babies whose mothers had no obstetric complications. In the preterm group three-quarters of the babies were born alive and presented problems in the neonatal period, requiring specialised paediatric care. In the growth-retarded group the problem was that of intrauterine death, and if such babies are to survive the obstetrician must be able to detect that the fetus is compromised and deliver it before fetal death occurs. This is also true for babies of "normal" birth weight, 82 such infants dying before delivery. Postmaturity is not now an important cause of fetal death; the problem is that of unexplained intrauterine death before term. Such babies were not growth-retarded.

From the retrospective analysis we could not discern whether many of the deaths caused by trauma were preventable. Traumatic breech deliveries would, of course, be prevented by earlier recourse to caesarean section. This operation is not without complications, however, and undiagnosed breech presentations will continue to occur. In the antepartum haemorrhage group the fetal heart was not heard on admission in 57% of cases in which death occurred before delivery. We cannot see how these deaths could have been averted. Earlier antenatal attendance, so that screening for central nervous system abnormality could be carried out and the patient offered termination of pregnancy, would reduce the number of abnormal births, although clearly would not prevent the occurrence of the abnormality. In the pre-eclamptic group 39 babies died before delivery and weighed 1500 g or more. The mothers of these babies did not have fulminating pre-eclampsia, and the diagnosis that the fetus was stressed had not been made. In all but a few cases of maternal disease the condition had been recognised by the obstetrician and appropriate care given. Hence these deaths could probably not have been prevented. The problems of rhesus incompatibility are now only a small part of obstetric and paediatric practice, partly because of the introduction of anti-D immunoglobulin and partly as a result of changes in family size.

We believe that analysing perinatal deaths in the manner described is of value in illustrating the main causes of perinatal mortality and of directing attention to issues of contemporary importance. Hence we are now studying perinatal deaths that occurred in Scotland during 1979 to see whether yearly assessment by regional assessors (obstetricians and paediatricians) is a feasible and practical way of monitoring trends in perinatal mortality that, in turn, will lead to a reduction in preventable deaths.

We are grateful for the help we received from all concerned in perinatal care throughout Scotland. We acknowledge the help we received from Sir John Brotherston and the Scottish Home and Health Department in launching the survey. Frances Dunn is supported by a grant from the Scottish Home and Health Department.

Copies of the survey report may be obtained from Frances Dunn, price £1.20 (including postage).

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Essential hypertension: effect of an oral inhibitor of angiotensin-converting enzyme

GRAHAM A MacGREGOR, N D MARKANDU, J E ROULSTON, J C JONES

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Summary and conclusions

Captopril, a specific oral inhibitor of angiotensin-converting enzyme, was given to 18 unselected patients with moderate essential hypertension. Mean blood pressure fell by 14·5% at the maximum dose given, and this fall was significantly correlated with the initial plasma renin activity. The main fall in blood pressure occurred two hours after the first dose of captopril.

These results suggest that captopril effectively lowers blood pressure in patients with essential hypertension and that the renin-angiotensin aldosterone system may maintain blood pressure in essential hypertension. This does not necessarily imply that the renin-angiotensin system is the cause of the high blood pressure.

Department of Medicine, Charing Cross Hospital Medical School, London W6 8RF
GRAHAM A MacGREGOR, MRCP, senior lecturer and honorary consultant physician
N D MARKANDU, SRN, research sister
J E ROULSTON, MA, biochemist
J C JONES, BSC, biochemist

Introduction

The importance of the renin-angiotensin aldosterone system in maintaining blood pressure in essential hypertension is disputed.[1 2] Infusions of competitive inhibitors of angiotensin II, such as saralasin, cause no change in blood pressure in most patients with essential hypertension receiving their normal sodium intake.[3] Saralasin, however, is a potent agonist,[4-6] and when given as a short-term infusion blocks only the immediate effects of circulating angiotensin II. Both of these factors will tend to underestimate the role of the renin-angiotensin aldosterone system in maintaining blood pressure. The converting-enzyme inhibitor teprotide, which blocks the enzyme that converts angiotensin I to angiotensin II, has, when given as a single injection, a slight lowering effect on blood pressure in some patients with essential hypertension studied while receiving their normal diet.[3] More recently an oral drug, captopril, that specifically inhibits the angiotensin-converting enzyme has been developed.[7 8] With this drug long-term inhibition of the formation of angiotensin II is now possible.

Preliminary studies with captopril have shown that it is a highly effective antihypertensive agent in patients with renovascular hypertension and in a few patients with essential hypertension who are resistant to other treatment.[9 10]

We report here a study of the effect of captopril on blood pressure and the renin-angiotensin aldosterone system in 18 unselected patients with moderate essential hypertension.

Patients and methods

Eighteen patients (10 men, eight women) with uncomplicated essential hypertension were included in the trial. All had been referred by local general practitioners and were included in the study only if, after an initial observation period of one to two months when they received no treatment, their sitting diastolic pressure was between 100 and 120 mm Hg. The mean age of the patients was 52 years (range 41-65). Patients were excluded if there was evidence of renal failure, ischaemic heart disease, or cerebrovascular disease and if they were taking oral contraceptives or any other drug. Informed consent was obtained from each patient.

PROCEDURE

After the initial assessment period of one to two months all 18 patients were observed weekly for a further four weeks while receiving no treatment. Twelve patients were then treated with a matching placebo three times a day for one month and seen every week. All 18 patients then started to take captopril, 25 mg three times a day. Observations were made two hours after the first 25 mg dose in 15 patients. After one week of captopril the dose was increased to 50 mg thrice daily during the second week, 100 mg thrice daily during the third week, and 150 mg thrice daily during the fourth week. If the supine diastolic pressure was less than 85 mm Hg the dose was not increased but was kept at the same amount as the previous week. Two patients had diastolic pressures below 85 mm Hg during the third week of treatment while taking captopril 100 mg thrice daily; the next week, however, the diastolic pressure was greater than 85 mm Hg, so that the dose was increased to 150 mg thrice daily in the fifth week of treatment. For the analysis of treatment the blood pressure in this fifth week in these two patients was taken as though it was the pressure in the fourth week. In the 16 other patients the diastolic pressure did not fall below 85 mm Hg and the dose of captopril was increased each week.

All patients were seen in the blood pressure clinic in the same room, at the same time of day, on the same day of the week for weekly visits, and by the same nurse. Patients were instructed to take their tablets two to three hours before a visit, and one hour before or two hours after a meal. All blood pressures were measured by nurses using semi-automatic ultrasound sphygnomanometers[11] (Arteriosonde 1217 with recorder). The mean values of five readings made at one- to two-minute intervals with the patient supine, sitting, and standing were taken as the blood pressure in that position. Blood pressure was also measured one minute after a standard period of exercise on a treadmill. Pulse rate was measured on a Cambridge 3048 pulse monitor, with ECG display and recording. Weight was measured at each visit.

Blood samples for plasma renin activity and plasma aldosterone, urea, electrolyte, and creatinine concentrations were taken before active treatment, two hours after the first dose, and at weekly intervals during treatment. Routine haematological and biochemical examinations were done before active treatment and during the second and fourth weeks of treatment. Blood was taken without stasis after the patient had been sitting upright for five minutes. Plasma renin activity was measured by radioimmunoassay.[12] Plasma aldosterone was measured by radioimmunoassay, using a modification of the method described by James *et al.*[13] The normal ranges for plasma renin activity and plasma aldosterone in subjects sitting upright between 10 00 am and noon and with a sodium intake of 100-200 mmol (mEq)/day are 0·5-2·5 ng/ml/h and 100-600 pmol/l (3·6-21·6 ng/100 ml) respectively. All patients ate their normal diet. Twenty-four-hour urine collections for measurement of sodium excretion were done before active treatment.

Statistical analysis was performed using the University of London computer and North-western University's statistical package for social sciences. Mean blood pressure was calculated as diastolic pressure + (1/3 × pulse pressure). Results are reported as means ± SE of means.

Results

The mean supine blood pressure in the 18 patients before the start of the trial but after the initial assessment period of one to two months was 178±4·5 mm Hg systolic and 113±2·9 mm Hg diastolic. Four weeks' observation in six of the patients and four weeks' treatment with placebo in the 12 others made no significant difference to the mean blood pressures. Mean supine blood pressures for the six patients before and after four weeks of weekly observation were 178±5·2 mm Hg systolic and 115±5·7 mm Hg diastolic (before) and 178±5·3 mm Hg systolic and 114±4·3 mm Hg diastolic (after). Mean supine blood pressure for the 12 other patients before and after four weeks' placebo were 175±5·8 mm Hg systolic and 107±2·7 mm Hg diastolic (before) and 177±6·2 mm Hg systolic and 110±2·5 mm Hg diastolic (after).

Two hours after the first dose of captopril (25 mg) the mean supine blood pressure fell significantly. Blood pressures before and two hours after captopril were 178±5·3 mm Hg systolic and 112±2·6 mm Hg diastolic (before) and 158±5·4 mm Hg systolic and 103±3·2 mm Hg diastolic (after) ($n=15$, $P<0.001$). The blood pressure two hours after a single dose of 25 mg of captopril in these 15 patients was not significantly different from the average supine blood pressure at one week, when the dose was 25 mg thrice daily, or during the second and third weeks of treatment at 50 mg and 100 mg thrice daily respectively. During the fourth week of treatment (150 mg thrice daily) the mean supine blood pressure was significantly lower than that two hours after the first dose of captopril, being 150±5·4 mm Hg systolic and 97±3·6 mm Hg diastolic compared with 158±5·1 mm Hg systolic and 103±3·2 mm Hg diastolic ($n=15$, $P<0.01$). Figure 1 shows the average blood pressures supine and standing for all 18 patients and for the 15 after the single dose. Changes in sitting and after-exercise blood pressures showed no significant difference from the changes in supine and standing pressures. The percentage fall in mean blood pressure at the maximum dose of captopril as compared with control values was 14·5%.

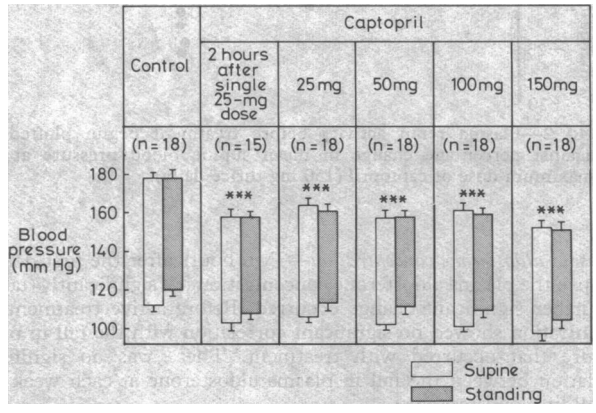

FIG 1—Mean (± SE of mean) systolic and diastolic blood pressures, supine and standing, before and during treatment with increasing doses of captopril (each dose given thrice daily for one week).

Significance of difference: *** $P<0.001$ compared with control values.

Difference between blood pressures with increasing doses of captopril and values two hours after single 25 mg dose significant only for 150 mg dose ($n=15$, $P<0.01$).

Plasma renin activity—Plasma renin activity before active treatment was within the normal range in 13 patients and below the normal range in five. The mean 24-hour urinary sodium excretion in the 18 patients before receiving captopril was 150·6 mmol (mEq) (range 90-198 mmol). Plasma renin activity rose two hours after the first dose of captopril and did not change significantly from this value during further treatment in these 15 patients. The table shows the mean values for all 18 patients. The fall in blood pressure that occurred with captopril at the fourth week of treatment was significantly correlated with the logarithm of the pretreatment plasma renin activity ($r=0.59$, $P<0.005$, $n=18$). This correlation was significant for the falls in supine, upright, sitting, after-exercise, systolic, and diastolic pressures, as well as the mean blood pressure two hours after the first dose and on the second, third, and fourth weeks, but there was no significant correlation for the first week of treatment. The

Mean (± SE of mean) results of biochemical tests, weight, and pulse rate before and during treatment with captopril at increasing doses

	Control (n = 18)	Captopril treatment: 2 hours after single 25-mg dose (n = 15)	25 mg† (n = 18)	50 mg† (n = 18)	100 mg† (n = 18)	150 mg† (n = 18)
Plasma renin activity (ng/ml/h)	0·97 ± 0·12	1·83 ± 0·34**	2·20 ± 0·36***	2·76 ± 0·63**	2·79 ± 0·57**	3·02 ± 1·36*
Plasma aldosterone (pmol/l) ..	303 ± 31	201 ± 34***	225 ± 30**	227 ± 28**	198 ± 13**	207 ± 20**
Plasma potassium (mmol/l) ..	3·91 ± 0·07	3·91 ± 0·12	4·07 ± 0·11	4·19 ± 0·09**	4·17 ± 0·08**	4·10 ± 0·077*
Weight (kg)	76·4 ± 3·0	76·3 ± 3·0	75·8 ± 3·0*	75·5 ± 3·0**	75·1 ± 3·1***	75·1 ± 3·0***
Pulse rate, supine (beats/min) ..	80 ± 2·5	79 ± 2·7	76 ± 2·1	75 ± 2·4	78 ± 3·6	75 ± 2·3*

†Dose given thrice daily for one week.
Significance of difference: *P < 0·05; **P < 0·01; ***P < 0·001 compared with control values.
For plasma renin activity and aldosterone concentration values with increasing doses of captopril were not significantly different from values two hours after a single 25-mg dose.
Conversion: SI to traditional units—Plasma aldosterone: 1 pmol/l ≈ 36 pg/100 ml. Plasma potassium: 1 mmol/l = 1 mEq/l.

percentage fall in blood pressure was also significantly correlated against initial plasma renin activity (fig 2). When patients with abnormally low plasma renin activity were excluded from this analysis the correlation was not significant. There was no significant correlation between the fall in blood pressure that occurred with captopril at each week and the rise in plasma renin activity.

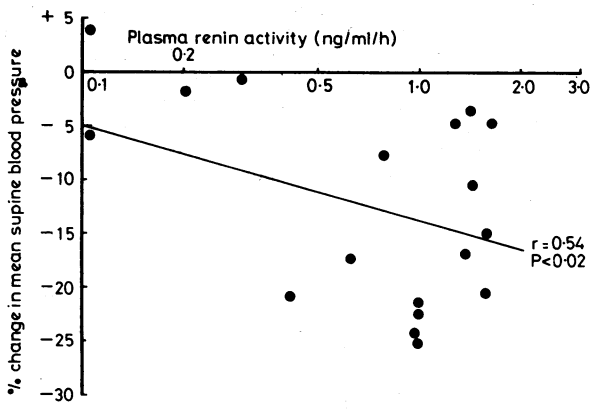

FIG 2—Plasma renin activity before treatment begun plotted against percentage change in mean supine blood pressure at maximum dose of captopril (150 mg thrice daily).

Plasma aldosterone concentration—Two hours after the first dose of captopril the plasma aldosterone concentration fell significantly (table). No further significant change occurred. Before active treatment the concentration showed no significant correlation with the fall in blood pressure that occurred with treatment. There was no significant correlation between the fall in plasma aldosterone at each week and the fall in blood pressure.

Plasma potassium concentration—Plasma potassium concentrations rose significantly during the second week of treatment (table). The mean rise in plasma potassium when the captopril dose was 150 mg thrice daily compared with the control value was 0·19 mmol (mEq)/l (P < 0·05). The correlation between this rise and the fall in plasma aldosterone concentration just failed to reach significance (r = 0·41, P < 0·06).

Weight—The mean weight had fallen significantly by the first week of treatment, and this weight loss increased significantly at the third week; the mean weights in the third and fourth weeks of treatment were not significantly different (table).

Pulse rate—The mean pulse rate showed no significant change except during the fourth week, when it fell significantly (table).

Other blood tests—Plasma sodium concentrations did not change significantly, but a significant fall in plasma bicarbonate concentration had occurred by the first week of treatment, which remained significant to the fourth week. Plasma bicarbonate concentrations before and during the fourth week of captopril (150 mg thrice daily) were 25·1 ± 0·42 and 24·2 ± 0·29 mmol (mEq)/l respectively (P < 0·02). Plasma chloride concentrations rose by about 1·5 mmol (mEq)/l, but at no stage was this rise significant. Blood urea, plasma creatinine, and urate concentrations showed no significant changes at each week of treatment. Blood sugar, alanine transferase, bilirubin, alkaline phosphatase, calcium, phosphate, cholesterol, and triglyceride concentrations showed no significant changes at the second and fourth weeks of treatment as compared with control values. The mean haemoglobin concentration showed a significant fall by the fourth week of captopril, from 14·6 ± 0·28 to 13·81 ± 0·29 g/dl (P < 0·001). This was associated with a significant decrease in packed cell volume. White cell count did not change significantly. No patient developed or had proteinuria.

Side effects—Captopril was well tolerated, and no patient had to be withdrawn from the trial because of side effects. Two patients complained of headaches on the second, third, and fourth weeks of treatment. One patient had a transient urticarial rash on both hands and arms two weeks after starting captopril. Captopril was continued and the rash disappeared after three days and did not return. One patient lost taste on the fourth week of treatment, necessitating stopping captopril during the fifth week of treatment. Taste was recovered within two weeks of stopping captopril. No other important side effects were recorded, and most patients volunteered spontaneously that they felt well while receiving captopril.

Discussion

Our results show that captopril is an effective antihypertensive drug in unselected patients with moderate essential hypertension. The 14·5% fall in mean blood pressure that occurred is greater than falls we have seen in trials in similar patients using either a beta-blocker or a diuretic alone. The main fall in blood pressure with captopril occurred two hours after the first dose of 25 mg. Blood pressure at the end of the first, second, and third weeks of treatment was not significantly different from this, although a greater fall occurred at the end of the fourth week of treatment, at 150 mg three times a day. This may suggest that increasing the dose of captopril does not greatly increase its blood-pressure lowering effect. In this trial we did not study the duration of action of captopril, but deliberately chose to see patients two to three hours after a given dose. Increasing the dose may well increase the duration of action but not the blood-pressure lowering effect at two to three hours after a single dose, when it might be expected that this effect would be greatest. These results suggest that a randomised trial of different doses of captopril is necessary, as well as an investigation into the duration of action of captopril when given long term.

The mechanism whereby captopril lowers blood pressure is not clear. It is a potent inhibitor of converting enzyme as judged by the rise in plasma renin activity and the fall in plasma aldosterone that occurred. Other workers have shown this[9,10] and have also shown the inhibition of converting enzyme more directly, by measuring converting enzyme[9] and the fall in angiotensin II that occurs.[14,15] The angiotensin-converting enzyme is also partially responsible for the breakdown of bradykinin. Inhibition of this enzyme could lead to an accumulation of bradykinin, a potential vasodilator, and this rise has been claimed to be a mechanism for the fall in blood pressure that occurs.[1,16] Patients do not, however, have symptoms of raised bradykinin concentrations, and more recent work has shown that there does not appear to be an increase in plasma bradykinin with captopril[17] or with the parenteral converting-enzyme inhibitor.[18] Amery *et al* have shown a direct correlation

between the falls in circulating angiotensin II and blood pressure with captopril.[14] This has also been shown in salt-depleted dogs.[19] The fall in aldosterone that occurs with captopril may also have a blood-pressure lowering effect—firstly, through a postulated but still unproved effect on the sensitivity or tone of the arterioles, and, secondly, through the loss of sodium and water.

Our study shows a significant correlation between the initial plasma renin activity before treatment and the subsequent fall in blood pressure. When patients who had a very low plasma renin activity were excluded, however, the correlation was not significant. This is hardly surprising, as the range of activity was then small. A significant amount of weight was lost, which appeared to increase as the dose of captopril was increased. The weight loss was similar to that seen in similar patients treated with thiazide diuretics: it could be due to sodium and water loss caused by the fall in aldosterone, the fall in angiotensin II, or a rise in another vasodilating substance, perhaps bradykinin in the kidney itself. If this weight loss was due to sodium and water loss, however, it is surprising that the blood pressure did not fall to a greater extent in the second and third weeks of treatment as compared with the two-hour blood pressure after the first dose, when no sodium and water loss could have occurred as there was no change in weight. The fact that the main fall in blood pressure occurred within two hours of the first dose of captopril certainly suggests an immediate action of captopril on blood pressure, and the evidence so far would suggest that it is probably due in part to the immediate fall in angiotensin II that occurs and a decrease in the direct vasoconstrictor effect of angiotensin II.

If the renin-angiotensin aldosterone system is maintaining blood pressure in patients with essential hypertension, either by a direct vasoconstrictor action of angiotensin II or by an effect of aldosterone either directly on arteries or on sodium and water balance, it does not necessarily imply that this is the mechanism of the raised blood pressure. Studies that we have performed in normal subjects receiving a normal diet show that captopril reduces blood pressure to the same extent as it did in these patients with high blood pressure, suggesting that whatever the mechanism of action of captopril it has the same effect on blood pressure in percentage terms in normotensive and hypertensive subjects.[20] If the mechanism of action of captopril is through the renin-angiotensin aldosterone system, this system does not appear to maintain blood pressure to a greater extent in hypertensive than normotensive subjects for a given plasma renin activity.

Captopril in this short-term trial was well tolerated by the patients. Rashes, a reported side effect, were not evident except transiently in one patient. The loss of taste in one patient may be a more important side effect. Out of a total of 47 patients whom we have now treated with captopril, six have developed loss of taste. In four of these six the loss was severe: the captopril had to be stopped and there was appreciable weight loss. The taste recovered within three to four weeks of stopping captopril. The loss of taste with captopril might be related to zinc, but in preliminary studies that we have done in collaboration with the trace-metals unit at the Hospital for Sick Children, Great Ormond Street, we have been unable to show any change in plasma zinc or copper with captopril. The fall in haemoglobin concentration and packed cell volume that occurred with captopril is difficult to interpret in view of the fact that about 450 ml of blood was taken during the trial.

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ONE HUNDRED YEARS AGO We have received the following notes from a correspondent with the First Division of the Peshawur Valley Field Force, dated Safed Sung, near Gundamuck, May 22nd, 1879. The troops are still suffering a good deal from fever and diarrhœa; the former of a remittent type, especially severe in young soldiers, and doubtless induced by malarial poison. Expeditions are frequently made by brigades into the neighbouring hills (Safed Rho), to an elevation of 8000 or 9000 feet, from which the men derive much benefit and pleasure, freeing them from the monotony of camp-life, where the dust is sometimes intolerable, and giving them advantage of shade under the magnificent forest-trees with which the lower ranges of the Safed Rho are thickly covered; the higher ranges having still a thick coating of snow. It is contemplated to send a portion of the division to occupy some of these heights, should the troops remain at Safed Sung during the summer. The divisional field-hospital system has done good work and held its ground in this division since the commencement of the campaign, notwithstanding the opposition and want of sympathy shown to it by some military authorities. Each regiment has a medical officer attached to it, who administers to the immediate wants of the men, and daily selects certain sick to be sent to the field-hospital, where the organisation is now so far complete that everything works smoothly. There is an experienced staff of seven or eight medical officers with the field-hospital, some of whom have duties detailed of a perfectly non-professional character, such as the supervision of camp-equipage, transport-animals, building of lavatories and latrines, etc, but which have been undertaken with an activity and zeal highly creditable to the officers of the Army Medical Department. The building of grass huts for the sick in the field-hospital continues. Three have already been completed by the Madras and Bengal Sappers and Miners, with the aid of those uncommonly handy little fellows the 4th Ghoorkas. These huts afford great relief, by their comparative coolness, to the fever-stricken cases. Nor is loyalty forgotten on the heights above Gundamuck, as we observe one tent bears the title in large letters of the "Victoria Ward"; another, the "Alexandra"; and the third, the "Alice." Bedside tables have been extemporised out of old tea-chests supplied by the commissariat, and tumblers from bottles cut in two. Though cholera has been reported at Ali Musjid, Lundi Rhotal, and Jellalabad, fortunately no cases have as yet occurred among the advanced troops. The medical authorities have, however, made all necessary arrangements for its approach. The nights are becoming warm. The thermometer during the day in the tents is 97° Fahr. Snow is still brought in large quantities from the Safed Rho, and is one of the greatest comforts to the sick for cooling other drinks. The Director of the Medical Department of Yakoob Khan's Army is, we are informed, in camp with him at Gundamuck. It is to be hoped he may be induced to visit our British and native hospitals. (*British Medical Journal*, 1879.)