

Does the pressure gradient in renal artery stenosis before and after percutaneous transluminal renal angioplasty predict initial and long-term outcome?

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Abstract

Aim: To evaluate whether measurement of mean pressure gradient (MPG) over a renal artery stenosis (RAS) before and after percutaneous transluminal renal angioplasty (PTRA) predicts initial and long-term outcome.

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Materials and methods: In our institution, PTRA has generally been performed in RAS patients with MPG > 10 mmHg. We related MPG before and after PTRA in 287 consecutive RAS patients (age 65 ± 14 years, 152 (53%) men) undergoing PTRA on 332 kidneys to blood pressures (BP), renal function and treatment during 4.1 ± 3.3 years of retrospective follow-up. The patients were divided into four groups according to MPG before PTRA: group A, MPG 0-29 mmHg, $n = 60$ patients, 79 kidneys; group B, MPG 30-59 mmHg, $n = 48$ patients, 57 kidneys; group C, MPG 60-199 mmHg, $n = 40$ patients, 43 kidneys; group D, occlusive pressure without numerical value obtainable, $n = 139$ patients, 153 kidneys.

Results: In groups A, B, and C, MPG before PTRA correlated with systolic BP (SBP) before PTRA, ($r = 0.242$; $p = 0.0032$) and with the reduction in SBP during follow-up ($r = 0.243$; $p = 0.0034$). MPG decreased from 38.7 ± 26.8 mmHg before PTRA to 1.3 ± 3.2 mmHg after PTRA ($p < 0.0001$). SBP and diastolic BP and the number of antihypertensive drugs decreased in all groups. Residual MPG after PTRA predicted the need for later endovascular re-do (52% vs. 14%; $p < 0.0001$).

Conclusion: PTRA has positive effects on BP and treatment in RAS patients with MPG > 10 mmHg. Whether PTRA is also indicated with MPG < 10 mmHg requires further evaluation. Residual MPG post PTRA predicts re-do but not the outcome of the intervention.

Keywords

Renal artery stenosis; PTRA; pressure gradient; blood pressure; stenting.

Introduction

If sufficient in magnitude, the hydrostatic pressure gradient between the aorta and the renal arteries resulting from renal artery stenosis (RAS)^[1] leads to arterial hypertension due to decreased glomerular filtration rate (GFR), and renal ischaemia^[1,2]. In the clinical setting of arterial hypertension and suspected RAS, angiography is the gold standard for diag-

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nosis, and in the 1980s this technique was further developed to allow percutaneous transluminal angioplasty (PTRA), which in the 1990s became the standard treatment for RAS^[3].

To evaluate which patients are candidates for PTRA and to predict the outcome of PTRA, precise measurements of the degree of RAS are needed^[4]. A comparison of renal angiograms by experienced radiologists evaluating the severity of renal arterial stenosis showed that the general agreement among experts is poor^[5].

Furthermore, the accuracy of digital subtraction angiography in interpreting the degree of RAS is insufficient because of variations in the evaluation of both minimum luminal and reference diameter^[6]. Magnetic resonance (MR) angiography and computed tomography (CT) angiography, which are often used nowadays for diagnosis of RAS^[7,8], do not allow an accurate judgment of the degree of stenosis. A role for renal artery Doppler ultrasonography has also been proposed in assessing the physiological significance of RAS, as well as the likelihood of a successful response to treatment^[9].

Intra-arterial measurement of the pressure gradient over the stenosis, as used before PTA in the iliac arteries^[10], is recommended in the guidelines for angiography and angioplasty of RAS^[11] as a complement to angiography in order to select patients for the invasive treatment of RAS. Different technical methods have been used for such assessments^[12,13] and there is no consensus regarding the level of mean or systolic pressure gradient that indicates a haemodynamically significant RAS. In the iliac arteries, peak systolic pressure gradients of more than 10%, or a mean pressure gradient of more than 5% ($100 \times$ [blood pressure proximal to the stenosis – blood pressure distal to the stenosis/blood pressure proximal to the stenosis]) across the narrowed vascular lumen have generally been accepted as signs of a haemodynamically significant stenosis^[14,15].

At our institution, we have measured intra-arterial pressure gradients during angiography in renovascular hypertension since the end of the 1980s, and normally performed PTRA on RAS patients with MPG above 10 mmHg. This retrospective study was undertaken to evaluate the importance of the pressure gradient before and after successful PTRA of RAS for clinical outcome, i.e. effects on blood pressure (BP), renal function, mortality, and the need for antihypertensive medication, as well as for the clinical need for endovascular re-do procedures.

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Methods

Materials

Our department is a referral centre for patients from southern Sweden (1.6 million inhabitants) diagnosed with renovascular hypertension caused by RAS. Our indications for angiography are hypertension (accelerated, refractory, malignant or hypertension with intolerance to medication), or renal salvage (unexplained worsening of renal function, or secondary to antihypertensive treatment particularly with an ACE inhibitor or an angiotensin II receptor blocker), or recurrent pulmonary oedema^[16]. All 308 consecutive patients who underwent renal angiography with PTRA because of a diagnosis of RAS between 1987 and November 2004 were identified. Results of attempted measurements of the mean pressure gradient (MPG) over the stenosis during angiography were available for 287 patients (93%, age 65 ± 14 years, 152 (53%) men) and 332 kidneys. One hundred and sixty-six (50%) stenoses were located ostially, and 44 (15%) were due to fibromuscular dysplasia (FMD). Stenting had been performed in 126/332 (38%) procedures.

Methods

Calcium channel blockers were given before PTRA to pharmacologically achieve a maximal systemic vasodilatation of the peripheral vascular bed. Patients with S-creatinine $\geq 130 \mu\text{mol/l}$ underwent CO₂ angiography. Before PTRA, the intra-arterial BP in the aorta and in the poststenotic region of the renal artery was measured continuously by means of a pressure sensor and pressure curves were recorded. Normally, a 4 French (F) end-hole catheter was advanced through a 6F introducer from the femoral artery into the renal artery distally to the stenosis, and the two pressure curves from the introducer in the aorta and from the guiding catheter distal to the RAS were compared. The dilatation equipment was then advanced across the stenosis, and PTRA with or without stenting was made. After the PTRA, the renal artery pressure distal to the PTRA-treated area was again recorded

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followed by a continuous recording of the BP as the catheter was pulled back into the aorta. The mean arterial pressure gradient (MPG) was measured. The measurement of MPG after PTRAs guided the decision whether to rely on the PTRAs alone or whether to place a stent, and whether to repeat the dilatation until the pressure gradient was abolished. Stenting was performed in cases of angioplasty failure^[16] (elastic recoil, dissection, >50% residual stenosis after repeated PTRAs attempts) or residual MPG > 10 mmHg. Several different types of catheters, balloons and stents have been used during the 17-year study period.

Control renal angiography was performed if reocclusion or stenosis was suggested by the recurrence of uncontrollable hypertension or by deterioration of renal function.

Definitions of MPG and different patient groups

We defined MPG as: aortic (systolic BP – diastolic BP/3 + diastolic BP) – renal artery (systolic BP – diastolic BP/3 + diastolic BP), i.e. aortic pulse pressure/3 – renal pulse pressure/3.

Balloon diameter at PTRAs was $(6.1 \pm 0.9 \text{ mm})$. In 139 patients (153 kidneys); no numerical value for the MPG could be registered because of occlusive pressure from the pressure catheter during the measurement procedure. We arbitrarily divided the 287 patients into four groups according to the following cohorts of estimated MPG: group A, MPG 0–29 mmHg, $n = 60$ patients, 79 kidneys; group B, MPG 30–59 mmHg, $n = 48$ patients, 57 kidneys; group C, MPG 60–199 mmHg, $n = 40$ patients, 43 kidneys; group D, occlusive pressure without obtainable numerical value, renal artery occlusion, or such a severe stenosis that the 4F catheter occluded the lumen, $n = 139$ patients, 153 kidneys.

Follow-up

We retrospectively registered the MPG, systolic and diastolic BP (SBP and DBP), serum (S-) creatinine, GFR estimated according to the Cockcroft–Gault formula^[17], and the number

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of antihypertensive drugs used before PTR A, at discharge, after 1 month, 1 year and at the last follow-up after mean 4.1 ± 3.3 years from patient files at our and other referring hospitals in the region. Supine blood pressure recordings were identified from patient files; when several measurements were available, the mean was used for calculations. Due to their effects upon BP, diuretics and nitrates were defined as antihypertensive drugs, even though they may have primarily been given for other reasons.

Information about mortality and cause of death was obtained through the registers of the Swedish Board of Health and Welfare. The ethics committee of the University of Lund approved the study.

Statistics

Differences between groups were evaluated with the Kruskal-Wallis test and the Mann-Whitney *U*-test, and differences over time within a group with the Wilcoxon signed rank test. Differences in frequency were evaluated with the chi-squared test. The Spearman correlation coefficient was used to evaluate correlations. Tests were two-tailed and *p*-values <0.05 were considered significant. Results are presented as mean \pm SD. StatView 4.5 (SAS Institute, Cary, NC, USA) was used for the statistical calculations.

Results

Patient characteristics for the four groups A, B, C and D are given in Table 1. In groups A, B and C, MPG correlated with systolic blood pressure before PTR A ($r = 0.242$; $p = 0.0032$), and both MPG before treatment ($r = 0.243$; $p = 0.0034$) and the reduction in MPG achieved by PTR A ($r = 0.229$; $p = 0.0060$) correlated with the reduction in systolic blood pressure during follow up. On the other hand, MPG before PTR A did not correlate with diastolic blood pressure, S-creatinine, glomerular filtration rate, or the number of antihypertensive drugs. In groups A, B and C, the MPG decreased from 38.7 ± 26.8 mmHg before PTR A to

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Table 1: Patient characteristics in different groups

	Group A	Group B	Group C	Group D
Patients (<i>n</i>)	60	48	40	139
Kidneys (<i>n</i>)	79	57	43	153
Fibromuscular dysplasia (<i>n</i> (%))	13 (22)	7 (15)	6 (15)	18 (13)
Age (years)	64 ± 13	65 ± 14	65 ± 13	65 ± 13
BMI (kg/m ²)	25 ± 5	26 ± 5	24 ± 6	24 ± 5
Pressure gradient before PTRAs	15.7 ± 6.3	40.7 ± 8.8	78 ± 16.6	
Pressure gradient after PTRAs	1.0 ± 2.2	0.8 ± 1.9	2.6 ± 5.2	1.1 ± 2.9
Stent (<i>n</i> (%))	18 (23)	25 (44)	19 (44)	64 (42)
Re-do (<i>n</i> (%))	14 (18)	14 (25)	10 (24)	36 (24)
Mortality (<i>n</i> (%))	23 (38)	18 (38)	13 (33)	52 (37)

Group A, pressure gradient 0–29 mmHg; group B, pressure gradient 30–59 mmHg; group C, pressure gradient 60–199 mmHg; group D, occlusive pressure without numerical value. BMI, body mass index; PTRAs, percutaneous transluminal renal angioplasty.

1.3 ± 3.2 mmHg after PTRAs ($p < 0.0001$). In group D, MPG was 1.1 ± 2.9 mmHg after PTRAs. Patients with FMD were equally distributed in the groups (Table 1). Before PTRAs in groups A, B and C, MPG was equal in patients with atherosclerotic stenosis or FMD (41.6 ± 27.7 mmHg vs. 37.3 ± 27.3 mmHg; $p = 0.488$), whereas after PTRAs, MPG was lower in the atherosclerotic group (0.9 ± 2.8 mmHg vs. 4.0 ± 4.6 mmHg; $p = 0.0002$). In groups A–C, MPG was higher before PTRAs (44.3 ± 24.8 mmHg vs. 35.6 ± 27.4 mmHg; $p = 0.008$), but lower after PTRAs (0.2 ± 1.1 mmHg vs. 1.9 ± 3.8 mmHg; $p = 0.0031$) in patients receiving stents than in patients that were not stented, and the reduction in MPG was larger in stented patients (44.1 ± 24.8 mmHg vs. 33.6 ± 26.7 mmHg; $p = 0.0021$). When the effects of PTRAs upon blood pressure, renal function, antihypertensive drugs, mortality, and endovascular re-do were assessed (Table 2), only systolic blood pressure

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Table 2: Effects of PTRAs upon blood pressure (BP), S-creatinine (crea), estimated glomerular filtration rate (GFR) and number of antihypertensive drugs (drugs) in different MPG groups

	A (n = 60)	B (n = 48)	C (n = 40)	D (n = 139)
Before PTRAs				
BP (mmHg)	175 ± 30/92 ± 16	179 ± 26/92 ± 15	191 ± 28 ^d /94 ± 17	179 ± 28/94 ± 14
S-crea (μmol/l)	189 ± 187	184 ± 164	174 ± 124	175 ± 136
GFR (cm ³ /min)	50 ± 30	50 ± 29	48 ± 30	51 ± 31
Drugs (n)	2.7 ± 1	2.7 ± 1.2	2.7 ± 1.4	2.7 ± 1.2
At discharge				
BP (mmHg)	145 ± 24 ^c /90 ± 19 ^c	145 ± 20 ^c /77 ± 11 ^c	151 ± 21 ^c /79 ± 10 ^c	144 ± 21 ^c /77 ± 12 ^c
S-crea (μmol/l)	182 ± 172	190 ± 145	155 ± 91	170 ± 132
GFR (cm ³ /min)	53 ± 32	48 ± 30	52 ± 32	52 ± 29
Drugs (n)	2.3 ± 0.9	2.4 ± 1.3 ^b	2.3 ± 1.3 ^b	2.2 ± 1.3 ^c
After 1 month				
BP (mmHg)	159 ± 6 ^b /90 ± 19	156 ± 24 ^c /84 ± 10	162 ± 31 ^c /88 ± 17	155 ± 27 ^c /81 ± 14 ^c
S-crea (μmol/l)	156 ± 98	179 ± 115	157 ± 108	164 ± 116
GFR (cm ³ /min)	50 ± 27	53 ± 32	51 ± 32 ^a	53 ± 31
Drugs (n)	2.3 ± 1.07 ^a	2.4 ± 1.4	2.2 ± 1.3 ^b	2.1 ± 1.3 ^c
After 1 year				
BP (mmHg)	156 ± 25 ^b /87 ± 15	154 ± 24 ^c /91 ± 41	157 ± 24 ^c /86 ± 14	154 ± 25 ^c /83 ± 14 ^c
S-crea (μmol/l)	181 ± 125	161 ± 110	148 ± 109	154 ± 126
GFR (cm ³ /min)	49 ± 29	56 ± 31	54 ± 30	56 ± 30
Drugs (n)	2.5 ± 1.1	2.4 ± 1.4	2.8 ± 1.3 ^a	2.2 ± 1.3 ^c
Final follow-up				
BP (mmHg)	151 ± 26 ^c /81 ± 13 ^c	144 ± 25 ^c /79 ± 13 ^c	147 ± 25 ^c /80 ± 13 ^c	148 ± 23 ^c /80 ± 13 ^c
S-crea (μmol/l)	219 ± 195	216 ± 180 ^a	167 ± 127	197 ± 194
GFR (cm ³ /min)	47 ± 32 ^a	46 ± 29 ^b	50 ± 34	52 ± 31
Drugs (n)	2.5 ± 1.9	2.8 ± 1.3	2.5 ± 1.3	2.3 ± 1.3 ^c

^a*p* < 0.05 compared to same group before PTRAs. ^b*p* < 0.01 compared to same group before PTRAs. ^c*p* < 0.001 compared to same group before PTRAs. ^d*p* < 0.05 compared to same group before PTRAs and <0.01 compared to groups A and D before PTRAs.

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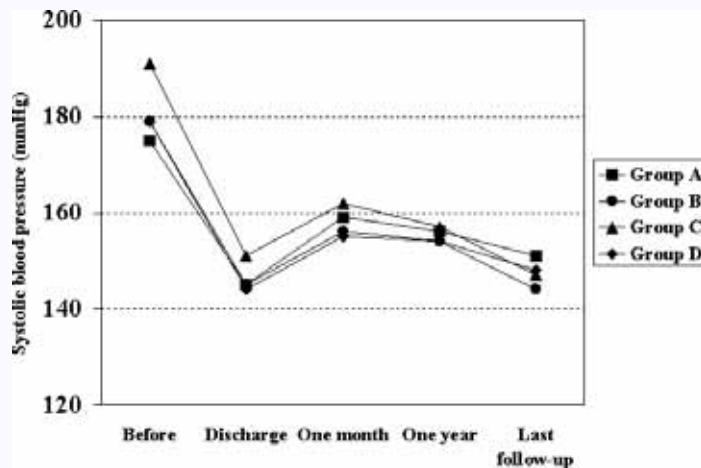


Fig. 1. Systolic blood pressure before and after PTR in patients with renal artery stenosis grouped with respect to mean pressure gradient (MPG) before PTR. Group A, MPG 0-29 mmHg, $n = 60$; group B, MPG 30-59 mmHg, $n = 48$; group C, MPG 60-199 mmHg, $n = 40$; group D, occlusive pressure without obtainable numerical value, renal artery occlusion, or such a severe stenosis that the 4F catheter occluded the lumen, $n = 139$.

before treatment, which was higher in group C than in groups A, B and D, revealed significant differences (Kruskal-Wallis and Mann-Whitney tests) between groups during the 4.1 ± 3.3 year follow-up (Table 2, Fig. 1). No differences were seen between groups concerning mortality, clinical outcome, systolic (Fig. 1) or diastolic (Fig. 2) blood pressures. The number of drugs decreased equally in all groups (Table 2). Even in patients with the lowest mean pressure gradients (<15 mmHg, $n = 34$) systolic blood pressure decreased significantly (from 174 ± 32 to 153 ± 22 mmHg; $p < 0.0021$) whereas no decrease in blood pressure could be demonstrated in the six patients (eight kidneys) undergoing PTR despite a MPG <10 mmHg.

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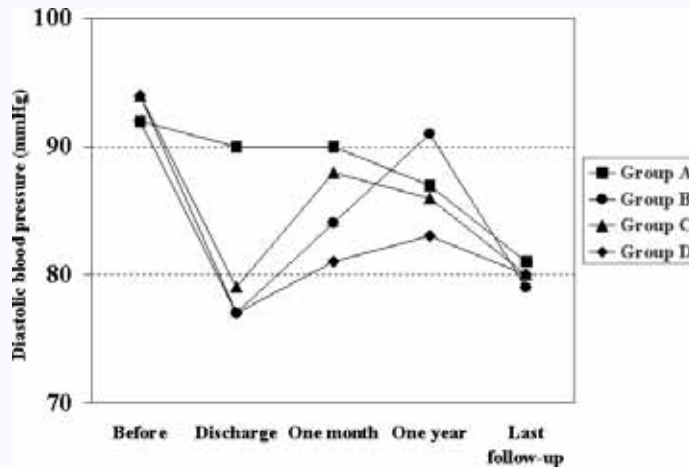


Fig. 2. Diastolic blood pressure before and after PTR in patients with renal artery stenosis grouped with respect to mean pressure gradient (MPG) before PTR. Group A, MPG 0-29 mmHg, $n = 60$; group B, MPG 30-59 mmHg, $n = 48$; group C, MPG 60-199 mmHg, $n = 40$; group D, occlusive pressure without obtainable numerical value, renal artery occlusion, or such a severe stenosis that the 4F catheter occluded the lumen, $n = 139$.

When patients were divided with respect to residual pressure gradient or not after PTR, the clinical need for endovascular re-do during follow-up occurred in 52% of patients with residual MPG vs. 14% of patients without ($p < 0.0001$). On the other hand, there were no differences with regard to the effects on blood pressure, S-creatinine or antihypertensive drugs at the time points analysed. The need for re-do procedure was 18% among stented patients and 25% among non-stented patients ($p = 0.097$). Neither were there any significant differences regarding effects on blood pressure, S-creatinine or the number of the antihypertensive drugs between patients stented or not. There were no complications specifically related to the measurements of MPG.

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Discussion

This retrospective study evaluating the predictive value of the MPG both before and after treatment regarding the long-term effects of PTRA confirmed that the MPG measured by the 4F catheter method is significantly lowered by the procedure^[12] as previously shown for both pressure measurements with the 4F catheter, and the guidewire with a pressure-sensing electronic tip method^[13]. In a previous, smaller study using the same technique as in our institution, the magnitude of the pressure gradient was not significantly related to BP before PTRA, number of antihypertensive drugs, or renal function^[12], whereas in our material, we found a significant correlation between MPG before PTRA and SBP. Our data therefore partly confirm the results of Gross *et al.*^[13] who reported that both systolic and mean pressure gradients measured by the guidewire with a pressure-sensing electronic tip method were highly correlated with stenosis severity, systolic BP and S-creatinine, and suggested a cut-off point of 20 mmHg systolic pressure gradient for intervention^[13].

In a small comparative study, the guidewire with a pressure-sensing electronic tip method used by Gross^[13] and others^[18-22], has indicated lower pressure values than obtained with the 4F catheter when used in the renal arteries^[18], except in patients with very severe stenosis. Colyer and co-workers therefore suggested that the guidewire with a pressure-sensing electronic tip method provides more accurate measurements of pressure gradients across renal artery lesions^[18]. The method is less documented in renal than in coronary arteries^[19-22] however, and carries substantially higher costs than the 4F catheter method. Like several other investigators of RAS^[23-25], we have therefore only used the 4F catheter method in our department.

The effects of PTRA on the MPG were more evident in patients with atherosclerotic RAS than in those with FMD, and in patients receiving stents compared to those not receiving stents. The MPG might be more difficult to evaluate in patients with FMD in which the relationships between the morphologic angiographic picture, the MPG and the pathophysiological consequences of the RAS might not necessarily be the same as in atherosclerotic disease.

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Stenting resulted in a significantly higher reduction of the MPG than PTRA alone. Indications for stenting among our patients were persistent pressure gradients after repeated angioplasty attempts or the occurrence of dissection. However no differences could be demonstrated between stented and non-stented patients concerning BP or renal function or the need for endovascular re-do during clinical follow-up. Importantly, we present data showing that the residual pressure gradient is a predictor of future need for endovascular re-do.

The correlation between MPG and SBP in groups A, B and C, and the fact that SBP was highest in group C before PTRA might be interpreted as an increasing effect on renal haemodynamics with increasing pressure gradient. This effect could only be demonstrated up to a certain level, however, since SBP was again lower among patients in group D with occlusive pressures. Among group D patients with the most narrow RAS, in whom the small 4F catheter causes an occlusion of the vessels not allowing numerical measurements of the MPG, the BP increase seems to have stabilised, as has been described when a severely stenosed or occluded kidney loses its biological function^[26]. Effects upon blood pressure were evident in all groups, however, and patients with occlusive pressure are also candidates for PTRA. Even if MPG before PTRA was related to long-term effects on systolic BP, we found significant effects on BP also in the group with the lowest MPG, just above 10 mmHg. Indeed, in all RAS patients with an MPG > 10 mmHg, PTRA conferred reductions of BP and the need for antihypertensive medication. The clinical importance of defining MPG values above 10 mmHg is therefore limited; if an MPG above this limit is established PTRA should be performed as it confers significant effects upon blood pressure and antihypertensive medication.

A mean pressure gradient of 10 mmHg, obtained either at rest or after vasodilation, was initially proposed as an indicator for the need for angioplasty in iliac arteries^[9], whereas a systolic pressure gradient of 20 mmHg has been proposed as a cut-off for intervention in RAS^[13]. There is no consensus as to whether absolute systolic, peak systolic or mean pressure gradients should be used when evaluating the significance of RAS^[11], however, and at our institution the 10 mmHg mean pressure gradient has been arbitrarily used as the cut-off value for intervention also in RAS. Concerning patients with MPG <10 mmHg,

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we cannot therefore draw any conclusions from our material, as the number of patients was too small. We have shown that it is reasonable to perform PTRA at MPG levels >10 mmHg, but whether an even lower cut-off value should have been chosen will be further studied at our institution, as we also plan to retrospectively study patients that were not treated because of low MPG at angiography with respect to blood pressure and medication.

The residual MPG after PTRA, on the other hand, showed a more evident clinical value in our study, as we found that a residual MPG predicted the need for endovascular re-do. A limitation of our study that has to be noted in this context was the absence of routine angiographic control after revascularisation. Such investigations were performed mainly in cases of recurrent arterial hypertension, which is a strong clinical indicator of restenosis^[27]. Repeated angiography was performed at least once in a substantial proportion of our material (157 patients (55%)). Our findings indicate that the interventionist should aim to eliminate the pressure gradient during PTRA, and that patients should be carefully followed clinically especially in those cases where this cannot be achieved due to technical reasons. Another limitation of our study is that changes in medical therapy between 1987 and 2004 might have influenced the follow-up results. During this time period, medical therapy has improved in all patients irrespective of their MPG at PTRA, however, and this factor is therefore unlikely to have influenced our conclusions concerning pressure gradients. Doppler measures of velocity are related to pressure changes^[9], and another limitation of our study is that we did not routinely perform Doppler ultrasonography which might have provided useful data for comparison with the MPG in the prediction of blood pressure responses to PTRA.

In conclusion, PTRA has positive effects upon BP and antihypertensive medication in all RAS patients with MPG of 10 mmHg and above. Apart from that, the MPG before PTRA was related to the reduction in SBP during follow up, and the clinical effects of PTRA were independent of MPG levels before PTRA. Measurement of MPG is a safe procedure. A residual pressure gradient post PTRA predicts later clinical need for re-do, but not the outcome regarding blood pressure control.

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