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. 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.

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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. 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.
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), Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL), 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) 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. 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. 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.

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% to 60% 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. This was a 11 cm kidney; what if it had been a 10 cm kidney? I think it is difficult and that is where more data are required.

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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蔣雄京 审校

病例介紹

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。她的全科医师之前对她进行过24小时动态血压监测, 并没有发现白大衣高血压的证据。该患者正服用4种抗高血压药物 (硝苯地平长效剂型60 mg/d, 坎地沙坦32 mg/d, 比索洛尔10 mg/d, 贝那普利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患者。我认为这容易引起怀疑, 你认为你应该如何对此进行说明?

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病。对于这一诊断没有任何争议。如果患者有蛋白尿, 那要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。这并不是明确诊断的直接检查。

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。我也听了股动脉杂音。她没有腹部杂音或股动脉杂音。

肾脏超声检查发现, 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, 超声显示肾脏不对称, 以上表明高度提示肾血管疾病。

E.L. Schiffrin: 你之前提到已有肾动脉狭窄, 那么我想问, 在没有吸烟或糖尿病、或严重弥漫性动脉硬化的其他病因的情况下, 你是否考虑了动脉粥样硬化性肾动脉狭窄?

P.B. Mark: 通常我会说不, 但是, 我刚刚接诊过一位相似的病例, 在一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, 这让人惊讶。

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。我们考虑采用频共振血管成像、计算机断层扫描血管成像, 以及侵人性血管造影等影像学方法来评价肾动脉狭窄。肾动脉受损 (估计的肾小球滤过率为18 mL/min/1.73 m²), 是磁共振血管成像的相对禁忌证, 因为有肾原性全身纤维化的危险。因此, 预水化和计算机断层扫描血管成像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)。

腎动脉支架介入治疗及预后

根据进行性肾功能下降, 危害性高血压, 单功能肾存

Hypertension is available at http://hyper.ahajournals.org

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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，无并发症发展(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。最近一次在门诊时看到她是事后18个月，患者正服用一种药物(比索洛尔和硝苯地平)，血压控制良好(148/88 mm Hg)，血清肌酐水平为122 μmol/L。估计的肾小球滤过率为40 mL/min/1.73 m²。

虽然患者至今没有进行过影像学检查[5]，术后18个月肾功能轻度下降提示可能存在中段支架内再狭窄。尽管肾功能出现轻微下降，但证实该病例采用肾动脉成形支架植入对动脉粥样硬化性肾动脉狭窄的短至中期预后良好。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，包括戒烟，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：你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，是他的选择。我们想再次进行影像学检查。我们与放射科医生有一些争议，因为支架可能影响非创伤性检查显示狭窄，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。这项检查将表明病变是否可逆。

P.B. Mark：是的，我认为这很有道理。如果肾脏变小，或者皮质与髓质分界不好，那么可以说明，治愈的可能性越来越小。

E.L. Schiffirin：你劝她戒烟成功了吗？

P.B. Mark：我们一直非常努力地劝她戒烟。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，引人注目的比较肾动脉血管成形和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双盲临床试验(Angioplasty and Stenting for Renal Artery Lesions, ASTRAL)[9]，肾动脉粥样硬化性病变血管预后试验(Cardiovascular Outcomes in Renal Atherosclerotic Lesions CORAL)[9]，支架置入，降压和降脂治疗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 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)[9]，均未能证明肾动脉支架置入相对于药物治疗有更多的益处，包括患者生存、心血管事件，肾功能或血流。ASTRAL是一项全球性研究，纳入了格拉斯哥的一半患者，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[9]。较为肾动脉支架置入(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[9]。近期，北美CORAL试验将94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，在中位43个月的随访中，对于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间无明显差异[9]。

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，如果有病例能从中获益，那这例就是。我确实同意你的说法。该例患
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：如你所述，动脉粥样硬化性肾动脉狭窄的主症实验结果已经显示，介入相对于药物治疗并无获益。因此，这就产生了一个问题，如果在最后一点，即药物治疗毕竟是治疗推荐的方法，你认为此患者也该如此治疗吗？该病例表明确有部分患者可以从支架置入术中获益。多数临床医师认为，对于肾动脉狭窄患者，反复发作或一过性血尿伴左肾部收缩功能保留是行肾动脉成形术支架置入术的适应症[7]。对于服用多种血浆药物控制良好，肾功能稳定的患者，肾动脉血运重建可能不会使其获益。小肾、萎缩的肾脏受到了不可逆的缺血损害，不应期待经过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[8]。上述这些报告的临床实验中招募的患者不能代表每一种临床情形，并且，与本病例病情相仿的一些高危患者，也不允许避免机会遇到特殊的情况下。所以我们不可能有大量这类患者进入这些临床试验[9]。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帮助[10]。另外，对患者肾功能的肾组织进行功能评估，显示肾功能组织可从血运重建中获益[11]。对某些精选病例，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，或者在介入治疗的情况下，快速进展至终末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？有非常微薄的血流量，血管接近于闭塞。如果我们什么都不做，接下来将会发生什么？好吧，我有一位同样病情的患者。这是我们2005年在高血压门诊接诊的最后一位患者，我们与Chris Isles教授一起发布了这篇论文。这位患者在这次研究中出现了尿失禁，在同样的年龄，一切都很相似。她患了恶性高血压。她完全依赖于1个近闭塞的肾动脉。因此，这是一例单侧的严重狭窄，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替代治疗，所以说，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。就我们招募患者而言，类似上述这种狭窄的患者，可能永远不会被纳入试验。他们去做血管成形术了。对于肾动脉狭窄达到50%~60%的患者，即使你不同意他们是否适合入选，但他们会仍然会进入试验。我并不能代表CORAL，但是，我敢打赌许多CORAL中心存在这样的情况，这些中心主要在北美，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随机性狭窄的结果。我认为，对于这例患者而言介入治疗获益是明确的。

J. Dawson（格拉斯哥）：我赞同你的观点。我本来应该推荐这名患者行支架置入术，如果我们能够回到过去，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：估算的肾小球滤过率能否恢复多少，滤过率改善及其之后的变化是因而在无静脉内服用血管紧张素受体拮抗剂，接着能重新开始，或者在整过程中保持治疗不变？

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，在支架置入后的那天，患者血压下降。由于大量的尿量排泄和多尿，血压下降。患者停用了所有降压药物，随后出现不稳定的情况，这使得我们需要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，因为没有其他强制性的原因需要这样做。只是我认为，重新使用任何抗血管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：只评论一下，我们确实不知道真正的严重狭窄患者没参加这些试验。CORAL研究所作的回顾性亚组分析表明，那些狭窄超过80%的患者没有显示出与狭窄<80%的患者有任何不同。要使用其有实质性差异，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，我记不清确切的入组标准，但是，狭窄程度大约在50%~70%。这并不是非常严重的狭窄。我们没有研究方面的选择。我们有一些实验室数据，开展了磁共振灌注性影像检查，结果已由Manchester研究小组发表[12]。在预测肾动脉重建反应方面，其确实令人印象深刻，但是，我们没有任何的病例样本。实际上我认为，应该再次回到透析检查。如果他们的皮质厚度合适，肾脏大小也不合适，那么可以认为，他们的结果较好。如果肾实质9 cm或更小，则不要太有好的结果。对于这位患者的肾实质大小11 cm，如果肾实质大小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）：我准备检验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的9个月开始恶化，那么问题是否只有即刻再狭窄。无对比剂成像发挥作用，或者有潜在的作用吗？例如，使用时间飞跃法磁共振血管成像（TOFMRA），可以避免对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。严肃地说，我认为，磁共振血管造影，时间飞跃法成像，这种非常好的概念。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。
J.A. Staessen: 为什么你要为这位患者再次做成像检查呢？你发现，你发现支架内有血栓形成。你会怎么做？

P.B. Mark: 我不是血栓形成，因为那句话，肾功能将会变得相当差。我想，这是一个非常难以回答的问题。

我们发现患者有非常明显的支架内再狭窄，那么我们想要让患者再做一次手术，这不是又回到在原地了吗？

E.L. Schiffirin: 如果我们正在花费或准备花费这么多的钱在患者身上，那么，我们为什么不能花费更多钱让患者戒烟呢？毫无疑问，自从近期的干预之后，吸烟的危害已经发生，并且导致了更多血管的损害。

J.-G. Wang: 你看到这类患者的频率是多少？如果你很少看到这类患者，一年一例或两例，我认为，这不是个问题，我们也看到过一例相似的病例，患者的肾功能快速下降，狭窄的严重程度不是一个很好的提示指标，但是，肾功能快速下降是一个很好的提示指标。

P.B. Mark: 除临床诊断外，我们没有着重关注肾动脉狭窄。在ASTRAL和CORAL时代，我们并不像20世纪90年代后期和21世纪初那样积极开展肾动脉支架置入术。我认为，我们一年完成不了5-10例。我们之前做得比这多得多。

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声明

无。