Clinical–Pathological Conference

Renovascular Hypertension
To Stent or Not to Stent?

Patrick B. Mark, Ernesto L. Schiffrin, Garry L. Jennings, Anna F. Dominiczak, Ji-Guang Wang, Marc De Buyzere, Jan A. Staessen

Presentation of Case
A 69-year-old women smoker was referred to the nephrology clinic for assessment of hypertension and declining kidney function. At the time of referral, serum creatinine was 241 μmol/L and office blood pressure was 191/100 mmHg. Her general practitioner had already performed 24-hour ambulatory monitoring and found no evidence of a white coat component to the hypertension. The patient was taking 4 antihypertensive agents (nifedipine long acting 60 mg daily, candesartan 32 mg daily, bisoprolol 10 mg daily, and bendroflumethiazide 2.5 mg daily). Serum creatinine was 110 μmol/L when last recorded, 1 year before referral. Physical examination was unremarkable with negative urinalysis for blood and protein.

E.L. Schiffrin: On the history, you have a smoker with impaired renal function and no proteinuria. I think this should evoke suspicions and I thought you should comment on it.

P.B. Mark: Absolutely. The diagnosis is clear that this is probably atherosclerotic renal artery disease. I don’t think there is any debate on this. If there had been proteinuria, it would have opened up the diagnosis to all kinds of glomerulonephritides. We happened to have access to 1 test that day, the ultrasound test. That wouldn’t have been the ideal test to seal the diagnosis.

G.L. Jennings: Was there an abdominal bruit? And would you like to comment on the usefulness of abdominal bruit?

P.B. Mark: I remember examining this lady and there was not an abdominal bruit. I also listened for femoral bruit. She did not have an abdominal or femoral bruit.1 Renal ultrasound revealed asymmetrical kidneys with the left kidney measuring 8.1 cm with loss of cortical tissue. The right kidney measured 11 cm and appeared normal. The positive smoking history, renal impairment, resistant hypertension, and asymmetrical kidneys on ultrasound were highly suggestive of renovascular disease.

E.L. Schiffrin: You mentioned already renal artery stenosis, so I can ask whether you see atherosclerotic renal artery stenosis, in the absence of smoking or diabetes mellitus or other causes of severe disseminated atherosclerosis?

P.B. Mark: I would say no in general, but I have just had a similar case referred to me, which I have yet to see. I think they may need more lipid work-up. It is surprising to see a 40-year-old nonsmoker referred with atherosclerotic renal artery disease.

Her referring physician debated whether further imaging was likely to lead to alteration in management. The rapid decline in kidney function in the presence of a normal-sized right kidney with preserved cortical tissue gave rise to the possibility of remediable critical right renal artery stenosis. We considered magnetic resonance angiography, computed tomographic angiography, and formal invasive renal angiography as imaging modalities for assessment of renal artery stenosis. Impaired renal function with estimated glomerular filtration rate 18 mL/min/1.73 m² is a relative contraindication for magnetic resonance angiography, in light of the risk of nephrogenic systemic fibrosis.2 Therefore, computed tomographic angiography with prehydration was performed as first choice noninvasive imaging. Computed tomographic angiography confirmed the presence of a tight calcific ostial stenosis of right renal artery (arrowed), as well as moderately heavy aortic calcification and an atrophic left kidney (Figure 1).

Intervention With Renal Artery Stenting and Outcome
On the basis of declining kidney function, with resistant hypertension, in the presence of a critical stenosis to a single functioning kidney, we elected to proceed to renal artery intervention. The patient underwent renal artery CO2 angiography with right renal artery angioplasty and stenting without complication (Figure 2).

For the subsequent days, there was a rapid normalization of renal function and substantial improvement in blood pressure, with creatinine falling to 92 μmol/L at 9 months post procedure. When most recently seen at clinic, 18 months post procedure, office blood pressure was well controlled (148/88 mmHg) on 2 agents (bisoprolol and nifedipine) and serum creatinine was 122 μmol/L (estimated glomerular filtration rate 40 mL/min/1.73 m²).

The slight dip in renal function at 18 months post procedure suggests the possibility of late in-stent restenosis, although no repeat imaging has been performed to date.3 Even with this

3. Hypertension is available at http://hyper.ahajournals.org
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minor dip, it is clear that this case represents successful short-to medium-term outcome with renal artery angioplasty plus stenting for atherosclerotic renal artery stenosis. We will continue to work hard with the patient to address her other risk factors for atherosclerosis, including smoking cessation, treatment of dyslipidemia, and optimizing blood pressure control to try and protect the function of the single functioning kidney.

A.F. Dominiczak: Would you want to reimage and perhaps be ready to push the balloon across?

P.B. Mark: That is what we would like to do. However, the patient is extremely reluctant. And with the previous history of smoking and the several drugs, that is her choice. We would like to reimage. We had some debate with our radiologist because with the stent, we may have more difficulty imaging the stenosis and whether a straight angiogram might be better.

J.-G. Wang: You need to use ultrasound imaging to look at the change in the kidney and image the size of the kidney. That will tell us whether it is reversible or not reversible.

P.B. Mark: Yes, I think that is reasonable. If the kidney has become smaller or if the corticomedullary differentiation is less good, then it is possible to say that this may become less and less treatable.

E.L. Schiffrin: Have you succeeded in stopping her from smoking?

P.B. Mark: We have tried very, very hard. The answer is no. It makes you wonder about throwing all these treatments and exposing the patient to procedural risk as well.

**Successful Result in the Context of Recent Clinical Trials**

This successful result contrasts with recent well-conducted, high-profile randomized controlled trials of renal artery angioplasty and stenting compared with optimal medical therapy. The Angioplasty and Stenting for Renal Artery Lesions (ASTRAL),4 Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL),5 and Stent Placement and Blood Pressure and Lipid-Lowering for the Prevention of Progression of Renal Dysfunction Caused by Atherosclerotic Ostial Stenosis of the Renal Artery (STAR)6 trials have consistently failed to show benefit with renal artery stenting compared with medical treatment, in terms of either patient survival, cardiovascular events, renal function, or blood pressure. ASTRAL, which was a global study, including several patients in Glasgow, randomized 806 patients with uni- or bilateral atherosclerotic renal artery stenosis to stenting or medical therapy and showed no difference in blood pressure, renal function, or progression to end-stage renal disease between the groups undergoing intervention compared with medical therapy. Renal artery stenting is not without risk, and in ASTRAL, serious adverse events directly related to renal revascularization were seen in 2.3% patients, including death and toe of limb amputation.4 The smaller STAR trial compared renal artery stenting (64 patients) to medical therapy (76 patients) and showed no overall difference in progression of renal dysfunction between the groups.6 More recently, CORAL, in North America, randomized 947 patients with atherosclerotic renal artery stenosis and either hypertension or chronic kidney disease to renal artery stenting or medical therapy. For a median of 43 months follow-up, there was no difference in the composite end point of death from cardiovascular or renal causes, myocardial infarction, stroke, congestive heart failure, progressive chronic kidney disease, or end-stage renal disease between the stented group and those treated with medical therapy.5

M. De Buyzere: Was she a good candidate to stent?

P.B. Mark: We had a good result. We have stented plenty over the years sometimes, with much poorer results. We would argue if ever there was a case for some benefit to be had, this was it. I do take your point. There was definitely established damage there, the renal function was poor, and the kidney was relatively small in size and there were other patient-related factors.

G.L. Jennings: As you just said, the results of the major trials in atherosclerotic renal artery stenosis have shown no benefit with the intervention over medical therapy. So that raises the question whether you went back a little bit further, would you really consider whether you should image her at all because medical therapy is what the guidelines are going to recommend.

This case demonstrates that there remains a group of patients who do benefit from stenting. Most clinicians accept that recurrent or flash pulmonary edema with preserved left ventricular systolic function in patients with renal artery stenosis
is an indication for renal angioplasty ± stenting. Renal revascularization is unlikely to benefit patients with well-controlled blood pressure even on several agents and stable renal function. Small, shrunken kidneys have undergone irreversible ischemic damage, and functional improvement should not be anticipated with revascularization. Uncertainty remains in patients with renal artery disease and coexistent heart failure. The patients enrolled in these admirable clinical trials cannot represent every clinical scenario, and it is inevitable that high-risk patients, similar to the case presented, present particular diagnostics challenges and may not have been randomized in large numbers to these trials. Better characterization of the renal artery lesion using measures of fractional flow reserve may be helpful. Alternatively, functional assessment of the kidneys for hibernating renal tissue, which may benefit from revascularization, has been described. The results of these clinical trials should not deter clinicians from considering renal artery intervention, in carefully selected cases, where benefit is likely or the risks of stenting are outweighed by the likelihood of rapid progression to end-stage kidney disease in the absence of intervention.

Final Discussion of Hot Topics in Renal Artery Stenosis

A.F. Dominiczak: Can we go back to the picture with the narrowing and closing renal artery? There is a tiny, tiny flow there and it is about to close. What would happen next if nothing had been done? Well, I have had an identical patient. This was my first patient in the blood pressure unit many years ago, and we published this paper with Professor Chris Isles. What happened to the patient next was that she became anuric, the same age; everything was similar. She had malignant hypertension. She completely relied on the tiny bit of 1 closing renal artery. So this is a tightening stenosis to a sole kidney and next is dialysis. It is easy to criticize, but clearly 2 years later, this patient still does not need renal replacement therapy. So something has been achieved.

A. Brady (Glasgow): We had a lot of patients from our series in ASTRAL. For people who don’t know how we recruited it; if a patient had a stenosis like this, they never went in the trial. They got angioplasty. For all the patients who had 50% stenosis, where you weren’t sure, they sort of got put in the trial. I can’t speak for CORAL, but I bet for the CORAL centers, which are mostly North American; those patients with really severe stenosis were never included in the studies. So those trials actually tell us nothing about critical stenosis, and I think for this individual there is clear benefit.

J. Dawson (Glasgow): I would support you. I would have referred that patient for stenting. If we were to go back in time, even with the trial data I would still refer that patient for stenting. I think the more interesting question is what would I do now? Now that the renal function has declined. And the question I have to help me make my mind up is: How much of the bounce in estimated glomerular filtration rate, the improvement, and subsequent change was because of perioperative stopping of the angiotensin receptor blocker and perhaps restarting or was therapy the same the whole way through?

P.B. Mark: The therapy was not the same. I can’t answer the exact magnitude of each change, but the day post stenting, there was a drop in blood pressure. She had a torrential natriuresis and diuresis, blood pressure dropped, all drugs were stopped, then it was a labile evolving situation and it makes it extremely difficult to reinterpret what happened with the reintroduction of the drugs. But we didn’t reintroduce an angiotensin receptor blocker because there was no other compelling reason to do so. Although I think reintroducing any antihypertensive drug will probably lead to a relative drop in the renal perfusion again. We don’t know what her actual baseline is.

G.L. Jennings: Just a comment on the people with really tight stenosis didn’t go into these trials, so we don’t really know. CORAL did a retrospective subgroup analysis; those with a stenosis over 80% didn’t show any different from those with a stenosis <80%. You probably need 80% for it to be functionally significant.

M. De Buyzere: For functional renal reserve, do you have a proposal for a cut-off where you should do it? For pressure-wire for instance.

P.B. Mark: I don’t. For ASTRAL, I don’t recall the exact entry criteria, but it was ≈50% to 70%. It was a less severe stenosis. We have no experience pressure-wiring. We have some experimental experience of doing magnetic resonance-perfusion renography, which has been published by the Manchester group. It does look impressive, for predicting response to renal revascularization, but we don’t have a big enough case series of those. I think it comes back to the ultrasound actually. If they have a decent-size cortex and a reasonable-size kidney, there is a reasonable chance it might be a good outcome. If it’s a 9 cm kidney or below, it’s unlikely to be a good result.

M. Walters (Glasgow): I am going to test Anna’s earlier assertion that there are no stupid questions. Now that the renal function is deteriorating 9 months after the stent was inserted, the question is whether it is instant restenosis or not. What is the role or is there a potential role for contrast-free imaging, using for instance time of flight magnetic resonance angiography, which would obviate any risk of contrast-induced injury to the patient but may be sufficient to answer the specific question about the presence or absence of in-stent restenosis?

P.B. Mark: I’m not sure that is a stupid question. That is way over my head. Seriously, I think that with magnetic resonance angiography, time of flight imaging is a beautiful concept. But the artifact with magnetic resonance imaging relating to the actual stent itself is going to make this difficult. I don’t have any experience with it.

J.A. Staessen: Why would you do the imaging again in this patient? Suppose you find out that the stent is thrombosed. What would you do?

P.B. Mark: I hope it is not thrombosed as the renal function would be considerably worse. I think that is an extremely difficult question to answer. If we find that there is a significant degree of in-stent restenosis, do we go back and subject the patient to another procedure and we will go round in the loop again?

E.L. Schiffrin: If we are spending so much money, or intend to, on this patient, why can’t we spend a lot of money on stopping her from smoking? Surely, this has contributed to any additional vascular damage that has occurred since the recent intervention.
J.-G. Wang: How often do you see this kind of patient? If you see rarely, 1 or 2 patients a year, I think that is not a problem. We also had a similar case as you had with a rapid renal function decline. The severity of stenosis is not a good indicator, but rapid renal decline is a good indicator.

P.B. Mark: We don’t look hard for renal artery stenosis beyond the clinical diagnosis. In the post-ASTRAL and post-CORAL age, we don’t pursue renal artery stenting as aggressively as we did in the late 1990s and 2000s. I don’t think we perform >=5 or 10 a year. We used to do many more than this.

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Disclosures
None.

References
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肾血管性高血压

置入支架或者不置入支架？

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蒋雄京 审校

病例介绍

患者，女，69岁。有吸烟史，为评价其高血压和肾功能下降的程度被转诊至肾病门诊。转诊时，患者血清肌酐水平为241 μmol/L，诊室血压为191/100 mm Hg。她的全科医生之前对她进行过24小时动态血压监测，并没有发现白天有高血压的证据。该患者正服用4种抗高血压药物（硝苯地平长效制剂60 mg/d，坎地沙坦32 mg/d，氢氯噻嗪2.5 mg/d）。转诊前一年，最后一次记录的血清肌酐水平为110 μmol/L。体格检查无异常，尿检中血和蛋白结果为阴性。

E.L. Schiffrin：从病史上看，这是一位肾功能受损但无蛋白尿的吸烟者，我认为这容易引起怀疑，我想你应该对此进行说明。

P.B. Mark：没错。诊断很清楚，这很可能是一例动脉粥样硬化性肾动脉疾病患者。我认为，对于这一点没有争议。如果患者有蛋白尿，其诊断可能为某种肾小球性肾炎。那天我们刚好进行了超声检查。这并不明确诊断的理性检查。

G.L. Jennings：有腹部杂音吗？你能对腹部杂音的用处发表评论吗？

P.B. Mark：我记得检查过这位女士，没有腹部杂音。我也听了腹动脉杂音。她没有腹部杂音或股动脉杂音。

肾静脉超声检查发现，肾脏不对称，左肾大小8.1 cm，无皮质组织。右肾大小11 cm，看似正常。有吸烟史，肾功能受损，难治性高血压，超声示肾脏不对称，以上表现高度提示肾动脉疾病。

E.L. Schiffrin：你之前提到已有肾动脉狭窄，那么我想问，没有吸烟或糖尿病或严重弥漫性肾动脉硬化症其他病因的情况下，你是否看到了动脉粥样硬化性肾动脉狭窄？

P.B. Mark：通常我会说不，但是，我刚刚诊过一位相似的病例，这个病例中我看到了肾动脉狭窄。我认为，他们可能需要更多的血脂检查。一位40岁的不吸烟者因动脉粥样硬化性肾动脉疾病转诊，这让很惊讶。

她的转诊医生辩称进一步的影像学检查是否能改变其治疗。在右肾大小正常、皮质保留的情况下肾功能快速下降，提出了关键性右肾动脉狭窄治疗的可能性。我们考虑采用磁共振血管造影、计算机断层扫描血管造影，以及侵人性肾血管造影等影像学方法来评价肾动脉狭窄。肾动脉受损（估算的肾小球滤过率为18 mL/min/1.73 m²），是磁共振血管造影的相对禁忌证，因为有肾脏全纤维化的危险。

因此，预水化和计算机断层扫描血管造影是非侵人性成像的首选。计算机断层扫描血管造影证实，右肾动脉开口存在严重的钙化性狭窄（箭头所示），以及主动脉中度钙化和左肾萎缩（图1）。

肾动脉支架介入治疗及预后

根据进行性肾功能下降，难治性高血压，单功能肾存

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在关键性狭窄，我们选择进行肾动脉介入。该患者进行了肾动
脉CO₂血管造影、右肾动脉血管成形和支架置入术，未并发
症发生（图2）。

术后数天，患者肾功能快速恢复正常，血压显著改善，
肌酐水平在术后9个月下降至92 μmol/L。最近一次在门诊诊
到她是术后18个月，患者无服用药物（比索洛尔和硝苯地平），诊
查血压控制良好（148/88 mm Hg），血清肌酐水平为122 
μmol/L。(估算的肾小球滤过率40 mL/min/1.73 m²)。

虽然患者至今没有再进行过影像学检查[3]，术后18个
月时肾功能轻度下降提示可能存在迟发支架内再狭窄。尽
管肾功能出现轻微下降，很清楚该病例采用肾动脉成形加
支架置入对动脉粥样硬化性肾动脉狭窄的短至中期内预后良
好。我们仍将继续努力，关注患者动脉粥样硬化的其他危险
因素，包括妊娠、治疗血脂异常、优化血压控制，尽力保护
单功能肾的功能。

A.F. Dominiczak：您想要再次进行影像学检查，并且
可能准备再次球囊扩张吗？

P.B. Mark：这正是我们想做的。然而该患者很不愿意。
有吸烟史和服用多种药物，那是她的选择。我们想再次进行
影像学检查。我们与放射科医师有一些争议，因为支架可能
影响无创影像检查显示狭窄，直接血管造影可能更好。

J-G. Wang：你需要使用超声成像来观察肾脏的变化，
以及肾脏的大小。该检查将阐明病变是否可逆。

P.B. Mark：是的，我认为这很有道理。如果肾脏变小，或
者皮质髓分界不明显，那么可以说，治愈的可能性越来越小。

E.L. Schiffin；你劝她戒烟成功了吗？

P.B. Mark：我们一直在努力劝她戒烟。但答案是
没有成功。这让你不仅怀疑所有这些治疗的效果，而且也将
患者暴露于手术风险中。

在近期一些临床试验大背景下本例的成功结果

这一成功结果与近期良好实施，引入住的比较肾
动脉血管成形加支架置入术和最佳药物治疗的随机对
照试验形成了对比。血管成形与支架置入术治疗肾动脉
病变试验（Angioplasty and Stenting for Renal Artery
Lesions, ASTRAL）[4]，肾动脉粥样硬化性病变血管预后
试验（Cardiovascular Outcomes in Renal Atherosclerotic
Lesions CORAL）[5]，支架置入、降压及降脂预防肾动脉粥
样硬化性开口狭窄所致肾功能障碍进展试验（Stent Place-
ment and Blood Pressure and Lipid-Lowering for the Preven-
tion of Progression of Renal Dysfunction Caused by Atheroscler-
ostic Ostial Stenosis of the Renal Artery, STAR）[6]，均未能证明肾
动脉支架置入相对于药物治疗有更多获益，包括患者生存、
心血管事件、肾功能或血压。ASTRAL是一项全球性研究，
纳入了格拉斯哥的一些患者，研究将806例单侧或双侧动脉
粥样硬化性肾动脉狭窄的患者随机分入支架置入组或药物
治疗组，结果显示介入治疗或药物治疗的两组在血压、肾功
能或终末期肾病进展方面均无差异。肾动脉支架置入并
不是没有风险，在ASTRAL研究中观察到，2.3%的患者出现与
肾血运重建直接相关的严重不良事件，包括死亡和截肢手
术[6]。比较肾动脉支架置入（64例患者）与药物治疗（76例患
者）结果（STAR）这一小型试验表明，两组的肾功能障碍进
展无明显差异[6]。近期，北美CORAL试验将947例合并高血
压或慢性肾病的动脉粥样硬化性肾动脉狭窄患者随机分配
接受支架置入或药物治疗，中间43个月的随访中，对于心
血管或肾脏原因的死亡、心肌梗死、卒中、充血性心力衰竭、
进展性慢性肾病或终末期肾病组成的复合终点，支架置入组
或药物治疗组之间无明显差异[6]。

M. De Buyzere：这位患者适合支架置入术吗？

P.B. Mark：我们得到了较好的结果。这些年来我们作
了大量的支架置入术，有时结果很差。我们会争论，如果有个
病例能从中获益，那这例就是。我确实同意你的说法。该例患

图1. 计算机断层扫描血管造影显示，右肾动脉开口部狭窄。

图2. CO₂血管造影证实，右肾动脉成形和支架置入术之前存在
右肾动脉狭窄。
者确实存在明显的病损，即，肾功能较差，肾脏相对较小，以及其他患者相关的因素。

G.L. Jennings：你所指的，动脉粥样硬化性肾动脉狭窄的主流试验结果已经显示，介入相对于药物治疗并无获益。因此，这就产生了一个问题，如果在后退一点，因为药物治疗毕竟是指南推荐的方法，你可能同意患者也应该保留治疗吗？该病例表明确实有部分患者可以自支架置入术中获益。多数临床医师认为，对于肾动脉狭窄患者，反复发作或过性肾小球滤过率小数，肾动脉重建可能不会使其直接获益。小肾、萎缩的肾脏受到了不可逆的缺血损害，不应期待手术与血运重建可改善其功能。对于合并心力衰竭的肾动脉疾病患者，仍然存在不确定性[9]。上述这些出自的临床试验中招募的患者不能代表每一种临床情形，并且，与本病例病情相似的一些高危患者，也不可避免地遇到特殊的诊断挑战，所以不可能有大量这类患者进入这些随机试验[10]。通过测量血流储备分数，更好地了解肾动脉病变的特征可能有助于改善[11]。另外，针对慢性肾衰竭的肾红细胞进行功能评估，显示肾红细胞可能从血运重建中获益[11]。对于某些精选病例，这些临床试验的结果应该不会妨碍临床医师考虑肾动脉介入治疗。这类病例介入治疗可能获益，或者在介入治疗过程中，快速进展至终末期肾病的可能性超过了支架置入的风险。

**肾动脉狭窄热点总结讨论**

A.F. Dominiczak：我们可以回到肾动脉狭窄及闭塞的画面中吗？有非常微小的血流量，血管接近于闭塞。如果我们什么也不做，接下来会发生什么？好吧，我有一位同样病情的患者。这是我今年以前在血管门诊接诊的第一位患者，我们与Chris Isley教授之一研究以上的这篇论文。这位患者之前出现了无尿症，在相同的年龄，一切都很相似。她患了恶性高血压，她完全依赖于1个几近闭塞的肾动脉。因此，这是一例单肾的严重狭窄，接下来就是透析。批判这样的治疗是很容易的，但是，两年之后该患者并不需要肾脏替代治疗，所以，还是有一些收获。

A. Brady (格拉斯哥)：ASTRAL研究中有很多病例来自我们系列，不知道你们如何看待招募患者的人而，类似上述这类狭窄的患者，可能永远不会被纳入研究。他们去做血管成形术了。对于肾动脉狭窄达到50%～60%的所有患者，即使你不同意他们是否适合入选，他们仍然会进入试验。我并不能代表CORAL。但是，我被告知许多CORAL中心存在的这种情况，这些中心主要在北方，对于真正严重狭窄的患者从来不纳入研究。因此，上述试验事实上并没有告诉我们有关关键性狭窄的结果。我认为，对于这类患者而介人治疗获益是明确的。

J. Dawson (格拉斯哥)：我同意你的观点。我本来应该推荐那例患者行支架置入术。如果我们能够达到过去，即使有目前这样的临床试验数据，我仍然会将患者转诊行支架置入术。我想更有趣的问题是，既然患者的肾功能已经下降，现在我们能做什么？我更想确认的问题是：计算的肾小球滤过率能恢复多少，滤过率改善及其之后的变化是因而在围手术期使用血管紧张素受体拮抗剂，接着能重新开始，或者在整个过程保持治疗不变？

P.B. Mark：治疗并不一样。我不知道患者每一个变化的精确幅度，但是，在置入支架后的那天，患者血压下降。由于大量的失钠排泄和多尿，血压下降。患者停止了所有降压药物，随后出现不稳定的演变情况，这使得我们极难重新解释恢复用药发生了什么。但是，我们没有重新使用血管紧张素受体拮抗剂，因为没有其他强效的降压药物这样这样做。我只是认为重新使用任何抗高血压药物可能导致肾脏灌注再次出现相对下降。我们不知道她确切的基线情况。

G.L. Jennings：只评论一点：我们确实不反对真正的严重狭窄患者没有参加这些试验。CORAL研究的回顾性分析表明，那些狭窄超过80%的患者并没有显示出与狭窄<80%的患者有任何不同。要使功能有显著差异，可能需要狭窄达到80%。

M. De Buyzere：对于功能肾的保留，其切点值的处理你有什么建议吗？例如压力导丝。

P.B. Mark：我不知道。对于ASTRAL试验，我并不记得确切的入组标准，但是，狭窄程度约在50%～70%，这并不是非常严重的狭窄。我们没有压力导丝方面的经验。我们有一些建议性实验，开展过磁共振灌注肾灌注检查，检查结果由Manchester研究小组发表[12]。在预测肾血流重建的反应方面，它确实令人印象深刻，但是，我们没有足够的病例样本。实际上我认为，应该再次回到声学检查。如果他们的皮质大小合适，肾脏大小合适，那么可以考虑，他们的结果比较好。如果肾径9 cm或更小，则不太可能有这样的结果。这位患者如果较小11 cm，如果肾径大小10 cm呢？我们认为，这很难预测，还需要更多的数据。

M. Walters (格拉斯哥)：我准备检视安娜大夫前面提到的那个观点，这个观点没有愚蠢的问题。既然患者的肾功能在支架置入术后9个月开始恶化，那么问题是否有关即时再狭窄。无对比剂成像有什么作用，或者说有潜在的作用吗？例如，使用时间飞跃法磁共振血管成像（TOF-MRA），可以避免对比剂诱发的对患者的损害危险，但是，这能够回答有关有无支架内再狭窄的具体问题吗？

P.B. Mark：我肯定那不是个愚蠢的问题。只是我无法理解。严厉地说，我认为，磁共振血管造影，时间飞跃法成像是一个非常好的概念。但是，与狭窄本身相关的磁共振成像的伪影使这变得困难。我没有任何这方面的经验。
Mark et al. Renovascular Hypertension: To Stent or Not to Stent

2. Collidge TA, Thomson PC, Mark PB, Traynor JP, Jardine AG, Morris ST, None.


参考文献

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声明

无。