Box 1 Criteria for a screening programme

- Is the disease an important public health problem?
- Is there an effective treatment for localised disease?
- Are facilities for further diagnosis and treatment available?
- Is there an identifiable latent or early symptomatic stage of disease?
- Is the technique to be used for screening effective?
- Are the tests acceptable to the population?
- Is the natural history of the disease known?
- Is there a strategy for determining which patients should and should not be treated?
- Is the cost of screening acceptable?
- Is effective treatment available and does management of cases in the early stages have a favourable impact on prognosis?

programme. However, the guidelines fail to meet the screening criteria adopted by the World Health Organisation⁹ (box 1) because haemodynamically significant ARAS will be diagnosed without a strong evidence base for management. We would strongly discourage renal artery revascularisation at the time of coronary arteriography until a considerably more robust evidence base is available.

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Commentary: Shoot the renals!

F C Luft, C M Gross

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Atherosclerotic renal artery stenosis (ARAS) is a growing dilemma. The condition is increasingly common and can promulgate hypertension and result in renal failure. However, patients with ARAS generally die owing to their coronaries or cerebral vessels. Intervention, by stenting or angioplasty is beloved and believed, but not proved. The American Heart Association has recently published guidelines regarding patients at high risk for ARAS who are potential candidates for revascularisation. Since this phraseology includes practically every patient with atherosclerosis, these guidelines appear ill advised.

In this issue of *Heart*, Dear, Padfield and Webb¹ take umbrage at recent (2006) recommendations published by the American Heart Association (AHA) regarding new guidelines for “drive-by” renal arteriography during interventions for atherosclerotic lesions elsewhere in the body.² This Practice Guideline was followed by a multispecialty consensus document stating basically the same thing.³ The multispecialty pundits concluded that renal angiography should be

“selectively applied to patients at high risk for atherosclerotic renal artery stenosis (ARAS) who are potential candidates for revascularisation”. Dear *et al* draw attention to the fact that the guidelines recommend screening for ARAS in all patients at risk who are candidates for revascularisation.¹ “At risk” was defined according to hypertension, age of onset, accelerated course, kidney size discrepancy, “flash” pulmonary oedema, worsening renal function and multivessel atherosclerosis elsewhere. Since the “unstentable” patient is yet to be encountered, Dear *et al* suggest that the number of patients subjected to renal artery stenting is bound to increase. They point out that the AHA recommends a generalised “screening programme” for an entity that has no sound basis for management. A recent meta-analysis by Balk *et al* underlines their view.⁴ Have Dear *et al* indeed caught old Auntie AHA with her pantaloons at half mast?

The issues are complex. ARAS is a moving target in an ever-aging, ever-more diabetic population. Furthermore, the options

Abbreviations: AHA, American Heart Association; ARAS, atherosclerotic renal artery stenosis; CORAL, Cardiovascular Outcomes in Renal Atherosclerotic Lesions

Figure 1 (A) Typical left renal artery stenosis angiogram, most likely atherosclerotic disease in this middle-aged woman with atherosclerotic disease elsewhere. (B) Sudden stent occlusion is shown, an uncommon event but nonetheless a real risk. (C) The occlusion could be reopened and the patient did well. (D) A skin biopsy after renal artery stenosis stenting in a different patient showing a small vessel with typical "clefts" denoting cholesterol emboli. The biopsy specimen was taken from the patient's back, far from the expected "downstream" area of the intervention.

offered by medical treatments, such as optimal blood pressure reduction, cholesterol lowering, antiplatelet drugs, and renin-angiotensin-aldosterone system blockade, have improved substantially in recent years.⁵ In parallel, the invasive crowd has also steadily done its homework. Percutaneous interventions have improved dramatically and complications have (probably) decreased.⁶

The risk of renal artery stenting is low but not trivial. Figure 1 shows an angiogram from a middle-aged hypertensive woman with ARAS in her left renal artery (fig 1A). Stenting was uneventful with an excellent visual result (not shown). However, flank pain and an increase in her serum creatinine concentration prompted the second study (fig 1B) showing a stent thrombosis. Additional intervention, coupled with abciximab treatment, resolved the problem (fig 1C), albeit not without morbidity to the patient and soul searching by the authors. Another example is shown in fig 1D. This hypertensive 79-year-old woman with three-vessel coronary disease (raised troponin T), an aortic aneurysm (>5.5 cm), and ARAS underwent intervention. An interesting discussion was, "who goes first here"? The interventionalists put her on the list as requiring fixing of her coronary arteries, placement of an aortic endoprosthesis and stenting of her renal arteries. The three teams reached consensus and the befuddled patient gave her

consent to everything. The ARAS was first and easily stented. Her creatinine doubled and a localised livido reticularis developed on her back, far from the expected arterial run-off. The skin biopsy shows obvious clefts from cholesterol emboli in a small skin artery. Cholesterol embolisation is not a trivial problem and not necessarily a "downstream" event.⁷ We have no reason to believe that her brain arterioles looked any different from those on her back.

"There ain't substitutes for data!" Herein lies our dilemma. Fortunately, there are efforts to get some. One example is the Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL) study. The end points of CORAL are cardiovascular or renal death, stroke, myocardial infarction, admission to hospital for heart failure, doubling of serum creatinine concentration and dialysis dependency. Auntie AHA did at least publish a "pro" and "con" by the CORAL co-principal investigators. Cooper (a "dyed-in-the-wool" and superb interventionalist) presented the arguments for stenting.⁸ Dworkin, a nephrologist (we need say no more), defended the "optimal medical therapy" line.⁹

Our group applied for CORAL membership and was accepted. Our referring colleagues are enthusiastic, but only up to a point. "I think it is great that you are doing this, but not with my patient!" Herein lies the rub. Faith is an incredibly strong force,

but no substitute for data. In our view, only those patients should undergo interventions of their renal arteries that are included in a randomised, controlled, trial of ARAS such as CORAL. Dear and colleagues have done us a service in drawing attention to "Guideline Mayhem".¹

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IMAGES IN CARDIOLOGY

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Inappropriate ICD shocks

A patient was implanted with a guidant single-chamber implantable cardioverter-defibrillator (ICD) for episodes of ventricular tachycardia in March 2005. In September, he consulted the outpatient clinic after a shock was delivered. The detailed circumstances, however, remained unclear. The panel shows a print-out of the episode as recorded by the ICD. While the first two beats are of sinus origin, high-frequency discharges (10 Hz) of increasing amplitude are seen before shock delivery. Note that the tracing is compatible with an external electromagnetic source misinterpreted as an episode of ventricular fibrillation. The following day the patient confessed that the physiotherapist he was visiting had asked him to test electro-acupuncture equipment consisting of two metal wands plugged to a 10 Hz–0.9 V electrical generator. The patient reported some tingling sensations before shock delivery. This device, sold by a German company, warns users about its potential side effects in patients with an ICD or pacemaker.

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