was effective for controlling 64% of patients, with partial control achieved in an additional 13%. The incidence of side effects necessitating drug withdrawal was 15%. In the study of Hammill et al, most patients (60%) had no significant cardiovascular disease, while 74% of Kerr's group had primary AF without associated heart disease. We had a lower success rate than these 2 previous groups, but our patient population all had underlying heart disease and chronic AF. The arrhythmia may have been more difficult to control due to these 2 variables.

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Electrophysiologic Effects and Efficacy of Recainam for Sustained Ventricular Tachycardia

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Recainam is an investigational antiarrhythmic drug recently introduced into clinical testing. It is a phenylalkyl urea derivative that produces a concentration-dependent decrease in membrane responsiveness and maximal upstroke velocity.[1] The reduction in maximal upstroke velocity displays use-dependence,[1] similar to the class 1A drugs. However, unlike class 1A drugs, but similar to class 1B agents, recainam does not prolong action potential duration.[1] Other studies demonstrated that recainam slows conduction with minimal effect on ventricular refractoriness,[2] effects consistent with a class 1C antiarrhythmic action. Thus, these preliminary investigations suggested that recainam possesses a unique electrophysiologic profile with properties of each of the 3 class 1 subclasses. Other preclinical studies have demonstrated recainam's efficacy in suppressing ventricular arrhythmias in the postinfarction dog model[3] and initial clinical studies have demonstrated that it is highly effective in suppressing ventricular premature complexes.[4–6] However, no studies to date have reported the electrophysiologic effects of recainam in patients with sustained ventricular tachycardia (VT). Such is the purpose of this report.

The subjects of this study were 10 men who had unimorphic VT inducible by programmed ventricular stimulation. Their mean age was 60 ± 12 years (range 34 to 74). All 10 patients had had a myocardial infarction. The mean left ventricular ejection fraction by contrast or radionuclide ventriculography was 0.38 ± 0.05 (range 0.32 to 0.47). The indication for the electrophysiology study was sustained unimorphic VT in 7 patients, cardiac arrest in 2 and syncope in 1. The patients had failed to respond to or had intolerable side effects with a mean of 2.5 ± 1.2 (range 1 to 5) antiarrhythmic drugs before treatment with recainam. Exclusion criteria included unstable angina, myocardial infarction within 3 months before the study and a left ventricular ejection fraction <0.30.

The study protocol was approved by the Institutional Review Board and all patients gave written informed consent before taking part in the study. The electrophysiology study was performed with patients in the fasting, unsedated state at least 5 half-lives after discontinuation

TABLE I Electrophysiologic Effects of Oral Recainam in 10 Patients

	Baseline	Oral Recainam	P value
Sinus cycle length	920 ± 213	767 ± 173	<0.05
Atrial-His interval	102 ± 26	127 ± 47	<0.05
His-ventricular interval	52 ± 12	62 ± 13	<0.05
QRS Interval	109 ± 16	126 ± 19	<0.01
SNRT	1,110 ± 206	1,088 ± 321	NS
$SNRT_c$	208 ± 103	367 ± 241	NS
WBB	455 ± 77	458 ± 117	NS
Atrial FRP, DCL 500	264 ± 34	247 ± 26	NS
Atrial ERP, DCL 500	225 ± 14	192 ± 33	<0.05
AVN FRP, DCL 500	444 ± 66	468 ± 113	NS
AVN ERP, DCL 500	357 ± 88	356 ± 131	NS
RVA ERP, DCL 600	261 ± 23	263 ± 29	NS
RVA ERP, DCL 400	244 ± 23	254 ± 25	NS
RVOT ERP, DCL 600	245 ± 7	260 ± 28	NS
RVOT ERP, DCL 400	250 ± 14	240 ± 0	NS

All measurements are in ms.
DCL = drive cycle length; ERP = effective refractory period; FRP = functional refractory period; NS = not significant; RVA = right ventricular apex; RVOT = right ventricular outflow tract; SNRT = sinus node recovery time; $SNRT_c$ = corrected SNRT; WBB = longest cycle length at which atrioventricular nodal Wenckebach block occurred during overdrive atrial pacing.

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TABLE II Effect of Oral Recainam on Induction of Ventricular Tachycardia

	Baseline			Recainam		
Pt	CL (ms)	BBB Morphology	ES for Induction (n)	CL (ms)	BBB Morphology	ES for Induction (n)
1	370	R	OVP	420	R	2
2	330	L	OVP	350	L	2
3	250	L	3	270	L	3
4	320	R	1	460	R	2
5	310	L	3	290	L	2
6	270	R	2	340	L	1
7	370	R	2	—	—	—
8	370	L	2	480	L	2
9	280	R	2	330	R	2
10	240	R	2	460	R	1

BBB = bundle branch block; CL = cycle length; ES = extrastimuli; L = left; OVP = overdrive ventricular pacing; R = right.

of all antiarrhythmic drugs. A detailed description of the electrophysiology study protocol has been published previously.[7] After a rest period of 5 minutes, baseline electrophysiologic parameters were measured (Table I). Ventricular programmed stimulation was performed at the right ventricular apex and right ventricular outflow tract at 2 drive rates (600 or 500 and 400 ms) with up to 3 extrastimuli. Sustained VT was defined as VT lasting >30 seconds or requiring intervention for termination. After the baseline electrophysiology study all patients were treated with oral recainam. All patients had a baseline 48-hour ambulatory electrocardiographic recording (Cardio Data Systems) before commencing treatment with recainam. At the time of the follow-up electrophysiology study, 9 patients were receiving 400 mg every 8 hours and 1 patient 600 mg every 8 hours. The follow-up electrophysiology study was performed after a mean of 10 ± 3 doses of recainam, between the fourth and eighth hours of the dosing interval (mean plasma concentration 2.7 ± 1.4 μg/ml).

The patients underwent hematologic and biochemical screening and a 12-lead electrocardiogram during the baseline state and during treatment with recainam. Patients in whom the induction of sustained VT was suppressed by recainam had a 24-hour ambulatory electrocardiogram before hospital discharge and were eligible to continue to receive recainam in the long-term outpatient phase of the study. This phase of the study incorporated clinic visits every month for the first 6 months and every 3 months thereafter. A resting 12-lead electrocardiogram and 24-hour ambulatory electrocardiographic recording were obtained at each clinic visit. All data were analyzed using a paired t test and are presented as mean ± 1 standard deviation. A p value of <0.05 was considered significant.

Recainam significantly shortened the sinus cycle length and atrial effective refractory period and significantly prolonged the AH and HV intervals and QRS duration (Table I). None of the other electrophysiologic parameters was altered significantly. After treatment with recainam, sustained VT was no longer inducible in 1 of 10 patients (no. 7) (Table II). Overall, the mean cycle length of induced VT was prolonged significantly by recainam from 309 ± 49 ms baseline to 383 ± 83 ms (p <0.05). One patient (no. 6) was not included in this analysis because VTs of different morphologies were induced in the baseline and follow-up studies.

Examination of the 12-lead electrocardiograms revealed that the sinus rate was significantly faster with recainam compared to baseline (80 ± 16 vs 69 ± 10 beats/min; p <0.05). Recainam significantly increased the PR interval (0.22 ± 0.04 vs 0.18 ± 0.03 seconds; p <0.001) and QRS duration (0.13 ± 0.02 vs 0.11 ± 0.02 seconds; p <0.01). There was no significant change in the QT interval.

The 1 patient (no. 7) in whom the induction of sustained VT was suppressed by recainam entered the long-term outpatient phase of the study. He continues to receive 1,200 mg of recainam daily and remains well after 18 months of treatment. However, his serial 24-hour ambulatory electrocardiograms have revealed frequent episodes of nonsustained VT associated with minimal symptoms. In the baseline state, this patient had virtually incessant VT. No adverse reactions were noted during treatment of any patient with recainam and no significant side effects were reported.

This study demonstrates that recainam significantly prolongs the resting AH and HV intervals and QRS duration but does not lengthen the atrial, atrioventricular nodal or ventricular refractory periods. These data confirm that recainam's main electrophysiologic action is to prolong conduction time without altering refractoriness. Although preclinical studies suggested that recainam has properties of each of the 3 class I subclasses, the present results suggest that its class 1C properties predominate in the clinical setting. With recainam, resting sinus cycle length was significantly shorter. This finding has been noted previously[4] and may be secondary to a mild negative inotropic or anticholinergic action. In the present study recainam produced a barely significant reduction in atrial effective refractory period. This finding could not be explained on the basis of its known electrophysiologic properties and must await reexamination in further clinical studies.

In 1 of 10 patients, recainam prevented the induction of sustained VT, which had been unresponsive to conven-

tional antiarrhythmic drug therapy. This patient has remained well over a follow-up period of 18 months. Although he has continued to show episodes of nonsustained VT on 24-hour ambulatory recordings during follow-up, these episodes are shorter, slower and less frequent than during the baseline state when his VT was virtually incessant. In patients in whom VT remained inducible, recainam significantly slowed the mean VT rate from 194 to 157 beats/min ($p < 0.05$). However, none of these patients was treated for a long term with recainam because this partial response was deemed inadequate. A notable observation in our study was the absence of significant side effects. Recainam was well tolerated by all 10 patients during short-term administration and by the 1 patient in the long-term study. In particular, no patient developed signs or symptoms of congestive heart failure. These results are similar to those found in previous studies of ventricular premature complex suppression.[4-6]

In conclusion, recainam may occasionally suppress the induction of sustained VT in patients who have failed to respond to other antiarrhythmic drugs. It appears to be well tolerated during short-term administration. Further studies will be necessary to determine its long-term efficacy and safety in patients with sustained VT.

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Automatic Implantable Cardioverter Defibrillator Implantation for Malignant Ventricular Arrhythmias Associated with Congential Heart Disease

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The prognosis for patients with congenital cardiac abnormalities can be improved by surgical correction of hemodynamics. With[1] or without[2] surgery, sudden death from malignant ventricular arrhythmias is an important potential problem in congenital heart disease. Pharmacologic arrhythmia control may be limited by side effects, noncompliance or unreliable indexes predictive of drug efficacy. The automatic implantable cardioverter difibrillator (AICD) is a potential alternative. The incidence of arrhythmic death is <2% per year following AICD implant[3] in adults with ventricular arrhythmias refractory to medical management. This success rate favors AICD therapy in selected patients with congenital heart disease. We present 4 such cases (Table I) and discuss their management.

The first patient (no. 1) experienced no spontaneous arrhythmia. Intervention was prompted by sudden death in 2 siblings within 2 years. Electrophysiologic study revealed ventricular fibrillation during both atrial and ventricular stimulation. A left paraseptal accessory pathway with short anterograde refractory period was also demonstrated. Intervention was required in the remaining 3 patients (nos. 2, 3 and 4) because of cardiac arrest during daily activities. Arrhythmias were not inducible in the electrophysiology laboratory to guide therapy. One of these patients (no. 4) experienced nearly fatal failure of empiric management at another hospital. AICD implantation was recommended as more likely to provide long-term survival than empiric drug therapy or cardiac transplantation. Direct surgical ablation was not attempted because of inability to map the site of origin of the ventricular arrhythmias.

Preoperative evaluation included catheter studies of electrophysiology and hemodynamics, as well as exercise testing to define maximal heart rate. The surgical approach was dictated by factors including residual pathologic anatomy and physiology, surgical adhesions, shunts and prostheses. The need for accessory pathway ablation (no. 1), aortic coarctation repair (no. 2) and transvenous pacemaker insertion (no. 3) were specific issues in these patients. A bipolar dual chamber pacing system, physical separation of the pacing electrodes from AICD rate sensing electrodes and reduced pacing amplitude in patient no. 3 avoided undesirable pacemaker–AICD interaction.[4]

Since defibrillation thresholds can increase postoperatively, AICD function was retested 1 week after implantation. AICD failure during this test in patient no. 2 was corrected by implantation of a transvenous spring-coil electrode, although patch-coil systems are usually less effective than large patch systems.[5] Postpericardiotomy syndrome in 2 patients (nos. 1 and 4) responded to anti-

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