[5] Hackett TP, Cassem NH, Raker JW. Patient delay in cancer. *N Engl J Med* 1973;**289**:14-20.
[6] Gerson LW, Skipper JK. The influence of social factors on expectations of pain associated with osteoarthritis. *Scand J Rheumatol* 1975;**4**: 139-43.
[7] Parbrook GD, Steel DF, Dalrymple DG. Factors predisposing to post-operative pain and pulmonary complications. A study of male patients undergoing elective gastric surgery. *Br J Anaesth* 1973;**45**:21-33.
[8] Liebman WM. Recurrent abdominal pain in children. *Clin Pediatr (Phila)* 1978;**17**:149-53.
[9] Huskisson EC. Measurement of pain. *Lancet* 1974;ii:1127-31.
[10] Office of Population Censuses and Surveys. *Occupational mortality: the Registrar General's decennial supplement for England and Wales.* London: HMSO, 1970-72, 1978.
[11] *Inequalities in health: report of a research working group.* London: DHSS, 1980.
[12] Rook JC, Pesch RN, Keeler EC. Chronic pain and the questionable use of the Minnesota Multiphasic Personality Inventory. *Arch Phys Med Rehabil* 1981;**62**:373-6.
[13] Kremer EF, Hampton-Atkinson J, Ignelzi RJ. Pain management: the affective dimensional measure of the McGill Pain Questionnaire with a cancer population. *Pain* 1982;**12**:153-63.
[14] Waitzkin H, Stoeckle JD. The communication of information about illness. *Adv Psychosom Med* 1972;**8**:180-215.
[15] Hackett TP, Cassem NH. White-collar and blue-collar responses to heart attack. *J Psychosom Res* 1975;**20**:85-95.
[16] Croog SH, Levine S. Social status and subjective perceptions of 250 men after myocardial infarction. *Public Health Rep* 1969;**84**:989-97.
[17] Merskey H, Spear FG. *Pain: psychological and psychiatric aspects.* London: Baillière Tindall and Cassell, 1967.
[18] Melzack R, Torgerson WS. On the language of pain. *Anesthesiology* 1971;**34**:50-9.
[19] Sriwatanakul K, Kelvie W, Lasagna L. The quantification of pain: an analysis of words used to describe pain and analgesia in clinical trials. *Clin Pharmacol Ther* 1982;**32**:143-8.
[20] Blau JN. How to take a history of head or facial pain. *Br Med J* 1982; **285**:1249-51.
[21] Gould GM, Pyles W. *Anomalies and curiosities of medicine.* New York: Saunders, 1897.

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# Split renal function after captopril in unilateral renal artery stenosis

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## Abstract

**The renal extraction ratios of $^{131}$I-sodium iodohippurate ($^{131}$I-Hippuran) and $^{125}$I-thalamate were greatly reduced on the affected side by 50 mg captopril in seven out of 14 patients with unilateral renal artery stenosis. With long term captopril 150 mg daily the uptake of $^{99m}$Tc-diethylenetriaminepenta-acetic acid by the affected kidney, which was determined by scintillation camera renography, became almost zero in these seven patients, indicating severe reduction of the glomerular filtration rate. Function of the affected kidney returned on discontinuing treatment. The reduced extraction of sodium iodohippurate probably reflected a shortened plasma transit time through the kidney due to intrarenal vasodilatation. The reduced extraction of thalamate reflected a low filtration fraction, suggesting that the vasodilatation was, at least in part, at the level of the postglomerular arterioles. Captopril had little effect on the contralateral kidney and on the kidneys of 17 patients with essential hypertension, and serum creatinine concentrations showed minor changes.**

**Radioisotope renography should be performed after beginning captopril treatment in patients with renal artery stenosis. This is also recommended for patients given captopril as a third line drug when renal artery stenosis has not been excluded. Hypertension in these patients is often severe and difficult to control. Renal artery disease is not rare in this difficult group and finding seriously impaired renal function on one side during captopril treatment may be diagnostic.**

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## Introduction

Captopril is now widely used for severe hypertension, including that associated with renal artery stenosis.[1-4] Renal failure, however, may occur in patients receiving captopril who have bilateral renal artery stenosis or a stenosis affecting a solitary functioning kidney.[5-10] Increase in systemic arterial pressure, dilatation of preglomerular arterioles, postglomerular vasoconstriction, and possibly other mechanisms may help to maintain glomerular filtration when renal perfusion is compromised by artery stenosis.[11,12] Some of these mechanisms depend, at least in part, on an intact renin-angiotensin system. Converting enzyme inhibition, by interfering with angiotensin II formation, has therefore the potential to disturb the fine balance between pressure and flow required for optimal regulation of glomerular filtration in renal artery disease. In unilateral disease such an effect may easily go unnoticed because of the functional reserve of the opposite kidney.

We report on the effects of captopril on split renal function in these patients.

## Patients and methods

Thirty one hypertensive patients were selected from a larger series of consecutive patients because they were shown to have unilateral renal artery stenosis on renal arteriography (n = 14; table I) or because their renal arteries were found to be normal on both sides (n = 17). The patients were admitted to this hospital for a diagnostic work up because their hypertension was difficult to control; they remained hypertensive despite combined treatment with high doses of diuretics, β blockers, hydralazine, and in some cases also methyldopa. Results of urine analysis, serum electrolyte,

urea, and creatinine concentrations, and urinary excretion of vanillylmandelic acid were normal. Medication had been stopped for at least two weeks before renal function studies and renal vein catheterisation. The renal arteriogram was made after renal vein sampling in the same session.

$^{131}$I-Sodium iodohippurate ($^{131}$I-Hippuran) and $^{125}$I-thalamate were administered by constant infusion into an arm vein. After reaching the steady state blood samples were taken simultaneously from the abdominal aorta and the renal vein. Samples from the same sites were used for renin measurements. The extraction ratio of $^{131}$I-sodium iodohippurate ($E_H$) and of $^{125}$I-thalamate ($E_T$) and the aortic and renal vein plasma renin values were measured 10-15 minutes before captopril and 30-45 minutes after 50 mg of this drug. Blood samples were also taken at 15 minute intervals from a peripheral vein for estimating total renal clearance of sodium iodohippurate and thalamate.[13 14] All blood samples were centrifuged immediately and radioactivity measured in plasma. Single-kidney extraction ratio (extraction efficiency) was calculated as $(A-V)/A \times 100\%$, where A = activity in abdominal aorta and V = activity in renal vein. The clearance of sodium iodohippurate was taken as a measure of total effective renal plasma flow, and the clearance of thalamate was taken as a measure of total glomerular filtration rate.

The single kidney uptake of $^{99m}$Tc-diethylenetriaminepenta-acetic acid ($^{99m}$Tc-DTPA) was determined by scintillation camera renography.[15] Approximately 5-10 mCi $^{99m}$Tc-DTPA was injected intravenously. Lightpen "regions of interest" corresponding to the left and right kidneys were traced on the display screen using the three minute summation image. Time activity curves of each kidney region were displayed. Counting rates from the kidney areas were corrected for background activity using a region of interest between the kidneys. Single kidney function was estimated from the radioactivity over the kidney regions 60-120 seconds after injection and expressed as activity ratio—that is, right/(right + left). This ratio is a measure of the single kidney's contribution to total glomerular filtration rate.[16] The kidney scans were made before treatment and after three to five weeks of captopril 150 mg daily.

The concentration of active renin in plasma was measured by radioimmunoassay.[17] Blood pressure was measured intra-arterially in the acute study during renal vein catheterisation and indirectly with the London School of Hygiene sphygmomanometer in the long term study.

Grouped data are presented as means (SEM in parentheses), and differences were analysed for statistical significance by Student's *t* tests for paired and unpaired data.

## Results

Values of $E_H$ and $E_T$ were significantly decreased after captopril on both sides both in patients with unilateral renal artery stenosis and in essential hypertension (table II; fig 1). The effects of captopril on kidneys with a stenotic artery were much greater than on kidneys with normal arteries. The renal extraction ratio of a substance equals its renal clearance divided by the renal plasma flow. Thus $E_T$ = clearance of thalamate/true renal plasma flow, or glomerular filtration rate/true renal plasma flow—that is, filtration fraction. Our results therefore indicate that the single kidney filtration fraction was lowered by captopril, particularly when the kidney was affected by renal artery stenosis.

Since the clearance of sodium iodohippurate did not change after captopril (table II; fig 2) and $E_H$ = clearance of sodium iodohippurate/true renal plasma flow, the observed reduction of $E_H$ after captopril implies that true renal plasma flow and therefore the total renal blood flow was increased.

As shown in figure 3, $^{99m}$Tc-DTPA uptake by the affected kidney became almost zero after captopril in seven patients with unilateral renal artery stenosis (group 1) and was essentially unchanged in the remaining seven patients with renal artery stenosis (group 2). It was also unchanged in the patients with essential hypertension. Reductions in $E_H$ and $E_T$ after the first dose of 50 mg captopril were greater in group 1 than in group 2 (table II). Serum creatinine concentration rose significantly during long term captopril in group 1 but not in group 2 (table III). Neither the changes in blood pressure nor the pressure levels that were reached after captopril were, however, different in the two groups. None of the patients developed troublesome proteinuria.

The loss of renal function after captopril in group 1 appeared not to be due to irreversible parenchymal damage. In four patients DTPA uptake was restored one to two weeks after captopril had been stopped (figure 4 gives an example). By that time the plasma creatinine concentration had also returned to its original value. The

TABLE I—*Clinical data on patients with unilateral renal artery stenosis*

| Case No | Age (years) | Sex | Cause of renal artery stenosis | Plasma renin mU/l† | Renal vein to artery renin ratio | |
|---|---|---|---|---|---|---|
| | | | | | Affected kidney | Contralateral kidney |
| | | | *Group 1* | | | |
| 1 | 47 | M | Atherosclerosis | 233 | 5·97 | 0·92 |
| 2 | 57 | M | Atherosclerosis | 61 | 3·29 | 1·02 |
| 3 | 56 | M | Atherosclerosis | 37 | 2·42 | 0·94 |
| 4 | 64 | M | Atherosclerosis | 38 | 1·58 | 1·12 |
| 5 | 47 | M | Atherosclerosis | 37 | 1·71 | 1·17 |
| 6 | 31 | F | Fibromuscular hyperplasia | 23 | 2·65 | 0·88 |
| 7 | 57 | M | Atherosclerosis | 480 | 1·39 | 0·91 |
| | | | *Group 2* | | | |
| 8 | 68 | F | Atherosclerosis | 178 | 5·81 | 0·89 |
| 9 | 57 | M | Atherosclerosis | 353 | 2·09 | 0·81 |
| 10 | 58 | M | Atherosclerosis | 75 | 3·08 | 1·03 |
| 11 | 65 | M | Atherosclerosis | 208 | 2·20 | 1·40 |
| 12 | 25 | F | Fibromuscular hyperplasia | 34 | 1·53 | 1·04 |
| 13 | 68 | M | Atherosclerosis | 40 | 1·36 | 1·55 |
| 14 | 50 | F | Atherosclerosis | 42 | 2·17 | 0·96 |

Patients separated into two groups based on effect of captopril on uptake of $^{99m}$Tc-diethylenetriaminepenta-acetic acid by affected kidney (see text).
†Normal range 5-45 mU/l.

TABLE II—*Acute effects of captopril in patients with unilateral renal artery stenosis (group 1 v group 2). Values are means (SEM in parentheses)*

| | Group 1 (cases 1-7) | | Group 2 (cases 8-14) | | p Values for differences between groups 1 and 2 | |
|---|---|---|---|---|---|---|
| | Before captopril | After captopril | Before captopril | After captopril | Before captopril | After captopril |
| Mean arterial pressure (mm Hg) | 139 (5) | 114 (4)*** | 137 (10) | 110 (10)** | NS | NS |
| Total effective renal plasma flow (ml/min) | 333 (35) | 343 (42) | 320 (46) | 328 (48) | NS | NS |
| Total glomerular filtration rate (ml/min) | 95 (6) | 82 (7)** | 87 (8) | 81 (7)* | NS | NS |
| Single-kidney extraction ratio of sodium iodohippurate (%) — Affected kidney | 60 (7) | 29 (7)*** | 64 (5) | 53 (9)* | NS | <0·001 |
| Single-kidney extraction ratio of sodium iodohippurate (%) — Contralateral kidney | 75 (3) | 71 (3) | 74 (3) | 70 (5) | NS | NS |
| Single-kidney extraction ratio of thalamate (%) — Affected kidney | 18 (1) | 6 (1)*** | 17 (2) | 13 (3)** | NS | <0·001 |
| Single-kidney extraction ratio of thalamate (%) — Contralateral kidney | 24 (2) | 22 (3) | 22 (2) | 19 (3) | NS | NS |

NS = Not significant.
*p<0·05. **p<0·01. ***p<0·001.

TABLE III—*Long term effects of captopril in patients with unilateral renal artery stenosis (group 1 v group 2). Values are means (SEM in parentheses)*

| | Group 1 (cases 1-7) | | Group 2 (cases 8-14) | | p Values for differences between groups 1 and 2 | |
|---|---|---|---|---|---|---|
| | Before captopril | After captopril | Before captopril | After captopril | Before captopril | After captopril |
| Mean arterial pressure (mm Hg) | 143 (7) | 111 (6)*** | 140 (8) | 114 (5)*** | NS | NS |
| Serum creatinine (μmol/l) | 100 (6) | 122 (9)** | 113 (12) | 116 (12) | NS | NS |
| Uptake of $^{99m}$Tc-DTPA by affected kidney (% of total uptake) | 34 (3) | <10 | 31 (4) | 30 (4) | | |

NS = Not significant.
**p<0·01. ***p<0·001.
*Conversion: SI to traditional units*—Creatinine: 1 μmol/l ≈ 0·01 mg/100 ml.

remaining three patients were not restudied after discontinuation of captopril treatment, but DTPA kidney scans after reconstructive vascular surgery showed improved uptake on the affected side.

## Discussion

This study shows that in a substantial number of patients with unilateral renal artery stenosis the renal extraction ratio of both $^{131}$I-sodium iodohippurate ($E_H$) and $^{125}$I-thalamate ($E_T$) is greatly reduced on the affected side when captopril is given as the only drug. The fall in $E_H$ may be explained by a shortened plasma transit time through the kidney due to intrarenal vasodilatation. This has also been observed with vasodilatation induced by other agents.[18] $E_T$ equals filtration fraction, and the fall in $E_T$ after captopril may reflect the dilatation of postglomerular arterioles.[10] Captopril also lowered $E_H$ and $E_T$ of kidneys with a normal artery but the changes were not as great as for kidneys with artery stenosis. In our patients the fact that the decrease in $E_H$ on both sides was not associated with a decrease in total clearance of sodium iodohippurate is further support for vasodilatation in the kidney, probably on the affected as well as the non-affected side. Increase in total renal blood flow and decrease in total filtration fraction after captopril have been reported in patients with essential hypertension.[19] In those studies the clearance of para-aminohippurate was used as an estimate of renal plasma flow with the implicit assumption that the renal extraction efficiency was high and remained constant. This, however, may be misleading, as shown by our results; the effects of captopril on renal blood flow and filtration fraction would be grossly underestimated in some patients.

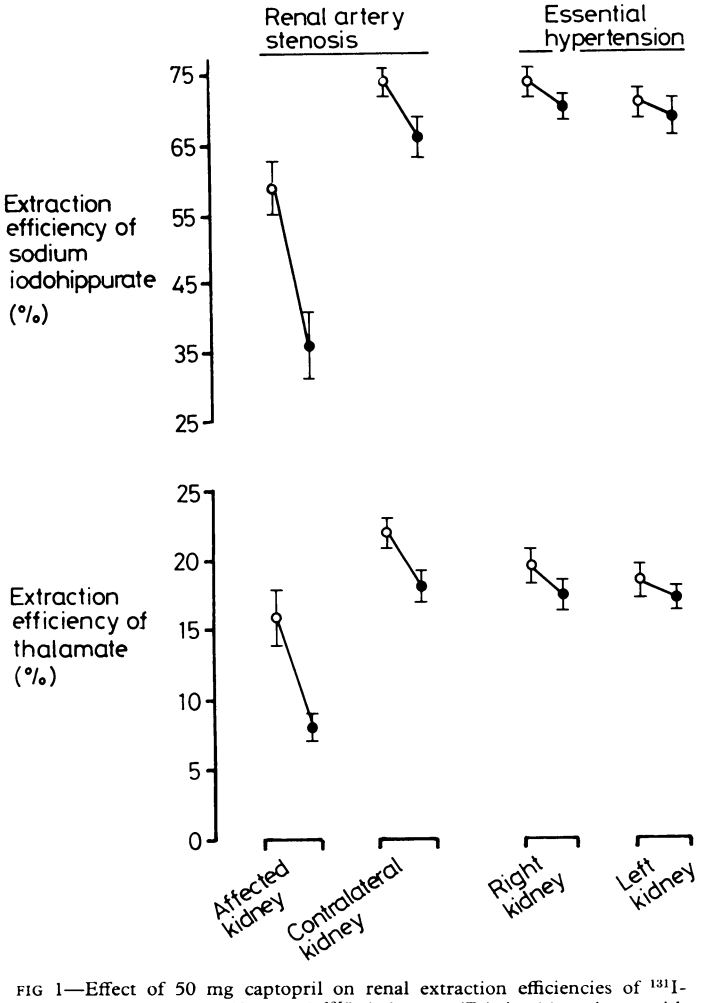


FIG 1—Effect of 50 mg captopril on renal extraction efficiencies of $^{131}$I-sodium iodohippurate ($E_H$) and $^{125}$I-thalamate ($E_T$) in 14 patients with unilateral renal artery stenosis and 17 patients with essential hypertension. In patients with renal artery stenosis changes in $E_H$ and $E_T$ were significant on both sides (p<0·01). Changes in essential hypertension were also significant (p<0·05).

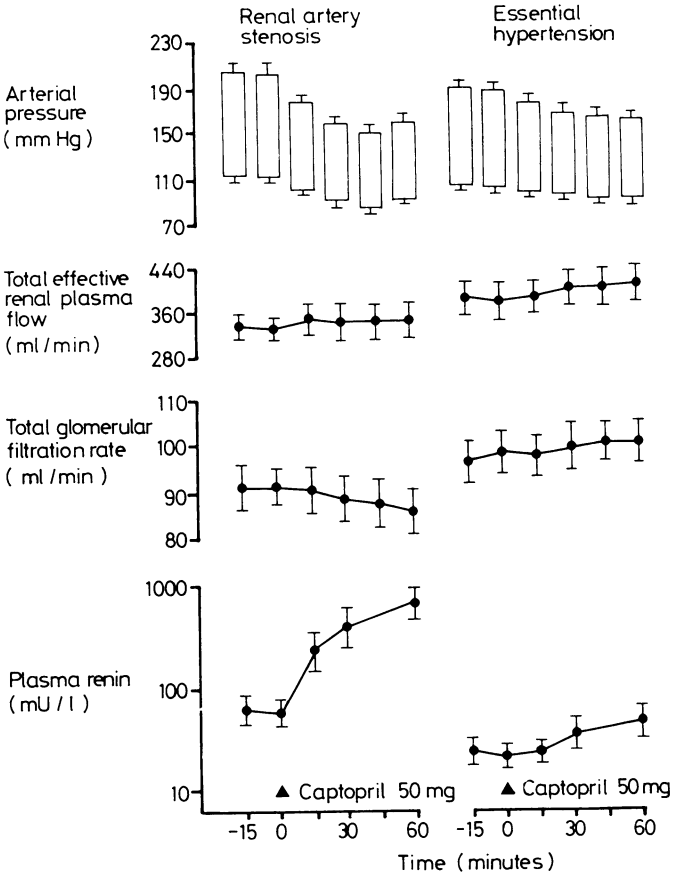


FIG 2—Effect of 50 mg captopril on total clearances of $^{131}$I-sodium iodohippurate (effective renal plasma flow) and $^{125}$I-thalamate (glomerular filtration rate) in 14 patients with unilateral renal artery stenosis and 17 patients with essential hypertension. Effect of captopril after 60 minutes was significant for systolic and diastolic intra-arterial pressure (p<0·001) and for renin (p<0·01).

Other significant findings were the changes in the $^{99m}$Tc-DTPA kidney scans showing a decrease in glomerular filtration rate during long term captopril treatment. This was seen only with kidneys affected by artery stenosis. It also appeared to be an all or none phenomenon—that is, the uptake of DTPA by the affected kidney became either almost zero or showed little change. The deterioration in renal function was observed in half of our patients, but this high incidence may have been related to selection; all had been referred to us because of severe hypertension that was difficult to control.

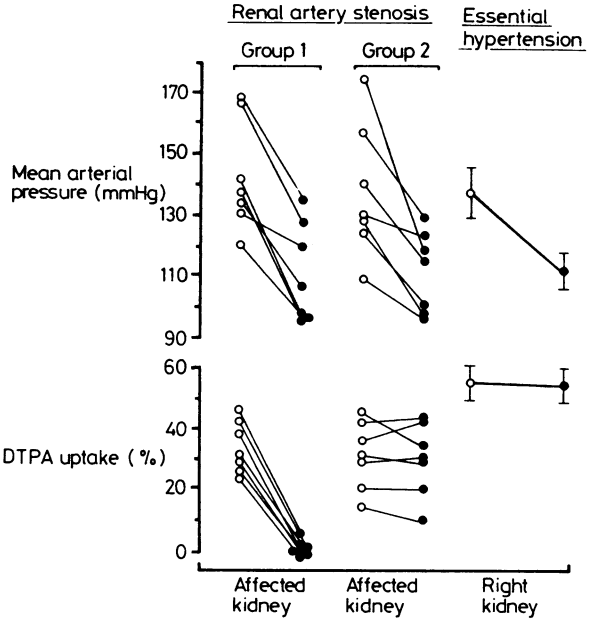


FIG 3—Effect of long term captopril 150 mg daily on blood pressure and single kidney uptake of $^{99m}$Tc-DTPA in 14 patients with unilateral renal artery stenosis and 17 patients with essential hypertension. Patients with renal artery stenosis divided into two groups according to change in DTPA uptake (see table III for statistics). Values in patients with essential hypertension presented as means and SEM. Mean arterial pressure calculated as diastolic pressure + 0·3 × pulse pressure. London School of Hygiene sphygmomanometer used. Three consecutive readings with patient in recumbent position were averaged. Effect of captopril on mean arterial pressure in patients with essential hypertension was not different from effect in two groups of patients with renal artery stenosis.

Deterioration of renal function does not occur only with captopril,[20] but conceivably converting enzyme inhibitors may be especially likely to cause this complication. Acute converting enzyme inhibition with captopril or angiotensin II blockade with saralasin caused renal failure in rats with chronic two kidney, two clip hypertension pretreated with frusemide.[21] By contrast, the direct smooth muscle relaxants minoxidil and dihydralazine did not have this effect, despite a similar fall in systemic arterial pressure. Such findings have also been reported in a few patients with bilateral renal artery stenosis or with a stenotic artery to a solitary functioning kidney.[7-9] Most of these patients had been treated with captopril in combination with other drugs, particularly diuretics. More work is needed to establish whether captopril either alone or combined with a diuretic is more harmful for the kidney affected by artery stenosis than other antihypertensive drugs.

The effect of captopril on systemic arterial pressure in our patients who responded with loss of filtration on the affected side was not greater than in those who maintained filtration. Thus the degree of reduction in blood pressure is probably not the only factor determining whether or not renal function can be maintained during captopril. Experimental constriction of a renal artery is known to be followed by vasoconstriction within the affected kidney, and there is good evidence that the post-glomerular vascular resistance is increased so that filtration pressure is restored and glomerular filtration rate is maintained. This mechanism is impaired by converting enzyme inhibition, and filtration pressure may fall, particularly when systemic arterial pressure also falls.[11,12] Increased glomerular blood flow after intrarenal vasodilatation may partly compensate for this.[22] When filtration pressure falls below a critical level, however, the kidney stops filtering. It is tempting to assume that this occurred in some of our patients. It was the patients with the greatest reductions in $E_H$ and $E_T$ after captopril who responded with loss of filtration. Presumably these were the patients with the most severe artery stenosis. An alternative or additional mechanism contributing to the fall in glomerular filtration rate might be that a critically severe stenosis of a large artery becomes more severe after dilatation of the distal vascular bed.[23,24] This has been reported in renal artery stenosis induced by cuff constrictors in intact instrumented dogs.[25]

Fortunately, in none of our patients were the effects of captopril on renal function associated with irreversible damage to the renal parenchyma. DTPA uptake was restored by discontinuing captopril or after reconstructive vascular surgery. Radioisotope renography should be performed in any patient with renal artery stenosis who is taking captopril. Perhaps we should go even further. Until now captopril has been used in hypertension mainly as a third line drug when other drugs have failed. Renovascular hypertension is not uncommon in this difficult group. Hence radioisotope renography should probably be performed in every patient who has been given captopril because of poor response to other drugs when the possibility of renal artery stenosis has not been excluded. We believe that finding severely impaired renal function on one side during captopril treatment calls for withdrawing the drug or perhaps lowering the dose. In such cases renal artery stenosis is likely to be the underlying disease.

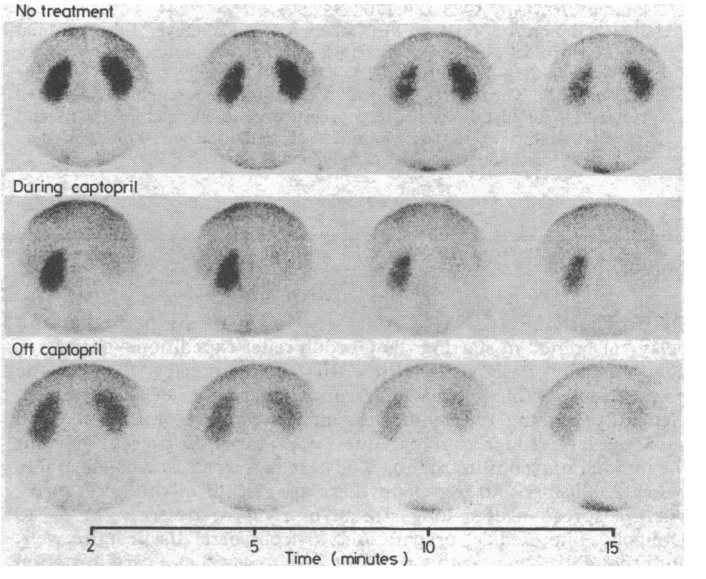


FIG 4—Sequential $^{99m}$Tc-DTPA kidney scans in patient with unilateral renal artery stenosis (case 3; table I) before captopril, after four weeks of captopril 150 mg daily, and one week after stopping captopril. Time after radioisotope injection indicated.

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## References


[1] Case DB, Atlas SA, Laragh JH, Sealey JE, Sullivan PA, McKinstrey DJ. Clinical experience with blockade of the renin-angiotensin-aldosterone system by an oral converting-enzyme inhibitor (SQ 14.255, captopril) in hypertensive patients. *Progr Cardiovasc Dis* 1978;**21**:195-206.

[2] Atkinson AB, Brown JJ, Cumming AMM, *et al.* Captopril in renovascular hypertension: long-term use in predicting surgical outcome. *Br Med J* 1982;**284**:689-92.

[3] De Bruyn JHB, Man in 't Veld AJ, Wenting GJ, Derkx FHM, Schalekamp MADH. Haemodynamic profile of captopril treatment in various forms of hypertension. *Eur J Clin Pharmacol* 1981;**20**:163-8.

[4] Staessen J, Bulpitt C, Fagard R, Lijnen P, Amery A. Long-term converting-enzyme inhibition as a guide to surgical curability of hypertension associated with renovascular disease. *Am J Cardiol* 1983; **51**:1317-22.

[5] Farrow PR, Wilkinson R. Reversible renal failure during treatment with captopril. *Br Med J* 1979;i:1680.

[6] Collste P, Haglund K, Lundgren G, Magnusson G, Ostman J. Reversible renal failure during treatment with captopril. *Br Med J* 1979;ii:612-3.

[7] Coulie P, De Plaen JF, Van Ypersele de Strihou C. Captopril-induced acute reversible renal failure. *Nephron* 1983;**35**:108-11.

[8] Curtis JJ, Luke RG, Whelchel JD, Diethelm AG, Jones P, Dustan HP. Inhibition of angiotensin-converting enzyme in renal-transplant recipients with hypertension. *N Engl J Med* 1983;**308**:377-81.

[9] Hricik DE, Browning PJ, Kopelman R, Goorno WE, Madias NE, Dzau VJ. Captopril-induced functional renal insufficiency in patients with bilateral renal-artery stenosis or renal-artery stenosis in a solitary kidney. *N Engl J Med* 1983;**308**:373-6.

[10] Silas JH, Klenka Z, Solomon SA, Bone JM. Captopril-induced reversible renal failure: a marker of renal artery stenosis affecting a solitary kidney. *Br Med J* 1983;**286**:1702-3.

[11] Hall JE, Guyton AC, Jackson TE, Coleman TG, Lohmeyer TE, Trippodo NC. Control of glomerular filtration rate by renin-angiotensin system. *Am J Physiol* 1977;**233**:F366-72.

[12] Anderson WP, Korner PI, Johnston CI. Acute angiotensin II-mediated restoration of distal renal artery pressure in renal artery stenosis and its relationship to the development of sustained one-kidney hypertension in conscious dogs. *Hypertension* 1979;**1**:292-8.

[13] Anderson CF, Sawyer TK, Cutler RE. Iothalamate sodium I 125 vs cyanocobalamin Co 57 as a measure of glomerular filtration rate in man. *JAMA* 1968;**204**:105-8.

[14] Schalekamp MADH, Birkenhäger WH, Zaal GA, Kolsters G. Haemodynamic characteristics of low-renin hypertension. *Clinical Science and Molecular Medicine* 1977;**52**:405-12.

[15] Pors Nielsen S, Lehd Møller M, Trap-Jensen J. $^{99m}$Tc-DTPA scintillation-camera renography: a new method for estimation of single-kidney function. *J Nucl Med* 1977;**18**:112-7.

[16] Bratt CG, Larsson I, White T. Scintillation camera renography with $^{99m}$Tc-DTPA and $^{131}$I-Hippuran. *Scand J Clin Lab Invest* 1981;**41**: 189-97.

[17] Derkx FHM, Tan-Tjiong HL, Wenting GJ, Boomsma F, Schalekamp MADH. Asynchronous changes in prorenin and renin secretion after captopril in patients with renal artery stenosis. *Hypertension* 1983;**5**: 244-56.

[18] Velasquez MT, Notargiacomo AV, Cohn JN. Influence of cortical plasma transit-time on p-aminohippurate extraction during induced renal vasodilatation in anaesthetized dogs. *Clin Sci* 1972;**43**:401-11.

[19] Mimran A, Brunner HR, Turini GA, Waeber B, Brunner D. Effect of captopril on renal vascular tone in patients with essential hypertension. *Clin Sci* 1979;**57**:421s-3s.

[20] Dean RH, Kieffer RW, Smith BM, *et al.* Renovascular hypertension. Anatomic and renal function changes during drug therapy. *Arch Surg* 1981;**116**:1408-14.

[21] Helmchen U, Gröne HJ, Kirchertz EJ, *et al.* Contrasting renal effects of different antihypertensive agents in hypertensive rats with bilaterally constricted renal arteries. *Kidney Int* 1982;**22**, suppl 12:S198-S205.

[22] Brenner BM, Troy JL, Daugharty TM, Deen WM, Robertson CR. Dynamics of glomerular ultrafiltration in the rat, II: plasma-flow dependence of GFR. *Am J Physiol* 1972;**223**:1184-90.

[23] Young DF, Cholvin NR, Kirkeeide RL, Roth AC. Haemodynamics of arterial stenoses at elevated flow rates. *Circ Res* 1977;**41**:99-107.

[24] Gould KL. Dynamic coronary stenosis. *Am J Cardiol* 1980;**45**:286-92.

[25] Anderson WP, Korner PI. The importance of renal vascular tone in determining the severity of renal artery stenosis in dogs. *J Physiol (Lond)* 1980;**305**:31-41.


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ONE HUNDRED YEARS AGO Owing to the hesitation and delay "at home" in starting the Gordon relief expedition—or, as it is now officially designated, "the Nile Expeditionary Force"—the preparations for the approaching campaign are not yet far advanced. Generally speaking, the military and medical portions of the expedition are about level, so far as preparedness goes; for at the present moment the number of troops in front of Wady Halfa is small, and considerably below 2,000 men, all told; and these are not all in the same place, but are spread over a long stretch of river or desert right up to Dongola. The medical preparations in front of Halfa promise well; and, though it is not at this moment easy to say that a certain station has or has not its station-hospital in good working order, I think I am well within the mark in stating that the chain of hospitals all along the river to the front is in a fair state of progress towards fitness for the reception of troops, when there are troops in their vicinity. But the difficulties in transporting stores and equipment up the river are enormous, even with the stupendous and costly machinery of men and material now engaged all day at the work. A long stretch of rapids and dangerous rocks, and a weary waterless desert-track, are truly obstacles enough to appal any but the most stout-hearted general. It is commonly supposed that the expedition will be ready to begin active operations from Debbeh or Ambukol by New Year's Day—possibly by December 15th. The officers and men of the Medical Staff Corps are doing good steady work; not professionally, for that has not yet come, except in a minor degree, but in veritably putting the "shoulder to the wheel," or (to speak more accurately) the towing-rope, and taking their turn at hauling their boats up rapids, or weary stretches of smooth water when the wind drops, which, at this season, very often occurs. This may seem a small matter to you at home in England. To some persons, on reading the above, agreeable thoughts of a short towing experience on the Thames, or some pleasant English river, with a good luncheon-basket on board, will come to mind. Towing, or rather, hauling, up the Nile, means quite a different thing; it includes the sober reality of a sweltering sun, flies, sand, and, last but not least, the everlasting bully-beef, ship's biscuits, and Nile water, for few are provided with anything more potable; while, at night, snatches of sleep on a boat crowded with soldiers and a sprinkling of Arabs, with their noises and other unpleasant surroundings, are a poor preparation for next day's toil. A movable field-hospital to carry 110 sick or wounded, and a camel-bearer company to convey 200 patients, are working their way up the river in sections, or small parties, all to concentrate at Dongola or Debbeh; but very possibly these numbers may be altered by unforseen circumstances; at all events, these two bodies will accompany the "striking force," that is, the Camel Corps and advanced line. The Camel Corps, it is reported, is daily increasing in strength by the addition of Marine and Mounted Infantry, and will eventually number 1,700 men, all on camels. I have little of medical interest yet to communicate. The campaign, in its medical or surgical aspects, has not yet commenced. The army is forming up, and the usual diseases of the Europeans of the soldiers' age—enteric, and other milder fevers, dysentery, and venereal maladies, are the causes of hospital admissions; and they are not of undue prevalence. Good beds, filtered water, good cooking, fair rations, ample ventilation, a temperature of 80° by day and of 60° or less at night, and fine salubrious northerly breezes, are the conditions for sick and healthy, favourable and restorative for the sick, agreeable and salubrious for the healthy. Two officers have been smitten with sunstroke. I am told they had both had it before, and had come out from home too soon, contrary to advice, military zeal overcoming prudence and medical representations.

(*British Medical Journal* 1884;ii:1157.)