EXPERIENCE WITH A NEW PACEMAKER/CARDIOVERTER/ DEFIBRILLATOR

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We investigated the safety and efficacy of a new pacemaker/cardioverter/defibrillator (PCD; Medtronic 7216) in 12 pts (2 female) ages 51-73 yrs (mean 61) with recurrent VT/VF. All pts had previous myocardial infarction with mean ejection fraction .33 (.20 to .46) and had failed 1 to 7 (mean 4.7) antiarrhythmic drugs. The PCD provides VVI and antitachycardia pacing, programmable (0.1-34J) synchronous cardioversion or defibrillation in a stepwise, escalating fashion in response to the spontaneous heart rate. Monophasic shocks were delivered via 2 epicardial patches (5 pts), via 3 patches (6 pts) in a sequential or simultaneous fashion, or via transvenous leads and a subcutaneous patch (1 pt). Mean pacing threshold was 0.88 V and R wave amplitude 15.0 mV. Defibrillation threshold was ≤18J. During followup of 1-9 months (mean 6), 9 pts had spontaneous therapy: pacing for VT in 7, shocks for VT in 4, and shocks for VF in 3. 4 pts were unaware of VT onset or termination. VT slower than the detection rate was corrected by reprogramming (2 pts). One pt had pacing-induced acceleration of VT to VF with subsequent detection and automatic defibrillation. 1 pt with slow VT (120/min) had sinus tachycardia-triggered VT pacing that induced VT and subsequent shocks. Neither mechanical device failure nor failure to recognize a tachycardia occurred. We conclude that programmability, pacing and stepwise therapy improve effectiveness without sacrificing safety. Optimal function requires careful pt selection and programming.

THE AUTOMATIC IMPLANTABLE CARDIOVERTER/DEFIBRILLATOR: DOES LOW ENERGY CARDIOVERSION OFFER ANY ADVANTAGE OVER ANTITACHYCARDIA PACING IN PATIENTS WITH VENTRICULAR TACHYCARDIA?

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Since 1988, 39 patients (pts) received the multiprogrammable AICD Ventak P 1600 (CPI) and 16 pts the PCD (Medtronic). In contrast to the Ventak P, the PCD offers low energy cardioversion (LEC) as well as antitachycardia pacing capabilities (ATP). In 13/39 pts and 9/16 pts a sustained monomorphic ventricular tachycardia (mVT) (CL 270±34 ms and 290±45 ms, respectively) could be induced. LEC (≤ 4 joules) reproducibly terminated mVT in 6/13 pts but caused acceleration into VF in 7/13 pts. In 5 of these 7 pts the second shock (S_2 twice DFT) terminated VF but S_2 to S_5 failed to terminate VF in the other 2 pts. When VF was induced in the 6 pts with successful LEC VT termination, VF was terminated in 5 but not in the 6th pt. LEC was always associated with moderate to severe pain. In the 9/16 PCD pts termination of mVT was either attempted by burst (fixed # of pulses with decreasing CL between attempts) or ramp pacing (increasing # of pulses with decreasing CL within attempts). In 7/9 pts mVT was reproducibly terminated by either pacing mode, failed VT termination in 1 pt and caused VT acceleration in another pt. VF was always successfully terminated due to a different algorithm for VT and VF detection. Conclusions: Ventak P mediated LEC terminates mVT in 46% but accelerates VT to VF in 54% and prevents conversion of primary VF in 23%. In contrast, PCD mediated ATP terminated mVT in 78%, accelerated in 11% and was unsuccessful in 11%. Since LEC was always painful for the pts, ATP and not LEC should be the treatment of choice for mVT. In addition, different algorithms for VT and VF detection make the PCD superior to the Ventak P.

SINGLE CENTRE EXPERIENCE WITH A COMBINATION ANTITACHYCARDIA PACEMAKER AND DEFIBRILLATOR

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Since Dec 1989, an investigational device (Telectronics ATP 4210) that combines antitachycardia pacing (ATP) with defibrillating capabilities has been implanted in 12 pts at the University of Toronto. The indication for implantation was drug refractory ventricular tachycardia (VT,CL=400±44ms, mean±SE) in 9/12 or ventricular fibrillation (VF) in 3/12. The mean age was 47.9±5 (range 10-72), left ventricular ejection fraction 33.2±3.5% (range 19-55). Post implantation medications included amiodarone (n=7) or sotalol containing regimen (n=5).

RESULTS: There have been no deaths during follow-up for 3.7±.7 months (range 1-10). ATP was used from 1-500 times in the 4/7 pts who had ATP enabled. Spontaneous termination during reconfirmation prior to therapy occurred in 5 pts. Pace acceleration of VT occurred on 1% of ATP attempts and resulted in shock delivery on two occasions. Shocks were delivered to 5 pts, four with preceding ineffective ATP. One pacemaker dependent pt required a separate DDD-R antibradycardia device. Detailed episode logs allowed precise reconstruction of all events. In 3 pts post-discharge programming changes were performed in response to telemetry readings that revealed asymptomatic non-sustained and sustained VT. One pt had persistent double counting requiring disabling of ATP functions.

CONCLUSION: The combination of ATP with defibrillation in one device is effective and safe. Such a combination allows for defibrillation in the event of VT acceleration and cardioversion for ineffective ATP. Telemetry reveals a discordance between rapid rhythms and symptoms on some occasions.

ACUTE EFFECTS OF INTRAVENOUS PROPAFENONE ON THE INTERNAL VENTRICULAR DEFIBRILLATION ENERGY REQUIREMENTS IN THE ANESTHETIZED DOG

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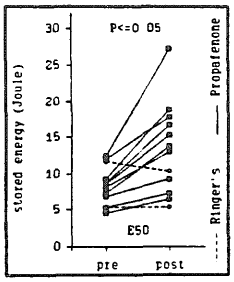

In 14 mongrel dogs the acute effects of intravenous propafenone (2mg/kg/10min and maintenance) on the internal ventricular defibrillation energy requirements (DER) were investigated using an intravenous spring and an epicardial patch electrode. Defibrillation was attempted 10 seconds after induction of ventricular fibrillation by testing randomly multiple stored energy levels. The percent successful defibrillation was plotted against the stored energy and the raw data fit by logistic regression. The DER for 50% (E50) and 80% (E80) defibrillation success increased post-propafenone treatment in every dog (Figure: continuous lines) and in the group (n=10) by a mean of 75% (E50: 8.4±2.4J to 14.7±5.9J, p≤0.05) and 59% (E80: 11.1±3.5J to 17.6±6.7J, p≤0.05), respectively. Plasma propafenone levels ranged from 1495±592ng/ml at the beginning to 1297±389ng/ml at the end of the defibrillation trials and were within reported therapeutic range. Two dogs received Ringer's solution instead of propafenone and showed no increase in DER (Figure: dashed lines). In two dogs the protocol could not be finished because of complications.

In conclusion, intravenous propafenone increases the internal ventricular DER in this canine model which is in contrast to previous results obtained in the pig model and supports the hypothesis that so-called class Ic agents elevate the DER. This may have important clinical implications in patients with an implantable defibrillator receiving concomitant drug therapy with propafenone and in patients receiving intravenous propafenone.