The Holmium:YAG laser can successfully ablate atherosclerotic tissue but its combination of high pulse energy (PE) and short pulse duration (PD) has been associated with the generation of shock waves. To determine if this phenomenon contributes to laser-induced vasospasm we irradiated rabbit aorta, isometrically mounted with 2.5 grams of tension in oxygenated Krebs' buffer. The tissue was irradiated with a Holmium:YAG laser (2100 nm, 6 Hz, 600-μm fiber) using both a free-running (PD: 250 microseconds) and Q-switched (PD: 200 nanoseconds) mode. PE was increased from 1-136 millijoules and tissue temperature was continuously monitored by thermocouple. Energy ablation threshold for each mode was determined by light microscopy. When PE was in the subablative range there was mild vasoconstriction that resolved to complete relaxation within 10 sec of cessation of laser irradiation. The contractile force was linearly related to PE (r=0.70, p<.001) and tissue temperature (r=0.50, p<.001) and was not different between the free-running and Q-switched modes (r=.82 vs .62, p=.008). At higher PE there was a distinctly different response with more severe contraction lasting for greater than 8 minutes. The contractile force was greater in the Q-switched than the free-running mode (55.5 vs 60.3 mg, p<.05) and this sustained response onset at lower PE in the Q-switched mode (6.7 vs 10.1 mg, p<.05). In the two modes the tissue temperature elevated by more than 3°C. Conclusion: Ablative doses of Holmium:YAG laser irradiation induce marked vasoconstriction particularly with the high peak PE associated with this mode. This suggests that significant vasospasm, possibly due to shock waves, may occur during Holmium:YAG laser angioplasty.

HISTOLOGIC AND ANGIOGRAPHIC EFFECTS OF A NEW PULSED HOLMIUM-YAG LASER IN NORMAL AND ATHEROSCLEROTIC HUMAN CORONARY ARTERIES

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Histologic effects of pulsed Holmium:YAG (Ho-YAG) lasers (2.1 μm wavelength) delivered by fiberoptic catheters have not been documented in human coronary arteries (CA). Histologic effects (HISTO) of increasing energy doses (50 to 700 mJ/pulse) were studied first in aortas strips. We then used angiographically normal (NL; n=15) or atherosclerotic (ATHERO; n=15) CA segments in 7 cadaveric hearts perfused at 1.6-2.0 MPa. The laser catheter (1.6-2.0 mm diam) containing multiple concentric optical fibers was advanced fluoroscopically over a guidewire. Pulses of 250 μs duration at 5-8 Hz and energy levels from 56-520 mJ/pulse (150-2,000 mJ/mm²) were delivered in 5 second groups. Quantitative angiography (ANGIO) PRE and POST laser demonstrated no perforations or dissections. Mean lumens diameters (μm; DIAAM) was unchanged in NL but increased significantly in ATERO segments. HISTO included vacuoles, enhanced staining, and ablated tissue "craters" 0.25-0.7 mm deep. Lack of changes in 7 ATERO segments was related to small energy delivery (56-186 mJ/pulse) in 5 calcific plaques. Ho-YAG laser energy can ablate atherosclerotic plaque in human coronary arteries and has minimal thermal effects on normal coronary arterial walls.

INCREASED DISPERSION OF VENTRICULAR ACTIVATION WITH EPICARDIAL VERSUS ENDOCARDIAL PACING IN CHILDREN AND YOUNG ADULTS

Gerald S. Serwer, Ravi Kodali, Brenda Eakin, Brian O'Connor, Macdonald Dick, J. C. Mott Hospital, Univ. of MI, Ann Arbor, MI.

Pacemaker electrode threshold testing is routinely performed with the patient at rest. Yet, with exercise such thresholds may change making programmed parameters inappropriate. To investigate changes in electrode thresholds with exercise 6 patients ages 14 to 24 yrs were exercised to exhaustion using the Bruce treadmill protocol. All had congenital heart disease that had been repaired with no significant residual lesions and no intracardiac shunts. Endocardial electrodes were present in 4 and epicardial electrodes were present in 2. Electrode thresholds were determined at all available pulse amplitudes (as determined by the implanted generator) prior to and immediately following exercise. Threshold testing was done at identical heart rates before and after exercise. Blood pressure, oxygen consumption (VO₂), and CO₂ production were continuously monitored. Average VO₂ for all patients increased from 6.6 ± 0.7 ml/kg to 26.5 ± 1.6 ml/min/kG. Maximal average CO₂ production was 24.2 ± 5.0 ml/kg. All patients reached anaerobic threshold with CO₂ production exceeding 10 ml/kg. In 5 of 6 patients the minimum pulse width necessary to pacer decreased at rest. Pulse amplitudes of 2.5 V or less with 0.1 to 0.3 ms changes. At 5.0 V or greater there was no change in threshold in any patient. In 1 patient there was no change in threshold at either 2.5 or 5.0 V amplitude. These changes imply a shift of the strength-duration curve with a decrease in the minimum amplitude necessary to pace at any pulse width. Whether such changes are due to changes in circulating catecholamine concentration and/or changes in blood temperature with its subsequent effect on membrane excitability is uncertain. This increase in membrane excitability with exercise should allow for use of decreased pulse amplitudes thereby increasing pacemaker longevity with less concern for the development of loss of capture with exercise.

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Pediatric Arrhythmias

CHANGES IN PACEMAKER THRESHOLD WITH EXERCISE IN CHILDREN AND YOUNG ADULTS

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While QRS duration and tRMSV were unchanged, mRMSV for EN was greater than that of EP. The interval with the greatest RMSV occurred in the 1st 20 ms interval in 7/9 studies for EN and at 60 to 80 ms in 6/7 studies with EP. As the magnitude of the RMSV is a function of the amount of myocardium depolarized during that interval, greater EN mRMSV implies greater early myocardial activation with less excitation dispersion later in QRS presumably due to sooner Purkinje activation with more organized wavefront propagation.