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Intracardiac electrophysiologic study of dilazep in man

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We studied the electrophysiologic effects of intravenous dilazep in 10 hospitalized patients.

Dilazep (Cormelian®) is successfully employed in the treatment of ischemic heart disease because of its sustained vasodilating effect and strong platelet antiaggregating activity [1].

We studied the electrophysiologic effects of intravenous dilazep in 10 hospitalized patients (6 males and 4 females), whose ages ranged from 43 to 79 years. The electrophysiological study was carried out because of a history of vertigo (6 cases) and during an atrial pacing study performed for atypical chest pain (4 cases). All patients had normal sinus rhythm and absence of documented atrial and ventricular arrhythmias. All the patients gave informed consent to the study. The electrophysiological investigation was performed in control conditions and 10 (D′) and 30 (D″) min after the end of 10-min infusion of 0.1 mg/kg of the drug.

Dilazep induced a significant shortening of the following parameters: sinus node cycle length, corrected sinus node recovery time, effective and functional atrial refractory periods, and right ventricular effective and functional refractory periods (Table). The drug had no effects on AH and HV intervals, intra-atrial conduction time, sinuatrial conduction time or on the heart rates during which the atrioventricular Wenckebach phenomenon occurred (Table).

Our results indicate that dilazep induces an improvement of sinus node automatism, as indicated by the significant shortening of cycle length and corrected recovery time with respect to the control values.

In previous studies we investigated the electrophysiological effects of nifedipine [2] and dipyridamole [3]. Nifedipine [2] induced a significant shortening of the sinus node cycle length but similar values of the corrected recovery time were observed before and after the drug. On the other hand, dipyridamole [3] shortened the recovery time but had no effects on cycle length. Thus, dilazep combines the effects of nifedipine and dipyridamole on the sinus node.

In our patients dilazep induced a significant shortening of atrial and ventricular refractoriness, whereas a prolongation of ventricular refractoriness was observed in animals when high dosages were used [4]. This difference between experimental and clinical effects is similar to

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TABLE

Electrophysiological data (mean ± SD) before and after dilazep.

	C	D′	D″	C vs. D′	C vs. D″	D′ vs. D″
SCL (msec)	900 ± 82	736 ± 115	804 ± 128	$P < 0.0005$	$P < 0.0025$	$P < 0.0025$
HRA-LRA (msec)	26 ± 7	25 ± 9	25 ± 6	NS	NS	NS
AH (msec)	108	108	108	NS	NS	NS
HV (msec)	43	43	43	NS	NS	NS
CSRT (msec)	352 ± 78	249 ± 80	288 ± 43	$P < 0.005$	$P < 0.05$	$P < 0.05$
SACT (msec)	98 ± 30	101 ± 35	100 ± 38	NS	NS	NS
WHR (beats/min)	150 ± 20	150 ± 20	150 ± 20	NS	NS	NS
AERP (msec)	284 ± 56	267 ± 47	268 ± 51	$P < 0.01$	$P < 0.01$	NS
AFRP (msec)	332 ± 46	313 ± 45	314 ± 46	$P < 0.005$	$P < 0.005$	NS
RVERP (msec)	246 ± 44	228 ± 28	230 ± 26	$P < 0.05$	$P < 0.05$	NS
RVFRP (msec)	282 ± 50	268 ± 39	268 ± 37	$P < 0.05$	$P < 0.05$	NS

SCL = sinus node cycle length; HRA-LRA = intraatrial conduction time; AH = atrial-His; HV = His-ventricular; CSRT = corrected sinus node recovery time; SACT = sinuatrial conduction time; WHR = Wenckebach heart rate; ERP = effective refractory period; FRP = functional refractory period.

that of bretylium [5]. As regards dilazep, the observed action in man may represent indirect sympathetic effects elicited by drug-induced vasodilatation [1].

In conclusion dilazep: (1) improves sinus node automatism; (2) does not affect sinuatrial, intraatrial and atrioventricular conduction; and (3) shortens atrial and ventricular refractory periods.

The knowledge of the electrophysiologic effects is important for the appropriate choice of a specific antianginal therapy. Further evaluation in patients with pharmacologically induced depression of sinus node automatism will provide a clearcut allocation of this compound among the antianginal drugs usually employed in the clinic.