Comparative effects of adimolol, labetalol and propranolol on heart rate and blood pressure in man

J. G. RIDDELL, D. W. G. HARRON & R. G. SHANKS

Department of Therapeutics and Pharmacology, The Queen's University, Belfast, UK

1 The cardiovascular effects of adimolol, a new, potent, long acting β -adrenoceptor blocking drug were investigated in three studies.

2 In the first study blood pressure and heart rate were measured in five male volunteers before and at 2, 4, 6, 8, 24, 32, 48, 72 and 96 h after single oral doses of adimolol.

3 All doses of adimolol reduced supine, standing and exercise heart rates in a dose dependent manner. The maximum effect ranged from 18% following 25 mg to 29% following 600 mg and all doses showed an effect at 96 h (range 3.5–17.2%).

4 In the second study the effects of adimolol, 25 and 400 mg, labetalol, 200 and 800 mg, propranolol 40 mg and placebo were compared on supine and standing heart rate and blood pressure and on exercise heart rate before and at 2, 4, 6 and 8 h after single oral doses.

5 The exercise heart rate was significantly reduced at all times following adimolol 25 and 400 mg, labetalol 800 mg and propranolol 40 mg. At 2 h all the drugs significantly reduced standing systolic blood pressure.

6 In the third study, 4 h after single oral doses of adimolol 400 mg, labetalol 400 mg and propranolol 40 mg six subjects received serial 4 min infusions of phenylephrine. The blood pressure was measured after each infusion.

8 Labetalol 400 mg significantly shifted the blood pressure dose-response curve to the right. There was no difference between propranolol 40 mg and adimolol 400 mg.

9 These studies show that adimolol is a potent, long acting β -adrenoceptor blocking drug without evidence of α -adrenoceptor blockade in man.

Keywords adimolol propranolol labetalol cardiovascular effects phenylephrine responses

Introduction

Adimolol is a new, long acting β -adrenoceptor blocking drug which, in animals, has been shown to be without cardioselectivity, membrane stabilising activity or partial agonist activity. It has also been shown in animals that the drug has weak α -adrenoceptor blocking activity (Hoefke *et al.*, 1984) and if this was to occur in man then the drug would be a welcome addition to those available for the treatment of hypertension.

We have investigated, in normal male subjects, the effects of the oral administration of adimolol and compared it with labetalol, a β -adrenoceptor antagonist with α -adrenoceptor antagonist properties (Boakes *et al.*, 1971; Richards *et al.*, 1979) and propranolol, a non-cardioselective β -adrenoceptor blocking drug, on heart rate and blood pressure in the supine and standing positions and on an exercise tachy-

Correspondence: Professor R. G. Shanks, Department of Therapeutics and Pharmacology, The Queen's University of Belfast, The Whitla Medical Building, 97 Lisburn Road, Belfast BT9 7BL, UK

cardia. We have also compared its effect to that of labetalol and propranolol on the increase in blood pressure caused by increasing intravenous infusions of phenylephrine.

Methods

The cardiovascular effects of adimolol were investigated in three separate studies in healthy male volunteers who gave written informed consent to the procedures involved. The protocol of the studies had been approved by the Ethics Committee of the Queen's University of Belfast. In the first study (an open study), following an overnight fast, five healthy male volunteers (mean age, 21.6 ± 2.3 years, mean weight, 76.6 ± 8.5 kg) received oral doses of adimolol 100, 200 and 400 mg at 2 weekly intervals. Four of these subjects also received 25 and 50 mg and two also received 600 mg of the drug. Observations were made before and at 2, 4, 6, 8, 24, 32, 48, 72 and 96 h after each dose. At each observation time the subjects were supine for 15 min, standing for 3 min and performed moderate exercise by stepping on and off a box 46 cm high at a rate of 32 steps/min for 3 min. Heart rate and blood pressure were measured using a Criticon Exercise Monitor (Model 1165), at the end of the periods supine and standing; heart rate was measured within 5 s of completing exercise.

In the second study, the effects of the oral administration of single doses of adimolol (25 and 400 mg), labetalol (200 and 800 mg), propranolol (40 mg) and placebo were compared in five healthy male volunteers (mean age, 21.2 ± 1.2 years; mean weight, 73.4 ± 3.5 kg) who had not taken part in the previous study. Following an overnight fast each subject received, at weekly intervals, double-blind, in a Latin Square design obtained by omitting the adimolol 400 mg (due to its long duration of action). One week later each subject received adimolol 400 mg. The same observations were made as in the first study, before and at 2, 4, 6 and 8 h following the dose. Heart rate was measured over five consecutive cardiac cycles on Lead II of a direct writing electrocardiogram and blood pressure was measured using a Hawksley Random-Zero sphygmomanometer (Wright & Dore, 1970).

In the third study the α -adrenoceptor blocking activity of adimolol was investigated in a further six subjects (mean age, 20.5 ± 1.5 years; mean weight, 67.8 ± 4.5 kg). After a light breakfast containing no caffeine, on separate occasions separated by at least 2 weeks, each subject received in random order, double-blind, adimolol 400 mg, labetalol 400 mg and propranolol 40 mg orally. Prior to the

administration of the drug on each study day the subjects performed an exercise test as described above and the tachycardia was measured. Three and one half hours later an intravenous infusion of saline was commenced in the right arm. Four hours after the administration of the drug the blood pressure was measured (as the mean of three observations) in the left arm using a Random-Zero Sphygmomanometer (Korotkoff I and V). Phenylephrine, 40 $\mu\text{g}/\text{min}$, was infused for 4 min and the blood pressure again measured. The rate of infusion of phenylephrine was increased stepwise through the concentrations 60, 100, 150, 200, 300, 400 and 600 $\mu\text{g}/\text{min}$ until the systolic blood pressure rose by 30 mm Hg when the infusion was discontinued. After 30 min rest, an exercise heart rate was obtained as previously described.

Results in the text are expressed as the mean $\pm$ s.d. Statistical analyses of the second and third studies were made using analysis of variance. A statistically significant difference ($P < 0.05$) exists when any value of a parameter differs from another by more than the minimum significant difference.

Results

Dose ranging study

The results of the open dose ranging study indicated that all doses of adimolol reduced supine, standing and exercise heart rates in a dose dependent manner. The percentage reductions in the exercise tachycardia are given in Table 1. Maximum effects ranged from $18.2 \pm 5.6\%$ for 25 mg to $29.1 \pm 1.4\%$ for 600 mg and occurred between 4 and 8 h. All doses showed reductions in heart rate at 96 h (range $3.5 \pm 3.4\%$ to $17.2 \pm 5.1\%$ for 25 and 400 mg respectively). No marked reduction with any dose of the drug was seen on supine or standing blood pressure.

Comparative effects of adimolol, labetalol and propranolol

The second study compared the effects of placebo, adimolol 25 and 400 mg, labetalol 200 and 800 mg, and propranolol 40 mg in five subjects. The effect of the six treatments on supine, standing and exercise heart rate is given in Table 2. In the supine position the pre-treatment heart rate values in the six groups differed only for adimolol 400 mg which was significantly lower. Placebo had no effect on supine heart rate. All drugs significantly reduced this parameter when compared to placebo

Table 1 Mean percentage reduction ($\pm$ s.d.) in exercise tachycardia following single oral doses of adimolol to normal volunteers

Time (h)	Number of subjects					
	4	4	5	5	5	2
	Dose (mg)					
	25	50	100	200	400	600
2	7.0 ± 2.8	9.0 ± 7.8	7.6 ± 5.1	8.9 ± 4.0	12.6 ± 5.4	13.3 ± 1.8
4	16.0 ± 7.0	18.5 ± 3.8	21.5 ± 8.3	24.2 ± 2.9	27.9 ± 3.1	29.1 ± 1.4
6	17.8 ± 6.4	18.3 ± 5.2	22.7 ± 7.2	24.1 ± 3.4	26.7 ± 4.5	28.1 ± 1.7
8	18.2 ± 5.6	20.7 ± 2.0	20.9 ± 8.7	22.7 ± 3.1	26.5 ± 3.6	27.7 ± 3.4
24	13.2 ± 5.6	16.3 ± 1.6	19.8 ± 7.6	25.7 ± 4.5	26.9 ± 4.5	23.8 ± 0.8
32	12.1 ± 3.0	13.2 ± 2.0	17.7 ± 6.3	22.9 ± 2.5	24.6 ± 3.8	24.4 ± 1.3
48	7.3 ± 3.0	12.7 ± 4.2	17.4 ± 8.3	20.4 ± 3.6	24.7 ± 5.1	21.6 ± 5.3
72	4.7 ± 4.0	9.5 ± 2.4	12.5 ± 6.7	17.1 ± 3.6	22.6 ± 10.3	16.4 ± 2.8
96	3.5 ± 3.4	7.5 ± 2.0	11.3 ± 5.6	10.7 ± 4.0	17.2 ± 5.1	12.0 ± 2.9

at 2 h there being no significant difference between them. At 4 h adimolol 25 and 400 mg and propranolol 40 mg reduced supine heart rate. Adimolol 400 mg and propranolol 40 mg also reduced supine heart rate significantly compared to placebo at 8 h. In the standing

position the mean pre-treatment heart rate values did not differ significantly and no change occurred in heart rate after placebo. Adimolol 25 and 400 mg and propranolol 40 mg significantly reduced standing heart rate at 2, 4, 6 and 8 h, however the two doses of labetalol reduced

Table 2 Mean heart rate ($\pm$ s.d.) in the supine and standing positions and after exercise before (0) and 2, 4, 6 and 8 h after the oral administration of adimolol, 25 and 400 mg, labetalol 200 and 800 mg, propranolol 40 mg and placebo to five subjects

		0	2	Time (h) 4	6	8
<i>Supine</i>						
Placebo		78.0 $\pm$ 19.2	86.8 $\pm$ 20.2	81.6 $\pm$ 19.3	76.8 $\pm$ 16.7	77.6 $\pm$ 13.2
Adimolol 25 mg		66.6 $\pm$ 11.1	68.4 $\pm$ 13.6*	64.2 $\pm$ 8.5*	66.6 $\pm$ 7.99	69.2 $\pm$ 12.0
Adimolol 400 mg		64.2 $\pm$ 13.6*	66.4 $\pm$ 8.2*	57.2 $\pm$ 6.1*	65.2 $\pm$ 10.6	60.8 $\pm$ 4.7*
Labetalol 200 mg		70.8 $\pm$ 12.5	73.8 $\pm$ 10.8*	76.2 $\pm$ 10.8	83.6 $\pm$ 9.6	83.0 $\pm$ 10.8
Labetalol 800 mg		78.4 $\pm$ 16.7	66.6 $\pm$ 9.4*	70.8 $\pm$ 9.3	71.8 $\pm$ 7.2	69.0 $\pm$ 9.0
Propranolol 40 mg		71.6 $\pm$ 18.1	62.6 $\pm$ 13.4*	63.8 $\pm$ 11.4*	71.0 $\pm$ 13.3	63.4 $\pm$ 8.4*
Minimum significant difference = 11.9						
<i>Standing</i>						
Placebo		107.6 $\pm$ 17.3	118.8 $\pm$ 17.9	114.2 $\pm$ 20.2	118.2 $\pm$ 18.5	109.2 $\pm$ 23.9
Adimolol 25 mg		97.8 $\pm$ 11.0	90.2 $\pm$ 13.2*	91.0 $\pm$ 16.1*	89.2 $\pm$ 14.8*	91.8 $\pm$ 14.1*
Adimolol 400 mg		99.8 $\pm$ 7.0	82.4 $\pm$ 11.1*	75.4 $\pm$ 5.1*	80.0 $\pm$ 8.0*†	78.2 $\pm$ 7.9*
Labetalol 200 mg		108.8 $\pm$ 11.4	100.8 $\pm$ 16.4*	104.6 $\pm$ 9.6	112.8 $\pm$ 12.0	109.0 $\pm$ 10.0
Labetalol 800 mg		114.0 $\pm$ 13.9	95.8 $\pm$ 7.8*	102.6 $\pm$ 5.0	106.4 $\pm$ 8.3	105.2 $\pm$ 9.9
Propranolol 40 mg		112.6 $\pm$ 22.0	85.8 $\pm$ 10.1*	84.6 $\pm$ 10.9*	98.4 $\pm$ 23.0*	88.6 $\pm$ 13.1*
Minimum significant difference = 15.5						
<i>Exercise</i>						
Placebo		188.4 $\pm$ 20.5	190.6 $\pm$ 13.7	182.6 $\pm$ 19.9	186.8 $\pm$ 11.8	186.4 $\pm$ 7.6
Adimolol 25 mg		176.6 $\pm$ 15.7*	167.6 $\pm$ 10.8*	154.6 $\pm$ 14.1*	150.8 $\pm$ 13.6*†	147.6 $\pm$ 12.8*†
Adimolol 400 mg		175.8 $\pm$ 20.5*	129.4 $\pm$ 11.5*†	124.8 $\pm$ 5.4*†	126.4 $\pm$ 5.5*†	122.8 $\pm$ 8.3*†
Labetalol 200 mg		179.4 $\pm$ 12.2	156.0 $\pm$ 10.5*	170.0 $\pm$ 10.0*	173.8 $\pm$ 4.8*	178.0 $\pm$ 8.8
Labetalol 800 mg		187.6 $\pm$ 8.7	141.8 $\pm$ 8.8*	149.2 $\pm$ 7.0*	158.0 $\pm$ 7.6*	162.0 $\pm$ 8.7*
Propranolol 40 mg		185.4 $\pm$ 14.3	145.6 $\pm$ 9.2*	154.4 $\pm$ 9.1*	164.2 $\pm$ 7.1*	167.6 $\pm$ 7.3*
Minimum significant difference = 10.8						

* $P < 0.05$ compared with placebo

† $P < 0.05$ compared with propranolol

standing heart rate only at 2 h. The effect of adimolol 400 mg was significantly greater than propranolol 40 mg at 6 h.

Following exercise the mean pre-treatment tachycardia was not significantly different in the six groups except for adimolol 25 and 400 mg which were lower and it did not change in the placebo group during the study period. The exercise heart rate was significantly reduced at all times following adimolol 25 and 400 mg, labetalol 800 mg and propranolol 40 mg. Labetalol 200 mg gave significant reductions at 2, 4 and 6 h. Adimolol 400 mg was significantly more

effective than propranolol 40 mg at all times observed and adimolol 25 mg was more effective than propranolol 40 mg at 6 and 8 h. Labetalol 800 mg and propranolol were more effective than labetalol 200 mg and adimolol 25 mg at 2 h.

The effects of placebo, labetalol, adimolol and propranolol on systolic and diastolic blood pressure in the supine and standing positions are given in Table 3. In the supine position no significant differences were seen in pre-treatment values except for the supine diastolic pressure prior to taking adimolol 400 mg. Placebo had no significant effect on supine blood pressure.

Table 3 Mean systolic and diastolic pressure ($\pm$ s.d.) in the supine and standing positions before (0) and 2, 4, 6 and 8 h after the oral administration of adimolol, 25 and 400 mg, labetalol 200 and 800 mg, propranolol 40 mg and placebo to five subjects

	0	2	Time (h) 4	6	8
<i>Supine systolic (mm Hg)</i>					
Placebo	121.2 $\pm$ 4.6	120.0 $\pm$ 14.4	116.0 $\pm$ 5.1	113.2 $\pm$ 7.6	113.2 $\pm$ 9.9
Adimolol 25 mg	116.4 $\pm$ 11.1	111.4 $\pm$ 8.1	108.4 $\pm$ 6.5	108.4 $\pm$ 3.0	111.6 $\pm$ 7.4
Adimolol 400 mg	125.6 $\pm$ 11.3	107.6 $\pm$ 11.1*	106.8 $\pm$ 10.1	108.8 $\pm$ 10.7	108.4 $\pm$ 9.9
Labetalol 200 mg	115.6 $\pm$ 8.3	110.8 $\pm$ 9.4	110.0 $\pm$ 3.7	111.6 $\pm$ 9.8	114.4 $\pm$ 11.2
Labetalol 800 mg	118.8 $\pm$ 10.1	102.0 $\pm$ 5.1*	104.0 $\pm$ 7.9*	109.2 $\pm$ 6.6	114.4 $\pm$ 4.8
Propranolol 40 mg	119.2 $\pm$ 12.9	110.8 $\pm$ 7.6	105.6 $\pm$ 8.3*	108.8 $\pm$ 4.6	108.0 $\pm$ 4.2
Minimum significant difference = 10.2					
<i>Supine diastolic (mm Hg)</i>					
Placebo	68.4 $\pm$ 9.6	70.0 $\pm$ 16.3	69.6 $\pm$ 9.3	70.8 $\pm$ 4.4	72.0 $\pm$ 9.7
Adimolol 25 mg	69.2 $\pm$ 18.1	70.6 $\pm$ 7.9	65.2 $\pm$ 11.5	65.6 $\pm$ 12.9	65.2 $\pm$ 8.7
Adimolol 400 mg	84.4 $\pm$ 8.6*	74.0 $\pm$ 15.8	71.8 $\pm$ 9.0	72.0 $\pm$ 14.0	74.4 $\pm$ 11.6
Labetalol 200 mg	67.2 $\pm$ 14.3	54.8 $\pm$ 19.9*†	60.0 $\pm$ 14.6	56.4 $\pm$ 22.0*†	62.8 $\pm$ 14.4
Labetalol 800 mg	72.4 $\pm$ 11.3	64.4 $\pm$ 5.9	61.2 $\pm$ 11.9	58.8 $\pm$ 13.3*†	63.6 $\pm$ 18.5
Propranolol 40 mg	70.0 $\pm$ 6.8	70.4 $\pm$ 7.0	71.6 $\pm$ 5.5	72.0 $\pm$ 3.7	67.6 $\pm$ 4.6
Minimum significant difference = 12.3					
<i>Standing systolic (mm Hg)</i>					
Placebo	119.6 $\pm$ 11.1	128.0 $\pm$ 16.4	114.0 $\pm$ 15.7	110.4 $\pm$ 14.2	114.8 $\pm$ 14.0
Adimolol 25 mg	116.4 $\pm$ 7.4	115.2 $\pm$ 10.7*	114.0 $\pm$ 8.5	106.2 $\pm$ 5.1	107.6 $\pm$ 7.4
Adimolol 400 mg	119.2 $\pm$ 16.2	109.6 $\pm$ 7.3*	104.0 $\pm$ 11.0	103.2 $\pm$ 14.5	107.2 $\pm$ 13.1
Labetalol 200 mg	116.8 $\pm$ 7.0	98.8 $\pm$ 11.1*	106.8 $\pm$ 8.1	108.8 $\pm$ 8.4	109.6 $\pm$ 9.4
Labetalol 800 mg	122.0 $\pm$ 15.6	88.0 $\pm$ 12.3*†	89.6 $\pm$ 7.9*†	96.4 $\pm$ 8.2*	99.2 $\pm$ 6.1*
Propranolol 40 mg	116.0 $\pm$ 13.6	107.2 $\pm$ 11.5*	108.4 $\pm$ 12.3	106.0 $\pm$ 8.7	101.2 $\pm$ 6.4*
Minimum significant difference = 12.7					
<i>Standing diastolic (mm Hg)</i>					
Placebo	87.2 $\pm$ 10.1	84.0 $\pm$ 3.7	85.2 $\pm$ 7.8	85.2 $\pm$ 14.6	87.6 $\pm$ 9.3
Adimolol 25 mg	81.6 $\pm$ 10.3	89.2 $\pm$ 11.8	77.2 $\pm$ 4.6	79.8 $\pm$ 1.5	81.2 $\pm$ 6.7
Adimolol 400 mg	86.0 $\pm$ 9.7	80.4 $\pm$ 5.7	78.8 $\pm$ 9.1	76.4 $\pm$ 12.1	79.2 $\pm$ 11.2
Labetalol 200 mg	83.2 $\pm$ 7.6	67.2 $\pm$ 13.8*†	73.6 $\pm$ 9.1*	78.0 $\pm$ 13.0	79.2 $\pm$ 8.4
Labetalol 800 mg	86.4 $\pm$ 10.3	60.8 $\pm$ 9.3*†	65.6 $\pm$ 8.2*†	69.6 $\pm$ 7.1*	73.6 $\pm$ 8.9
Propranolol 40 mg	82.0 $\pm$ 6.5	86.4 $\pm$ 7.1	83.2 $\pm$ 9.9	79.2 $\pm$ 9.9	77.2 $\pm$ 6.3
Minimum significant difference = 11.2					

* $P < 0.05$ compared with placebo

† $P < 0.05$ compared with propranolol

Small reductions were seen in supine systolic pressure with all treatments which were significant with adimolol 400 mg, at 2 h, with labetalol 800 mg at 2 and 4 h, and after propranolol 40 mg at 4 h. Similarly small reductions occurred in supine diastolic pressure which were significant at 2 and 6 h after labetalol 200 mg, significantly more than the effect produced by both doses of adimolol and propranolol.

In the standing position no significant differences were seen in pre-treatment values of blood pressure nor following placebo. At 2 h all drugs significantly reduced standing systolic pressure and the effect of labetalol 800 mg was significantly greater than adimolol 400 mg. At 4 h only labetalol 800 mg significantly reduced standing systolic blood pressure. At 2 and 4 h both doses of labetalol reduced standing diastolic pressure and neither dose of adimolol had a significant effect.

Phenylephrine study

The α -adrenoceptor blocking activity of adimolol 400 mg, labetalol 400 mg and propranolol 40 mg was compared in the third study. The log dose-response curves for the effect of serial 4 min infusions of phenylephrine on systolic blood pressure 4 h after the oral administration of the drugs are shown in Figure 1. The slopes of the regression lines for the increase in systolic pressure produced by the three largest doses of phenylephrine administered after each treatment did not differ significantly from each other. The regression line obtained following labetalol 400 mg was significantly shifted to the right compared with the position of the regression lines following adimolol 400 mg and propranolol 40 mg, which did not differ significantly. The curves for diastolic pressure are similar.

The mean exercise heart rate following adimolol 400 mg was 110 ± 9.9 , significantly less than after labetalol 400 mg (134.2 ± 13.8) and propranolol 40 mg (130.7 ± 12.2).

None of the subjects, in the three studies, complained of adverse effects.

Discussion

β -adrenoceptor blocking drugs have been used in the treatment of hypertension for nearly 20 years and labetalol, which blocks both α - and β -adrenoceptors for 10 years. Unlike a combination of propranolol and phenoxybenzamine, labetalol, at low doses, does not produce significant postural or exercise hypotension (Richards & Prichard, 1979). However, at higher doses postural falls in blood pressure tend to occur

Figure 1 Effects of labetalol 400 mg (●), propranolol 40 mg (■) and adimolol 400 mg (○) on the increase in systolic blood pressure following infusions of phenylephrine. The concentration of phenylephrine was increased stepwise until systolic blood pressure after each treatment increased by 30 mm Hg. The results are expressed as the mean $\pm$ s.e. mean.

(Dargie *et al.*, 1976) and a drug with a higher ratio of β - to α -adrenoceptor blocking activity might be less likely to exhibit this effect and remain useful for the treatment of hypertension. Animal studies suggested that adimolol was a long acting drug with β - and weak α -adrenoceptor blocking activity (Hoefke *et al.*, 1984) and these studies were undertaken to assess its effects in man.

The dose ranging study demonstrated that the drug was a potent β -adrenoceptor blocking drug with a long duration of action though the maximum effect did not appear for 4 to 6 h after dosage. This might suggest that absorption is slow or that metabolites play a significant part in the observed effects. The maximum percentage reduction (29%) in exercise tachycardia was seen after 600 mg and this is similar to that observed (32%) 3 h after propranolol 160 mg orally (Balnave *et al.*, 1981).

The doses of adimolol and labetalol used in the second study were chosen to give β -adrenoceptor blockade, as assessed by an exercise tachycardia, of approximately the same magnitude as that produced by propranolol 40 mg. For each drug the larger dose produced a slightly greater effect and the smaller dose produced a slightly smaller effect than propranolol 40 mg on the exercise tachycardia recorded at 2 and 4 h. At these times the effects of adimolol and labetalol

on blood pressure were quite different and there was little difference between adimolol and propranolol. Both doses of labetalol significantly reduced standing systolic and diastolic blood pressure at 2 h. Adimolol 25 and 400 mg and propranolol 40 mg did not produce a significantly different effect from placebo on standing diastolic pressure. These results suggest absence of an α -adrenoceptor blocking effect with adimolol.

The standard method of assessing α -adrenoceptor blocking activity of a drug in man is to study the effect of the drug on the log dose response curve of phenylephrine on blood pressure (Sumner *et al.*, 1982). As the maximum β -adrenoceptor blockade of adimolol occurred at 4 h it was decided to compare the effect of adimolol 400 mg with labetalol 400 mg and propranolol 40 mg on the phenylephrine dose response curve at 4 h. Labetalol 400 mg produced a significant parallel shift of the dose

response curve to the right compared to propranolol 40 mg and adimolol which were not significantly different. The fact that the exercise heart rates after the phenylephrine infusions for labetalol 400 mg and propranolol 40 mg were significantly greater than after adimolol 400 mg would suggest that the study would have overestimated the α -adrenoceptor blocking activity of adimolol, if it had been present.

These studies indicate that adimolol is a potent, long acting β -adrenoceptor blocking drug, without noticeable side effects. At the doses used there is no evidence from these studies for α -adrenoceptor blocking activity in man.

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