PERCUTANEOUS TRANSLUMINAL ANGIOPLASTY WITH STENTING FOR RENAL ARTERY STENOSIS

To the Editor:

The article “Screening, diagnosis, and treatment of renal artery stenosis by percutaneous transluminal angioplasty with stenting” by Kobo et al. published in IMAJ 2010;12(3):140-3, raises serious questions, both regarding its treatment algorithm and its final conclusions, which are contrary to contemporary recommendations. Its publication in a peer-reviewed journal should have included editorial comments.

The indications for treatment of renal artery stenosis (RAS) are questionable at best. The more established ones are uncontrolled hypertension, deteriorating renal function and flush pulmonary edema. Anatomic stenosis, by itself, is not an indication for this treatment. Current data on the clinical efficiency of stents in RAS are scarce, and controlled trials do not support the use of this procedure. Risk factor modification with medical treatment has an advantage over stent insertion to the renal arteries [1]. The ASTRAL investigators stated “We found substantial risks but no evidence of a worthwhile clinical benefit from revascularization in patients with atherosclerotic renovascular disease” [2].

Many, if not most, patients now being subjected to endovascular stenting of renal arteries show only limited benefits, either regarding blood pressure response or improvement in kidney function [3]. The American Heart Association guidelines state that the usefulness of percutaneous revascularization of an asymptomatic unilateral hemodynamically significant RAS in a viable kidney is not well established and is presently clinically unproven [4].

The work by Kobo et al., published in the March issue of IMAJ, presents a group of patients treated with percutaneous transluminal angioplasty and stenting of the renal arteries, based on angiographic stenosis of 70% alone. The patients were selected from a group undergoing coronary catheterization, yet one of the indications for selective bilateral renal artery injection was the presence of coronary artery disease regardless of the location of the stenosis, the patients’ blood pressure or kidney function.

The discrepancy between the accepted clinical guidelines and the recommendations of Kobo’s group is striking. We believe that patients should be treated according to evidence-based medicine or accepted clinical guidelines and not according to notions. The best approach, as we see it, is the creation of a multidisciplinary team composed of a nephrologist, internist with special interest in blood pressure control, interventional radiologist and a vascular surgeon, who will discuss in detail the indications for treatment for this selected group of patients.

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References
The seeming low prevalence of Asperger syndrome might appear surprising when compared with figures for later born birth cohorts in England. However, a comparison with England for autism suggests there has been a real increase in incidence and prevalence of all autistic conditions internationally since the mid-1980s. Approximately 30% of English autistic conditions are autism and 70% are Asperger syndrome. Baird and co-authors in 2006 [4] established a benchmark for the mid-1990’s prevalence in English child populations. This concerned 56,946 English children aged 9-10 years born in a 2 year period ending no later than 1996. For classic autism Baird et al. provide two estimates:
- 24.8:10,000 (17.6-32.0) for narrow definition autism
- 38.9:10,000 (95% CI 29.9-47.8) for autism

The narrow definition figure meets autism criteria under DSM IV/ICD10, but also on both ADI and ADOS plus clinical judgment (personal communication: Prof. Gillian Baird 20 September 2006).

For all other autistic spectrum conditions (ASCs) Baird et al. 2006 provide an estimate of 77.2:10,000 (52.1-102.3). Baird 2006’s total figure for all autistic conditions (i.e., including classic autism) is 116.1:10,000 (90.4-141.8).

### References

### Capsule
**Inflammation response by neutrophils**

Besides responding to microbial infection, our immune system also plays an important role in responding to sterile injury, for example, during trauma or organ necrosis. In a mouse model of sterile liver inflammation, McDonald et al. used dynamic in vivo imaging to visualize the innate immune response, which is dominated by neutrophils. Neutrophils were rapidly recruited to the site of inflammation through intravascular channels. Adenosine triphosphate generated from necrotic cells at the injury site and the Nlrp3 inflammasome were required for neutrophils to exit the circulation into the vascular endothelium, where they used integrins to adhere. A luminal chemokine gradient guided integrin-dependent, intravascular migration toward the site of injury. Finally, formyl peptides provided a signal to override the chemokine gradient and draw neutrophils into the site of injury.

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### Capsule
**EphB signaling directs peripheral nerve regeneration through Sox2-dependent Schwann cell sorting**

If severed, nerves outside the brain and spinal cord can reconnect and resume functioning. Unexpectedly, the molecular mechanism behind this remarkable ability turns out to involve fibroblasts — a type of cell that helps with wound repair. Alison Lloyd at University College London and her colleagues used fluorescence microscopy to observe how cut nerves mend themselves in rats. They observed fibroblasts at the site of injury make contact with Schwann cells, which surround and protect neurons. Signalling between the two cell types prompted the Schwann cells to clump into tiny cords that guide the regrowth of neurons across the wound. This response, the authors found, is mediated by a protein called SOX2, which is also involved in reprogramming cells to a stem cell-like state.

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“**You can preach a better sermon with your life than with your lips**”

Oliver Goldsmith (1730-1774), Irish writer and physician

“**We think caged birds sing, when indeed they cry**”

John Webster (1580-1634), British Jacobean dramatist