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# Renal Artery Stenosis: Diagnosis and Clinical Management

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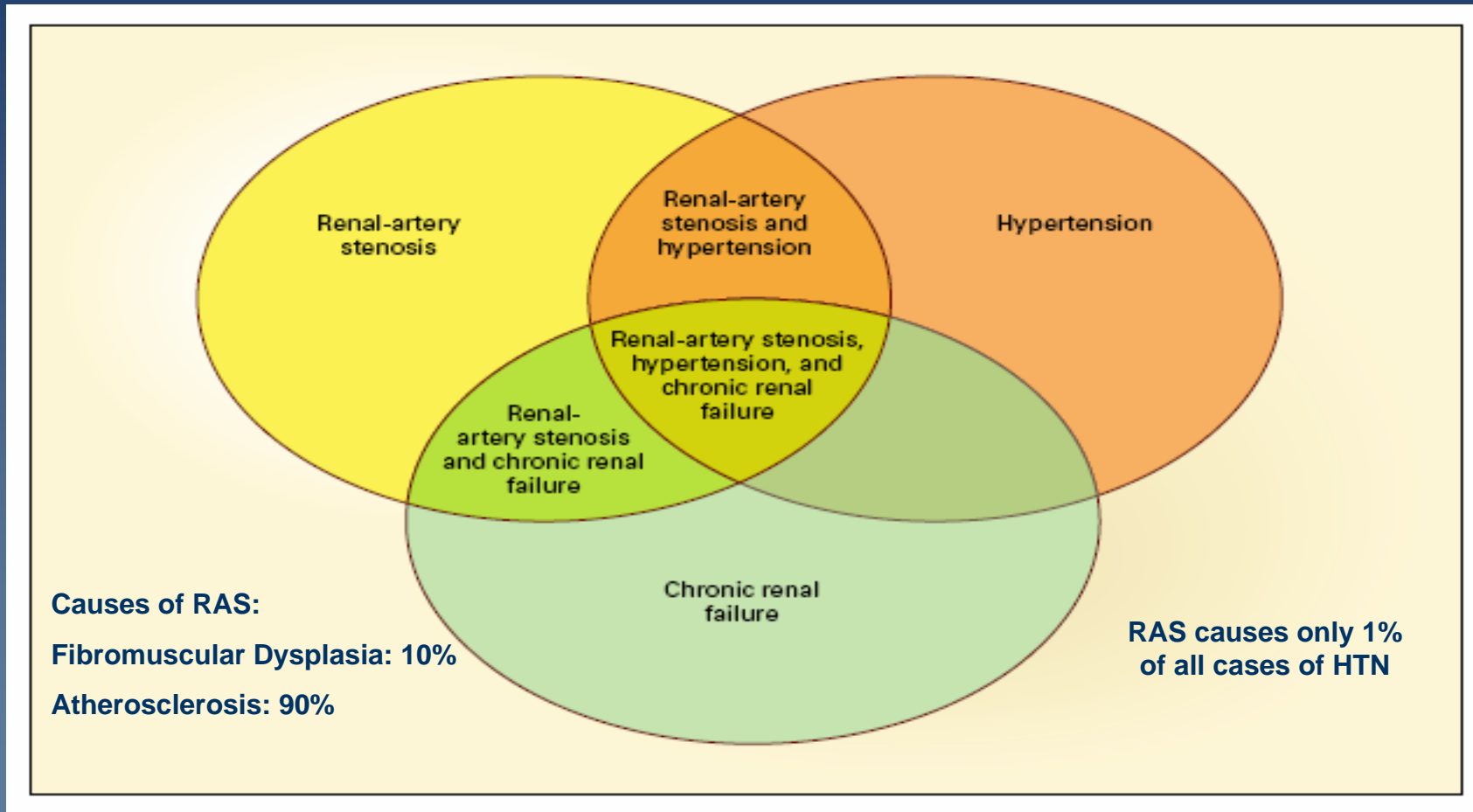
# GOALS

- ◆ Understand Renal Artery Stenosis (RAS) as an important sequela of atherosclerosis
- ◆ List three reasons to clinically suspect RAS
- ◆ Be familiar with the most up-to-date imaging and diagnostic modalities in RAS
- ◆ Consider the debate regarding medical vs. interventional management of RAS



## RAS Introduction: Part 1 of 3

RAS can stand alone or it can exacerbate other disease processes, particularly HTN and renal failure

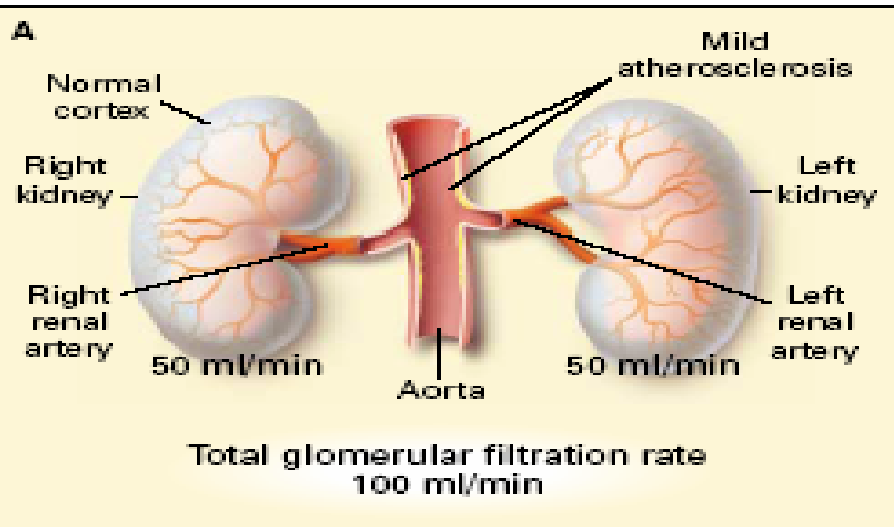




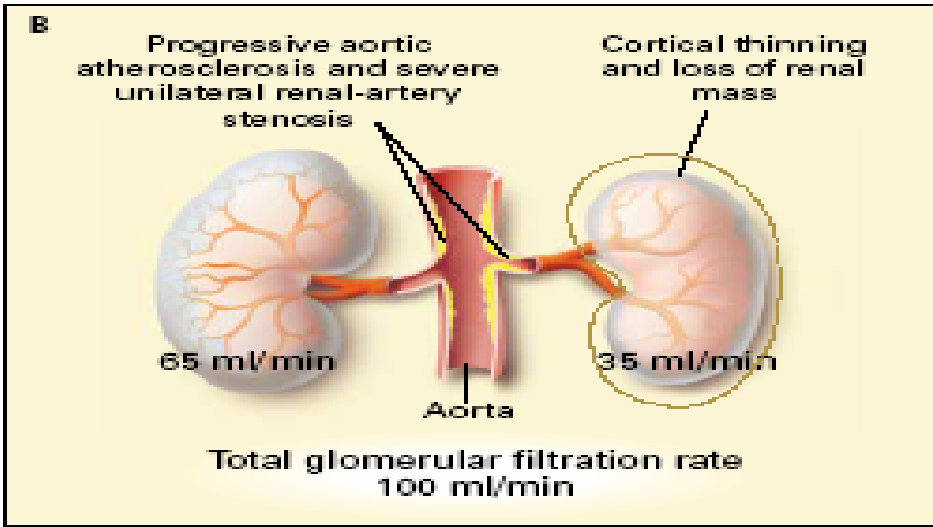
# RAS Introduction: Part 2 of 3

## RAS can lead to renal failure

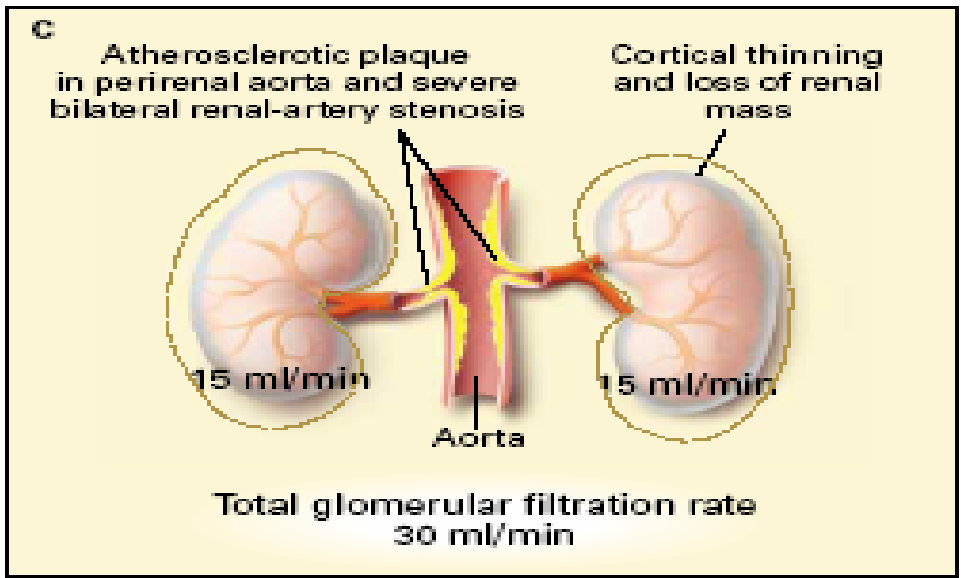
### Early Disease



### Progressive Disease



### Advanced Disease



Safian et al NEJM 344: 6  
See previous slide



## RAS Introduction: Part 3 of 3

# RAS can complicate pre-existing HTN

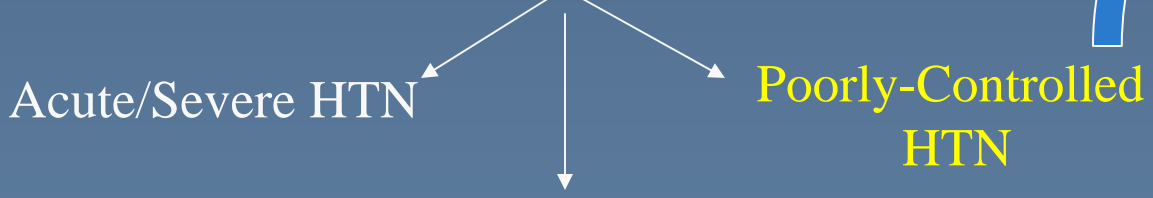
Atherosclerotic Disease  
of Renal Artery (represents 90% of RAS)



Renin-Angiotensin System Activated  
Increased Vascular Oxidative Stress



Renovascular Hypertension



Acute/Severe HTN

Well-Controlled HTN  
(NB: Most patients with HTN have ESSENTIAL HTN. And most of the time, it responds to medical management)

Poorly-Controlled HTN



In this presentation, we're concerned with patients for whom RAS is complicating the management of their HTN



# RAS: A Key Player in Refractory HTN

- ◆ Mild Hypertension: RAS causes 1% of cases
- ◆ Acute, Severe, Refractory HTN: RAS causes 10-40% of cases
- ◆ In a study of 300 patients with well-controlled HTN:
  - 53% had normal renal arteries
  - 28% had <50% stenosis of one renal artery
  - 19% had >50% stenosis of one renal artery
  - 7% had >70% stenosis of one renal artery
  - 3% had bilateral disease



# Meet Our Patient

## ◆ Mr. W.R.

54 year-old male

PMH: HTN

Meds: ASA, Plavix, Norvasc (10 mg qd), Diovan (160 mg qd), Toprol XL (50 mg qd)

Former smoker

FH: DM

**BP: 160-170 systolic**

BUN/Cr: 1.1-1.2 mg/dl

Physical Exam: **an abdominal bruit** is appreciated

## ◆ Should we suspect RAS?



# Clinical Findings of RAS

## ◆ Hypertension

- refractory to therapy with 3 or more drugs
- abrupt onset before age 50– likely fibromuscular dysplasia
- abrupt onset after age 50– likely atherosclerotic RAS
- malignant HTN

## ◆ Renal Abnormalities

- unexplained rise in BUN (azotemia)
- unilateral small kidney
- unexplained hypokalemia

## ◆ Other Findings

- unexplained CHF or acute pulmonary edema
- abdominal bruit, flank bruit, or both
- severe retinopathy
- peripheral or coronary vascular disease





# If you suspect it clinically, how should you evaluate RAS?

- ◆ In choosing assessment and imaging modalities, consider the questions you hope to answer with the study



# Physiologic/Perfusion Studies

- ◆ Guiding questions:
  - What is overall kidney function?
  - Is the lesion hemodynamically significant?
  
- ◆ Tests
  - Activity of renin-angiotensin system (renin levels, etc.)
  - Serum creatinine
  - Urinalysis
  - Nuclear Medicine: Captopril Renography and Technetium-labeled DPTA
    - ◆ What is the differential flow velocity across each artery? What is the GFR in each kidney?
  
- ◆ Drawbacks:
  - lack of specificity (e.g. many disease processes cause increased creatinine)
  - low predictive accuracy (i.e. studies do not indicate if re-vascularization would change clinical picture)



# Vascular Studies

Guiding Question: What is the anatomic status of the lesion?

## ◆ Gold Standard:

Contrast-Enhanced Angiography

## ◆ Non-Invasive Studies:

– Duplex Ultrasonography

– MR Angiography

– CT Angiography (not discussed here, but increasingly popular)



# Contrast-Enhanced Angiography

- ◆ Gold Standard, but no longer a diagnostic study– used for intervention
- ◆ Concerns: complications from procedure and contrast
  - consider use of gadolinium and visipaque as contrast agents in cases of renal insufficiency

plaque  
causing  
stenosis in  
left main  
renal artery–  
note catheter  
in the  
abdominal  
aorta





# Duplex Sonography

- ◆ Inexpensive but significant drawbacks
  - Operator-dependent
  - Time-consuming
  - Sensitivity: 81%
  - Specificity: 87%
- ◆ Yields important functional information
  - Hemodynamic information (what is the flow to each kidney?)
  - Predictive value with respect to revascularization (is lesion impeding flow?)

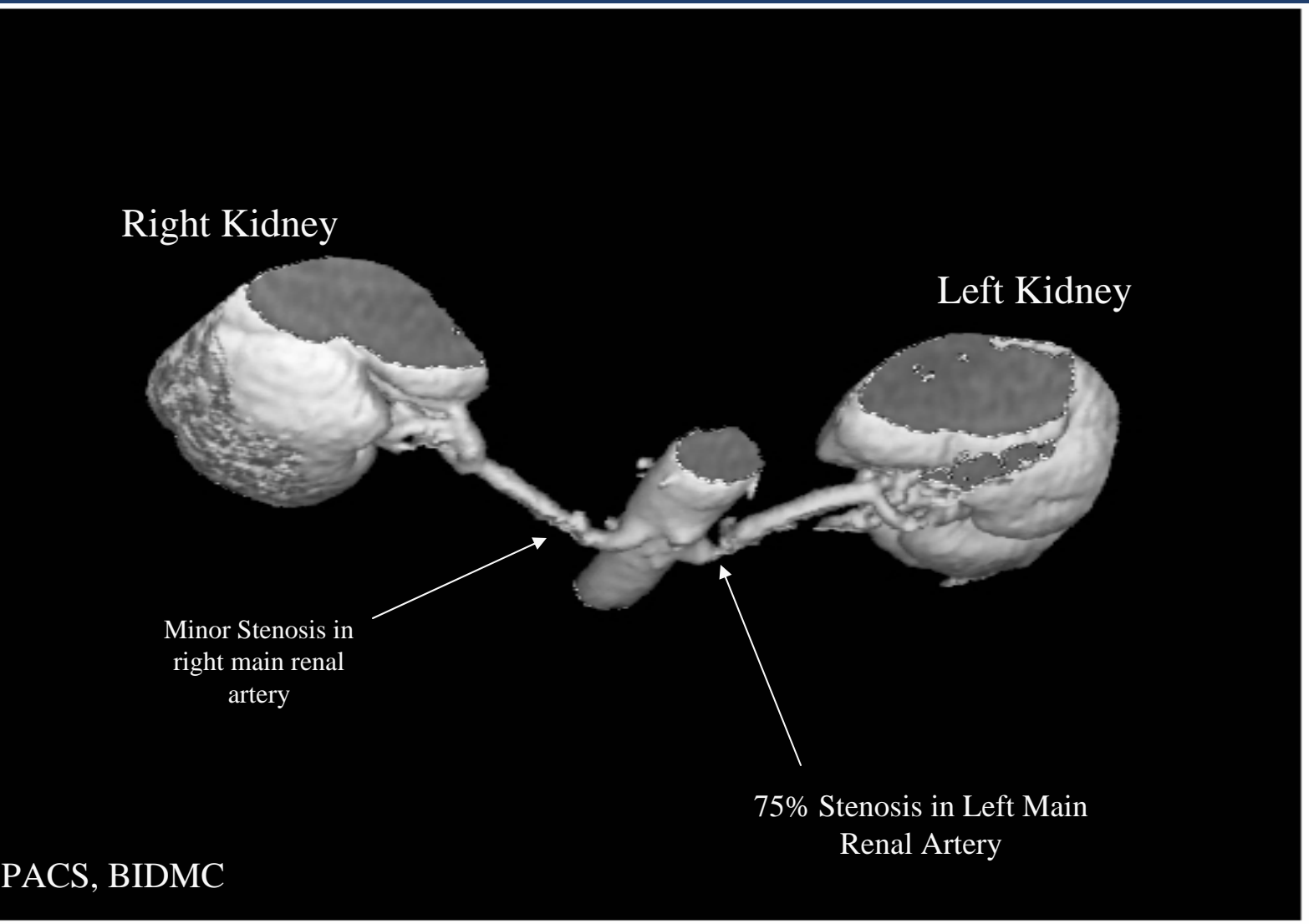


# MR Angiography

- ◆ Sensitivity = 97%
- ◆ Specificity = 86%
- ◆ Fast, Operator-independent, 3-D Imaging
- ◆ Some limitations
  - Pacemaker incompatible
  - Inferior spatial resolution to interventional angiography (may over-estimate stenosis)



# Our Patient's MRA





# Patient MRA: A Different View







# Management Dilemmas in RAS

Although our patient presents with significant stenosis and clinical symptoms,

- ◆ **Incidental findings increasingly common**
  - What if RAS is found on abdominal MRA for a different indication in an asymptomatic patient?
- ◆ **Difficult to correlate progression of stenosis with clinical manifestations**
  - What if lesion was 50% this year and 60% the next? Does 10% progression justify intervention?
- ◆ **No good data comparing optimal medical management with revascularization interventions**
- ◆ **What to do?**



# When faced with management dilemmas, consider the risks of disease progression and the goals of intervention

Progression of Atherosclerosis is Significant:

- Certain populations are at greater risk for worsening stenosis
- Although they rarely occlude, stenotic arteries narrow over time
- Ischemic nephropathy associated with significant mortality**
  - Dialysis patients have 50% mortality over 3 years

Clinical consequences of intervention may not be significant:

- Will intervention improve renal function?
- Will intervention change the medical management of the patient?**



# RAS: An Approach to Intervention

- ◆ After assessing via MRA/CTA and before intervention, consider:

- Clinical characteristics
- Baseline renal function
- Degree of asymmetry of flow

IF:

- 1) >50% Unilateral Stenosis  
or 10 mmHg gradient across lesion,
- 2) Bilateral Stenosis,
- or 3) Refractory HTN, Rising Cr,  
Recurrent Pulmonary Edema

→ Intervention

- ◆ No intervention with advanced renal disease or chronic HTN



# Intervention Strategies

## Part 1 of 2

### ◆ Surgical

- specific indications

  - ◆ Fibromuscular Dysplasia (FD)

  - ◆ Patient requires extensive reconstruction of aortic structures

- recent data

  - ◆ 2004 data available with long-term follow-up (11 years)

  - ◆ 93% of those with FD had stable or improved renal function

  - ◆ 71% of those with atherosclerotic disease had stable or improved renal function



# Intervention Strategies

## Part 2 of 2

### ◆ Percutaneous Interventions

#### – Balloon Angioplasty

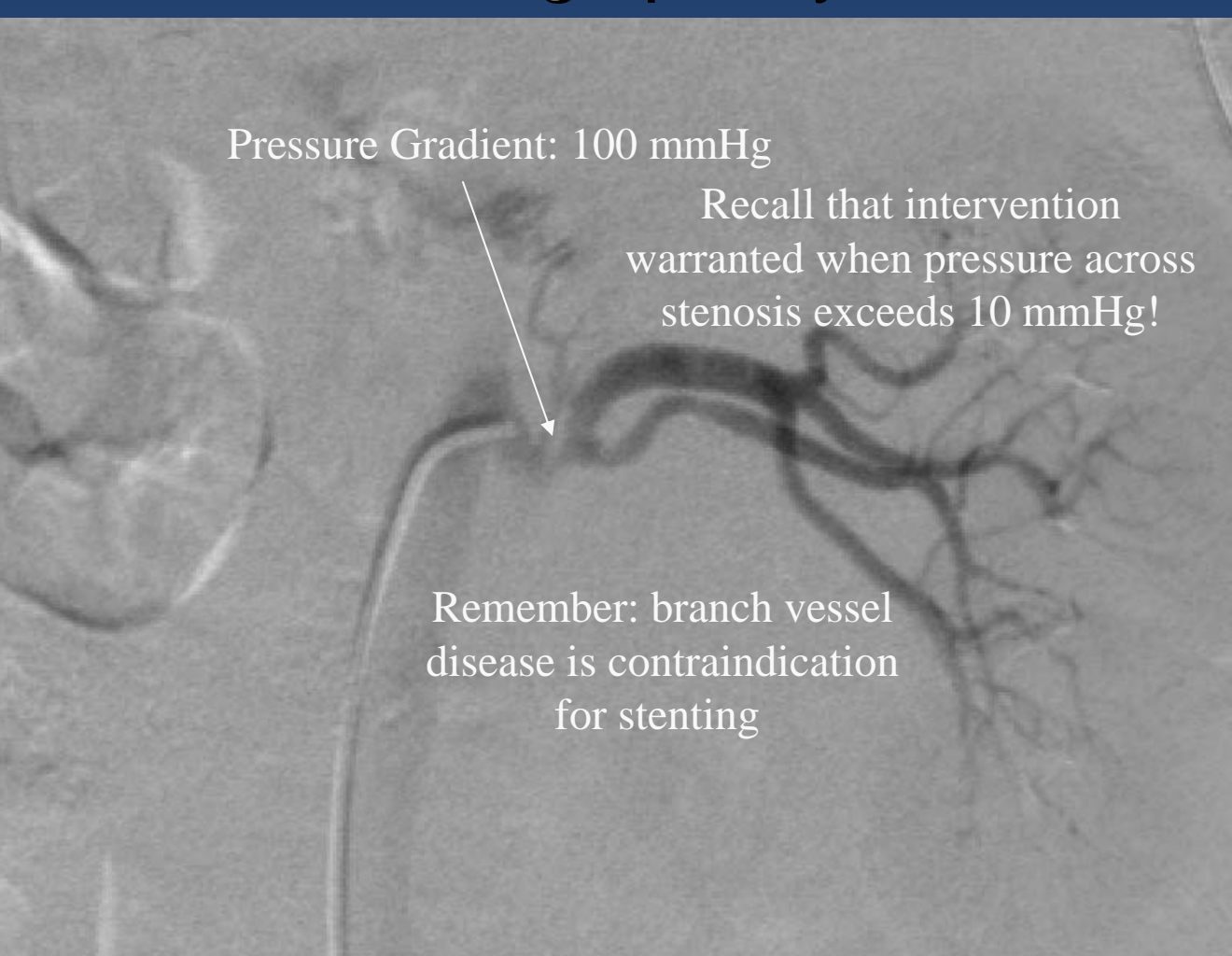
- ◆ technically successful, but procedural risks
  - e.g. distal embolization, artery dissection
- ◆ consider recurrence rates and effects on HTN
  - 15-42 percent re-stenose (inferior to stenting)
  - Unlikely to have long-term correction of HTN

#### – Stenting

- ◆ technically successful, avoids some risks of angioplasty
  - Stenting often repairs damage secondary to angioplasty
- ◆ consider impact on renal function
  - Creatinine decline: 1.7 to 1.3 over four-year follow-up (N=1058)
  - Most lesions: 5-13% re-stenose (superior to angioplasty)
- ◆ consider nature of stenotic lesion
  - No stenting in branch-vessel disease because stenting one vessel risks occlusion of the other



# Which intervention did our patient undergo? Angioplasty



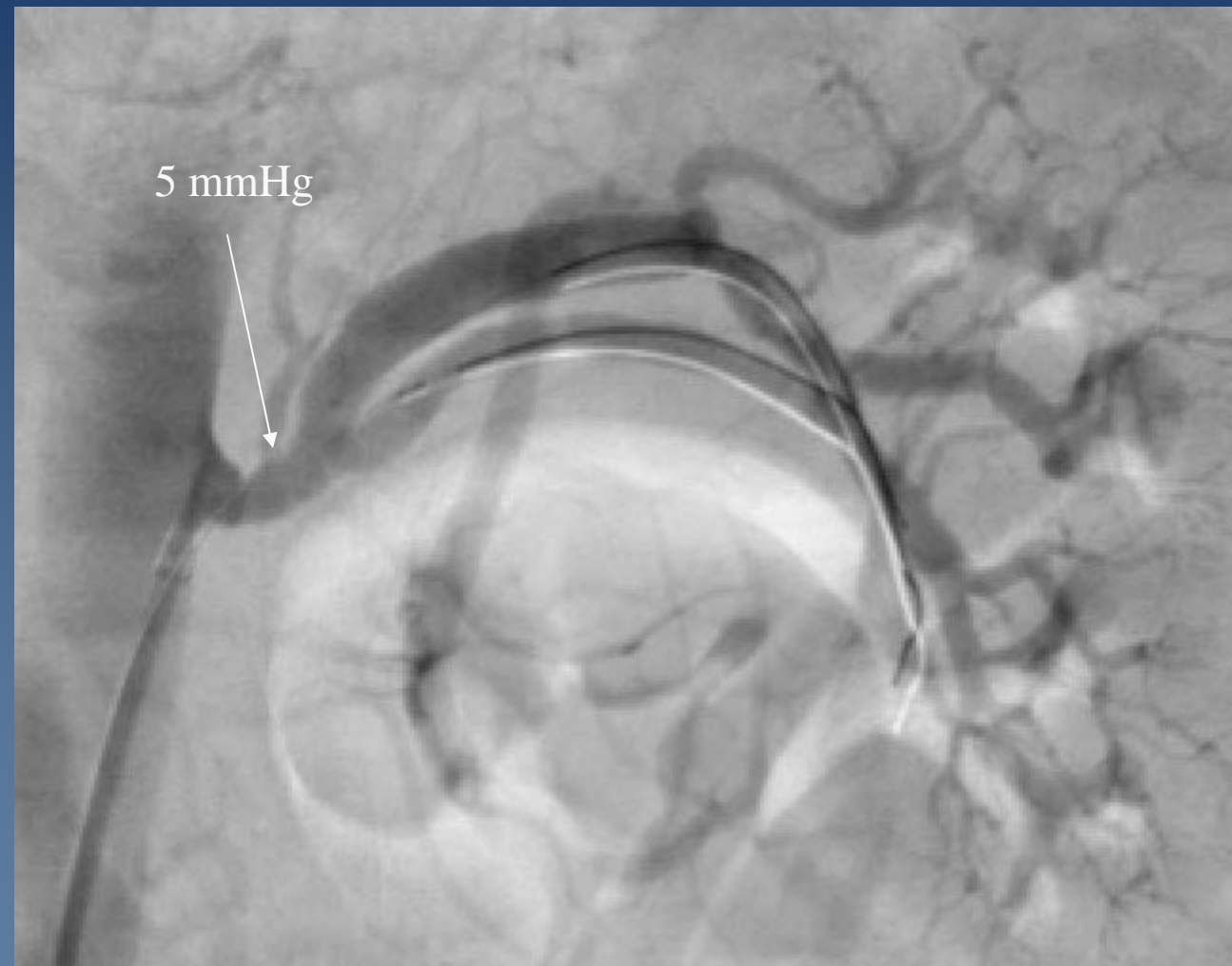




# Our Patient's Angiography After Angioplasty

Post-Procedure:

- BP: 140 systolic
- Cr: 1.1-1.2 mg/dl





# Recall our GOALS

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- ◆ List three reasons to clinically suspect RAS
- ◆ Be familiar with the most up-to-date imaging and diagnostic modalities in RAS
- ◆ Consider the debate regarding medical vs. interventional management of RAS





# References (1 of 3)

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