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Atrial Activity in Atrial Fibrillation after Intravenous Verapamil

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ABSTRACT

In 7 patients with atrial fibrillation the rate of the atrial activity was studied before and after intravenous injection of verapamil. No consistent change in this rate was observed.

INTRODUCTION

Verapamil (Isoptin®, Meda) was originally introduced for the treatment of myocardial ischaemia (3, 4, 6), but it has also been shown to possess antiarrhythmic properties (7, 10).

In atrial fibrillation verapamil has been attributed a unique ability not only to reduce the ventricular rate but also to regularize the ventricular response (5, 9). The mechanism of this regularizing effect is unknown. Schamroth has proposed that in addition to its action on the atrio-ventricular (A-V) nodal conduction, verapamil may also stabilize the atrial activation front (9). Another possible mechanism is through an alteration of the atrial fibrillatory rate. The purpose of this study was to investigate the rate of atrial fibrillation before and after an intravenous injection of verapamil.

MATERIAL AND METHODS

Seven patients with atrial fibrillation who were in hospital for cardiac evaluation or other reasons comprised the material. They were all in a compensated cardiac status, and all were on maintenance digitalis therapy. The dose of digitalis had not been changed for the last few months prior to the investigation. Further data concerning the patients are given in Table I. The patients consented willingly to the investigation. For the investigation the patient lay in the supine position and a bipolar ECG with special amplification was performed according to a previously described technique (8). The paper speed was 100 mm/sec. The ECG was recorded prior to and continuously during the injection of verapamil. After the injection, for 15 min, ECG recordings were made for about 20 sec each minute. In one patient 10 mg verapamil were given within one minute intravenously (the same dosage as used by Schamroth (9)). In the remaining six patients a dose of 1 mg/10 kg body weight was administered. The injection time was 1 mg/min. This is in accordance with the recommendations for parenteral administration of the drug given in the Swedish Physicians' Desk Reference (FASS, 1975). The calculations of the atrial as well as the ventricular rate were made on ECG strips comprising 10–20 sec.

RESULTS AND COMMENTS

The ventricular rate was decreased by 16% 5 min after the injection and there was no further reduction after 10 or 15 min. In other words the effect of verapamil was sustained throughout the experimental period. In this small group of patients with atrial fibrillation no obvious decrease of the variation of R-R intervals, i.e. regularization of the ventricular response, was observed. However, the material was small and the mean age was high, and in older persons this regularization in inconsistent (9).

The results of the atrial rate (fibrillatory wave rate) calculations are given in Table I. There was no consistent change in the rate after the injection of verapamil. The fibrillatory wave pattern, which was measured in a rough qualitative way previously described (1), showed no alteration after the injection as compared with the pre-injection pattern.

DISCUSSION

The finding reported by several authors (5, 9) of a certain regularizing effect of verapamil on the ventricular response in atrial fibrillation is interesting. As many patients with this arrhythmia cannot be maintained in sinus rhythm after conversion, there is a need for all possible means of helping them to improve their haemodynamic performance.

Table I. *Review of the material*

Atrial and ventricular rates before (B) and after (A) intravenous injection of verapamil

Sex	Age (y.)	Cardiac diagnosis	Atrial rate		Ventricular rate	
			B	A[a]	B	A[a]
♂	63	Atherosclerosis	400	390	96	87
♂	60	Atherosclerosis	540	550	120	105
♂	60	Unknown	560	550	76	58
♂	58	Unknown	410	460	124	117
♀	65	Rheumatic heart disease	400	430	88	71
♀	64	Rheumatic heart disease	560	500	48	41
♀	52	Rheumatic heart disease	430	440	95	67

[a] Mean of the rates 5, 10 and 15 min after the injection was ended.

Even a moderate regularizing effect on the chaotic heart should be of benefit. A drawback observed by Schamroth (9) is that the effect of verapamil is less pronounced in older people. At the same time the need for an effect is greatest within this higher age group. We could not verify the regularizing effect but the material was small and this was not the primary purpose of the investigation.

If this effect exists at all, there are two possible modes of action. The first is an alteration of the A-V node conduction. The mechanism of action of verapamil on the A-V node has been studied previously (2). Schamroth has proposed that it has a stabilizing effect on the A-V node (9). The second possible mechanism is an alteration of the atrial activity, permitting a more regular transmission of impulses to the A-V node. This has not been studied previously, mainly due to the difficulties in evaluating the atrial activity without the use of special techniques. By the use of amplified bipolar ECG recordings, however, an evaluation of this activity is made possible. In this study there was no difference in the rate or appearance of the fibrillatory waves before and after the injection of verapamil. The regularizing effect of verapamil on the ventricular response in subjects with atrial fibrillation, described by some authors, must therefore be due to alterations of the conduction through the A-V node.

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