regularly scheduled follow-up visit in the cardiology clinic. The following data were obtained from each patient: age, gender, type of implanted cardiac device, manufacturer, date of implant, indication for original implant, and number and position of leads. Each device was interrogated to obtain mode, bradycardia pacing parameters, sensing parameters, rhythm detection, and therapy parameters. When necessary, pacing rate was decreased to document the underlying rhythm then returned to its previous rate. In patients with ICDs, tachyarrhythmia therapies (i.e. defibrillating shocks) were disabled. In some devices (Ventritex), disabling tachyarrhythmia therapies also disabled tachyarrhythmia sensing, leaving only bradycardia pacing capabilities. Otherwise, there were no changes in any setting made to any device. All patients were told they could stop any time by raising their hand. Patients were awake during testing with the telemetry wand in place.

Stimulation Protocol

The dental devices tested were: (a) Root ZX (Morita Corp, Irvine, CA) and (b) Endo Analyzer Model 8005 (Sybron Endo, Orange, CA). The Root ZX is an electronic apex locator. The Endo Analyzer has dual functions as an electonic apex locator and electric pulp tester. Device specifications are shown in Table 1. With the telemetry wand in place, the surface and intracardiac electrocardiogram were continuously printed during testing, which consisted of seven phases each lasting 30 s. During the test, the rhythm was continuously observed by a cardiologist (CSB). Phase 1 (Rest 1) was recorded at rest, to serve as a baseline of normal device function. Phase 2 (EAL1) was recorded during stimulation with the Root ZX, followed by phase 3 without stimulation (rest 2). Phase 4 (EAL2) was during stimulation with the Endo Analyzer in the electronic apex locator mode, followed by phase 5 without stimulation (rest 3). Phase 6 (EPT) was during stimulation with the Endo Analyzer in the electric pulp tester mode, followed by phase 7 without stimulation (rest 4). Following all recordings, each device was interrogated to check for program changes, mode switching or tachyarrhythmia sensing, and all tachyarrhythmia therapies were enabled.

The EALs and EPT were employed to simulate normal clinical use by a practicing dentist (BLW). The contrary electrode was placed in contact with the patient's buccal mucosa using a metal clip placed in the corner of the mouth. The primary electrode (probe) was held in contact with each patient's gingival tissue. We did not have the patient hold the contrary electrode with their hand as is sometimes done in clinical practice. Function of EAL/EPTs was monitored to ensure good contact, but data from the devices were not recorded or analyzed.

Predetermined criteria for cessation of EAL/EPT use were: ventricular pause >2 s, detection of ventricular tachyarrhythmia by the defibrillator, sudden unexplained observable mode switch from the pacemaker, syncope, patient discomfort, or preference.

Data Analysis

All printed electrocardiograms were reviewed after the completion of the protocol. Each phase was reviewed, and the following data recorded: number of intrinsic heart beats, number of premature beats, number of paced beats, and RR interval (time duration between two consecutive R waves for each beat on the electrocardiogram). For all patients who were 100% ventricular paced (three total) the mean RR interval in milliseconds (*pacing frequency*) for each phase of testing were assessed. For all intermittently paced patients (six total) the *percent of pacing* (paced beats / total beats) for each phase of testing were assessed. For all patients who were not 100% paced (24 total), which includes intermittently paced patients (six total) and patients who had no pacing activity during testing (18 total), the mean RR interval in milliseconds for all naturally occurring heart beats, a measure of the *intrinsic heart rate*, for each phase of testing were assessed.

The differences between phases for the *pacing frequency*, *percent of pacing*, and *intrinsic heart rate* were assessed with paired *t* tests. The following grouping of data was used for this analysis: (a) stimulation phases in total (EAL1, EAL2, and EPT) versus the rest phases in total (rest 1-4), and (b) each stimulation phase individually versus the rest phases in total. Differences between the rest phases were assessed with ANOVA. The data were examined for any evidence of abnormal sensing, abnormal detection of tachyarrhythmia, abnormal device mode switching or program changes.

Of the 27 patients tested, only seven had devices that recorded RR intervals on the electrocardiograms. For the other 20 devices, the RR interval for each heart beat was manually recorded by measuring the distance between each R wave on the printed electrocardiogram using a digital micrometer (Mitutoyo, Tokyo, Japan), accurate to within 0.02 mm. The RR intervals for the seven patients with the recorded RR intervals on the electrocardiogram were also measured manually. These measurements were done in a blinded fashion by covering the RR interval data recorded on the electrocardiogram by the cardiac device until all the manual measurements were complete. The manually recorded RR interval data was compared with the RR interval data recorded by the cardiac devices for these seven patients using paired *t*-test. The results revealed no significant difference between the manually recorded RR interval and the RR interval recorded by the cardiac devices ($p = 0.425$, mean difference 1.17 ms, SD 12.14 ms). All data analysis involving RR interval data was performed with both the manually measured and device recorded RR intervals for these seven patients, with no significant differences seen in the results. The following results presented were obtained using the device recorded RR interval data for the seven patients for which it was available and the manually recorded RR interval data for the 20 patients in which the device recorded data was not available.

Results

Patients

Approximately half of the patients declined to participate, either because of unwillingness, pacemaker dependence, or inability to give informed consent. Twenty-seven patients were included in the study, five in the electrophysiology laboratory, and 22 in the cardiology clinic.

The details of the patient population with average settings for tachycardia therapies and bradycardia pacing are listed in Table 2. All patients were able to complete the testing protocol and no patient complained of significant discomfort during or after testing. Summary of the cardiac devices tested are found in Table 3. Twenty-three (85%) of the implanted devices were ICDs and four (15%) were ICPs. Five devices had been implanted in the left abdomen, while all others had been implanted in the left pectoral region. The leads of the cardiac devices tested represented a range of types and manufacturers, and included four true bipolar leads, eight types of integrated bipolar leads, and one epicardial patch.

TABLE 2. Patient and Device Characteristics

	Mean ± S.D.
Age	61.5 ± 13.5
Gender (f/m)	9/18
Years since implant	3.6 ± 3.2
Sensitivity of v lead (mV)	0.95 ± 0.97
Lower rate bpm	46 ± 11
Detection thresholds	
VT (ms)	363 ± 34
VF (ms)	299 ± 20
Device indication (no.)	
VT	15 (55%)
VF	8 (30%)
Sick sinus syndrome	2 (7%)
Atrioventricular block	1 (4%)
Tachy/brady syndrome	1 (4%)

VT, ventricular tachycardia; VF, ventricular fibrillation.

Only two patients were in DDD pacing mode, while all other patients were in VVI pacing mode. These letters describe the pacing mode as set forth by the NBG (North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group Generic Pacemaker Code). DDD is the NBG code for dual-chamber pacing, which is pacing and sensing in both the atrium and ventricle with dual responses (inhibited or triggered pacing) to sensing of an intrinsic heart beat. VVI is the NBG code for ventricle pacing in which pacing and sensing occur only in the ventricle and pacing is inhibited if an intrinsic heart beat is sensed. Four of the 27 patients had active rate-modulation programming that use special sensors or programming to monitor body changes (motion of limbs, respiratory rate or changes in the intrinsic heart rate) to help adjust the heart rate to meet the changing demands of the body. Rate-modulation programming is denoted by the letter "R" in the fourth position of the NBG code (i.e. DDDR, VVIR).

Results of Testing

No adverse events were detected in any patient. Specifically, there were no periods of abnormal detection by any pacemaker, no ICD detected any tachyarrhythmia, and no artifacts were observed on any of the intracardiac electrocardiograms. No patient experienced palpitations or any cardiovascular symptom.

Eight of the 27 patients were in normal sinus rhythm during the entire study. Nine patients experienced premature beats. In eight of these patients, ectopic beats were encountered with equal frequency throughout the study both with and without EAL/EPT use (including patient #1, who had periods of frequent ectopy including trigeminy and interrupted pacing, but no discernable difference with or without EAL/EPT use). In patient #16, six PVCs were encountered with EAL/EPT use and eight without, however, all six PVCs during dental device testing occurred during EAL2, with none experienced during testing with EAL1 or EPT. The PVC's were seen on both surface and intracardiac electrograms, and were not thought to represent electrical artifact.

Nine of the 27 patients were actively paced during the study. Of these nine patients, three were 100% ventricular paced. This includes patient #5 who was 100% ventricular paced during all test phases except in phase 5 (rest 3) and phase 6 (EPT) in which pacing occurred 88% and 89%, respectively. Patient #5, during these phases, had episodes of intrinsic heartbeats occurring before the programmed interval for delivery of a paced beat. Two of the three patients who were not 100% ventricular paced did not have rate-modulation programming, which includes patient #5. For these pa-

TABLE 3. Summary of Devices Tested and Outcome

ICDs	Pt# Device	Mode	rest	EAL1	rest	EAL2	rest	EPT	rest
Guidant	7 Ventak Prizm 2 VR 1860	VVI	nsr	nsr	nsr	nsr	nsr	nsr	nsr
	9 Ventak Prizm 2 VR 1860	VVI	nsr	nsr	nsr	nsr	nsr	nsr	nsr
	12 Ventak Prizm VR 1850	VVI	nsr	nsr	nsr	1 pc	2 pc	nsr	nsr
	20 Ventak Mini 2920	VVI	nsr	nsr	nsr	nsr	nsr	nsr	nsr
	21 Ventak Prizm VR 1850	VVIR	29%	30%	9%	29%	12%	38%	9%
	22 Ventak Prizm 2 VR 1860	VVI	sb	sb	sb	sb	sb	sb	sb
	25 Ventak Mini IV 2920	VVI	0%	27%	0%	26%	0%	31%	0%
Medtronic	1 Jewel + 7220	VVI*	10%	28%	16%	3%	15%	6%	15%
	2 Gem III R 7231cx	VVI*	nsr	nsr	nsr	nsr	nsr	1 pc	nsr
	3 Gem II DR 7273	DDD	100%	100%	100%	100%	100%	100%	100%
	4 Gem DR 7271	VVI	nsr	1 pc	nsr	nsr	nsr	nsr	1 pc
	6 Micro Jewel 7221	VVI*	nsr	nsr	nsr	nsr	nsr	nsr	nsr
	8 Micro Jewel II 7223 cx	VVI	afib	afib	afib	afib	afib	afib	afib
	10 Gem III VR 7231	VVI	3 pc	3 pc	2 pc	3 pc	4 pc	2 pc	3 pc
	13 Gem II VR 7229	VVI	nsr	nsr	nsr	nsr	nsr	nsr	nsr
	16 Micro Jewel 7223	VVI	2 pc	nsr	1 pc	6 pc	1 pc	nsr	4 pc
	19 Micro Jewel 7221	VVI*	nsr	nsr	nsr	nsr	nsr	nsr	nsr
	24 Gem 7227	VVI	1 pc	nsr	1 pc	1 pc	2 pc	1 pc	2 pc
	27 Gem II VR 7229	VVI	1 pc	nsr	nsr	nsr	nsr	nsr	nsr
Ventritex	5 V-186 HV3 Profile MD	VVI	100%	100%	100%	100%	88%	89%	100%
	11 V-185 D Contour II	VVI*	nsr	1 pc	nsr	nsr	nsr	nsr	nsr
	23 V-185 AC Contour II	VVI	nsr	nsr	nsr	nsr	nsr	nsr	nsr
	26 V-186 HV3 Profile MD	VVI	sa	sa	sa	sa	sa	sa	sa
Pacemakers Medtronic	14 Kappa KDR 901	VVI	47%	74%	47%	52%	61%	71%	73%
	15 Elite 7076	DDDR	4%	10%	7%	14%	10%	31%	10%
	17 Kappa KSR 401/403	VVIR	100%	100%	100%	100%	100%	100%	100%
	18 Sigma SSR 303	VVIR	46%	71%	39%	44%	44%	42%	35%

Devices tested grouped by type and manufacturer. Because Ventritex tachyarrhythmia therapics could not be turned off without also turning off sensing, these essentially were bradypacing only devices at the time of testing. Devices implanted into left upper abdomen are denoted by (*), all other devices were implanted in left pectoral region. nsr, normal sinus rhythm; pc, premature ventricular contraction; sb, sinus bradycardia; afib, atrial fibrillation; sa, sinus arrhythmia. Numbers indicate percent ventricular paced, with underlying sinus rhythm, except Pt 15 who was 100% atrially paced.

tients there was no change in the pacing interval (RR interval of paced beats) between any of the rest phases or stimulation phases with the dental devices. The RR interval for each paced beat remained constant throughout testing with these two patients with no delayed or premature pacing.

The third 100% ventricular paced patient, patient #17, had rate-modulation programming turned on. During stimulation with the dental devices (EAL1, EAL2, and EPT), the pacing frequency (paced heart rate) decreased slightly to a rate of 61, 59, and 59 ppm (paced beats per minute), respectively, as evidenced by the increasing RR interval between paced beats. Over the subsequent 30-s rest phases, after the stimulation was stopped, the pacing frequency increased to about 63 ppm by the end of the rest phases. The device was a Medtronic Kappa KSR 401 pacemaker that had been placed 6 wk earlier for tachy/brady syndrome. Pacing mode was VVIR. The rate-modulation programming included 9% activity of daily living rate, which adjusted the paced heart rate in accordance with changes in the respiratory rate. The optimization for the rate-modulation was on, and activity contribution set at minimum. The change in the pacing frequency throughout testing did not exceed the 9% activity of daily living rate. Changes in the paced heart rate during testing may be explained by changes in the patients respiratory rate during testing. Breath holding by the patient during the stimulation phases, when the probe was contacted to the oral cavity, would result in a detectable decrease in the respiratory rate over the 30 s period and result in slower pacing as measured by an increasing RR interval. A return to a normal breathing pattern by the patient, after the removal of the dental device probe from their mouth, would result in an increase in the pacing frequency to the normal resting rate as determined by their respiratory rate at rest.

Six patients were paced intermittently during the testing protocol. The percent of pacing during the combined stimulation phases with the dental devices (EAL1, EAL2, and EPT) shows a significant increase in pacing compared to the combined nonstimulation rest phases; 34.8% versus 22.4%, respectively ($p = 0.026$). No significant difference in the percent of pacing between the four separate rest phases 1 through 4 was detected ($p = 0.988$). Evaluation of each stimulation phase independently shows a significant increase in the percent of pacing during EAL1 (43.4%, $p = 0.026$), EPT (38.4%, $p = 0.023$) and a nonsignificant trend of an increase in pacing during EAL2 use (30.9%, $p = 0.148$) compared to the percent of pacing of the combined rest phases (22.4%) (Fig. 1).

The intrinsic (natural) heart rate for all patients who were not continuously (100%) paced was evaluated by comparing the mean RR interval for each phase of testing. The mean RR intervals of the stimulation phases (EAL1, EAL2, and EPT), assessed combined or individually, were significantly longer than the mean RR interval of the combined rest phases (p values <0.001); (Fig. 2). The average increase in the mean RR interval during the stimulation phases was 32 ms, which is a decrease in the heart rate of about 2 bpm. No significant difference in the mean RR interval between the four separate rest phases 1 through 4 was detected ($p = 0.946$). Fig. 3 is an example of patient #23 showing the change in the heart rate, as measured by the RR interval, over the entire testing protocol.

Examination of the RR intervals on the electrocardiograms of all patients in regards to the interval between each paced beat and the preceding heart beat revealed the following. First, for all patients without rate-modulation programming, each paced beat occurred at the time interval determined by the programming of the cardiac device. There was no detection of any delayed or premature pacing during the testing protocol. Second, for all patients with rate-modulation programming, each paced beat occurred within the specific range as determined by the cardiac device programming.

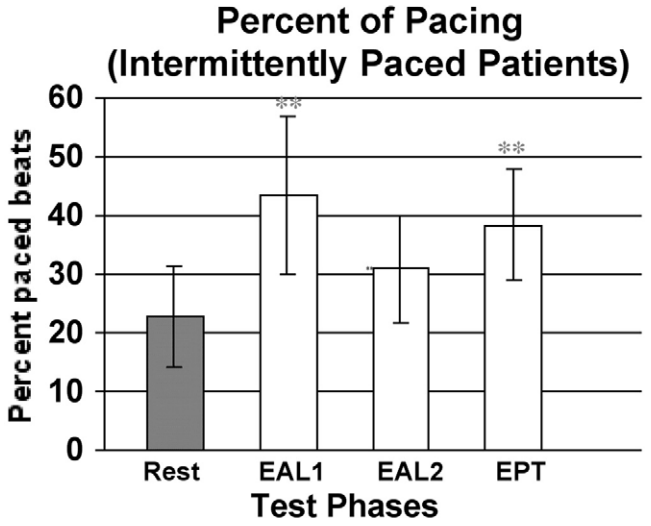

Figure 1. The mean *percent of pacing* of each phase of testing for the six patients (#1, 14, 15, 18, 21, and 25) with intermittent pacing. Significant differences (**) for EAL1 ($p = 0.026$, SE 0.0670), EPT ($p = 0.023$, SE 0.0493) and a nonsignificant trend for EAL2 ($p = 0.148$, SE 0.0498) (paired *t*-test) were seen when comparing each stimulation phase with the combined rest phases 1 through 4 (rest). No significant differences detected between the individual rest phases 1-4, $p = 0.988$ (ANOVA). *Percent of pacing* calculated by (# paced beats/# total heart beats). Error bars represent standard error.

Discussion

The patient population in this study represents patients coming to a university electrophysiology service and not necessarily those in a typical endodontic practice. The majority of the devices tested were ICDs. This reflects the fact that ICDs require more frequent generator replacement, that they receive more regular follow up in clinic, and that we excluded patients who were pacemaker dependent. Many of the devices we tested were newly implanted. Although one device was 14 yr old, it is difficult to be certain that other older devices would behave similarly.

There are two advantages to studying ICDs. First, ICDs have an increased sensitivity, since they are designed for early detection of ventricular tachyarrhythmias. Second, the most likely cause of concern is the detection of electromagnetic interference as false ventricular tachycardia or fibrillation by the ICD resulting in an inappropriate shock therapy to be delivered (9).

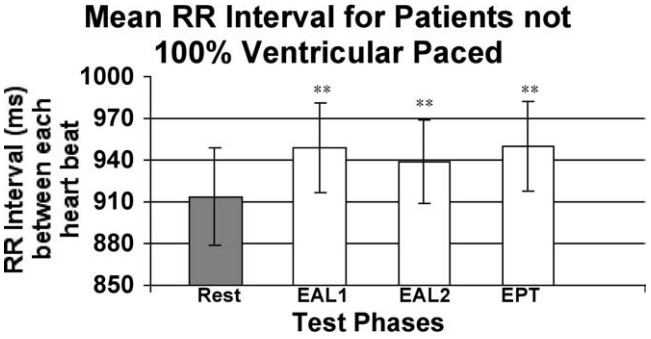

Figure 2. The data represents the mean RR interval (ms) of all heart beats for each phase of testing for patients not 100% ventricular paced (includes intermittently paced and nonpaced patients, 24 total). The mean RR interval of the stimulation phases (EAL1, EAL2, EPT), assessed combined or individually were significantly longer than the mean RR interval of the combined rest phases 1 through 4, ** ($p < 0.001$, SE 6.453 to 7.944) (paired *t*-test). The increased mean RR interval during each of the stimulation phases represents a slowing of the heart rate. No significant difference detected between the individual rest phases 1 through 4, $p = 0.932$ (ANOVA). Error bars represent the standard error.

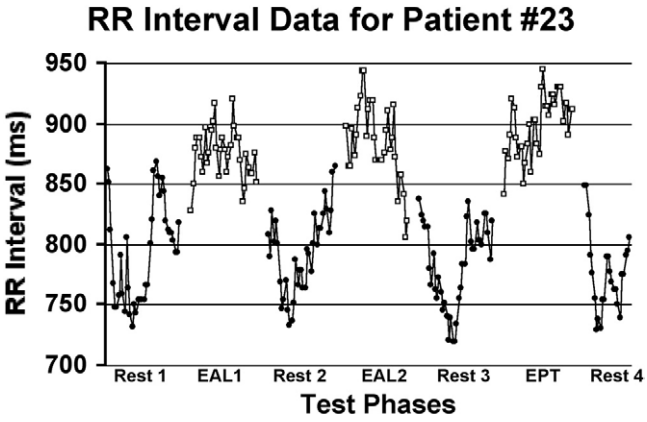

Figure 3. The RR interval (ms) of all heart beats for each phase of testing for patient #23. No paced beats were detected or delivered during the testing protocol. The RR intervals during the stimulation phases (EAL1, EAL2, and EPT) tended to increase, representing a slowing of the heart rate. During the rest phases (rest 1-4) the RR intervals tended to decrease, representing a faster heart rate.

A significant increase in pacing, measured as the percent of paced beats, was detected during stimulation of the oral tissues with the dental devices in patients who were intermittently paced. Before a conclusion may be drawn from this observation it is prudent to first determine if the increase in pacing seen during stimulation with the dental devices is a result of interference with the cardiac device or a normal response to a change in the patients physiology (i.e. intrinsic heart rate).

The underlying intrinsic heart rate during each phase of testing was determined for all patients not continuously paced, which included 18 patients who were not paced and 6 patients who were only intermittently paced during the testing protocol. The RR interval between each heartbeat was measured and then averaged for each phase of testing. The RR interval is the time in milliseconds between two consecutive heartbeats, a measure of heart rate. This data revealed an interesting finding in that a significant increase in the RR interval, a decreasing heart rate, was seen during each stimulation phase. The RR interval then decreased, an increase in the heart rate, during the following rest phases of nonstimulation. During the stimulation phases (EAL1, EAL2, and EPT) there was an overall significant decrease in the heart rate by an average of 2 to 3 beats/minute.

Two possible hypotheses are proposed for the slowing of the heart rate during the stimulation phases with the dental devices. First, the electrical current from the apex locators and pulp tester being delivered to the oral cavity may have stimulated the vagal nerve increasing the parasympathetic stimulation to the heart resulting in a slightly slower heart rate. A second possibility is that during the stimulation test phases, were a probe was placed in contact with oral tissues, patients responded by holding their breath. A slight decrease in a patients respiratory rate may have a physiologic response of slowing the intrinsic heart rate.

The RR interval for every paced beat was evaluated for all patients who were paced during the testing protocol, excluding patients with rate modulation programming. This data was compared with the programming of the cardiac devices, and this failed to show any delay or premature delivery of any paced beat. All paced beats occurred when expected as determined by the programming of the cardiac devices.

Patient #17, a patient who was continuously paced during the testing protocol, exhibited noticeable changes in the pacing frequency during both the stimulation and rest phases. These changes can be fully explained by potential physiologic differences. For patient #17, the pacemaker had rate-modulation programming turned on. For this particular device, the rate-modulation is based on respiratory rate, which the device samples at a rate of 16 Hz. Thus, during a 30-s period, if the patient held their breath or slowed their respiratory rate, the device would in fact respond quickly enough to slow the paced rate within that period, as we observed. Although the respiratory rates for patients with rate-modulation programming were not determined, it is reasonable to assume that placement of the EAL or EPT in their oral cavity during testing may result in a patient holding their breath or slowing their respiratory rate that would explain the changes in pacing frequency observed.

The six patients in this study with intermittent pacing had a resting heart rate close to the programmed lower rate limit of their cardiac devices. When the resting heart rate falls below the lower rate limit of the cardiac device a paced beat is delivered. A slight decrease in the intrinsic heart rate, as has been shown during EAL/EPT stimulation, would result in the intrinsic heart rate being slower than the lower rate limit set by their cardiac device a greater proportion of the time. This would result in an increased amount of pacing to maintain the heart rate at the lower limit set by the cardiac device.

There was no detection of any adverse symptom or effect from EPT/EAL use in any of the 27 patients. In no instance could we demonstrate that electrical stimulation with any of the dental devices was directly detected by any of the cardiac devices evaluated. Furthermore, we did not detect any interference with ICP/ICDs, including mode change, triggering, or device malfunction.

These findings lead us to conclude that the increased pacing seen during stimulation with the dental devices, in a subset of patients, was a result of the proper and expected functioning of the cardiac devices in response to a slowing of the patients natural underlying (intrinsic) heart rate and not to dysfunction of or interference with the cardiac devices.

Our study was limited to a relatively small patient sample size, and definitely not adequate to claim that no device in any patient would result in complications. However, we deliberately chose to enroll patients with any type of device or lead configuration, and purposely did not change any of the programmed settings. Our findings add confidence to allow more widespread study of interactions between these devices and challenge the manufacturers' claim that they not be used with ICP/ICDs. Future studies may wish to enhance the potential of detecting interferences by using the hand electrode or lowering the detection threshold of the devices.

No evidence of any interference was encountered when the EAL/EPTs were used as described in patients with working, implanted cardiac devices. Based on the findings in this study we conclude that EAL/EPTs are safe for use in patients with ICP/ICDs.

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