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PTA AND STENTING IN SUPRA-AORTIC ARCH ARTERIES

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Background: Percutaneous transluminal angioplasty (PTA) and stenting is an attractive alternative to surgery in relieving obstructions in the large supra-aortic arteries. This study evaluates the safety and efficacy of PTA and stenting for significant stenosis in the internal carotid artery (ICA), vertebral artery (VA) and subclavian artery (SA).

Methods: From Jan 1996, we performed 38 PTA and stenting procedures (24 ICA, 9 SA and 5 VA) in 34 patients. M/F 26/8. Mean age 69 ± 10 yrs, range 26-80 yrs. 4 patients had more than 1 vessel treated. 88% of the patients were referred from outside our division. All the patients treated were symptomatic, 44% had previous stroke, 38% had TIA in the ipsilateral carotid territory, while 32% had VBI \pm arm claudication. Mean stenosis was $91 \pm 8\%$, range 70-100%. Significant comorbidity included coronary artery disease (CAD) in 25/34 (74%); significant contralateral or multivessel disease in 16/34 (47%). 7 patients (21%) had either renal artery stenosis or renal insufficiency. 23/24 (96%) of the patients who underwent PTA to the ICA failed to meet NASCET inclusion criteria. Patients with ICA or VA stenosis are assessed independently by ≥ 1 neurologist. NIHSS was performed before and 24 hours after the procedure.

Results: Recanalisation was successful in 37/38 (97.3%) vessels. One patient with a totally occluded right ICA and recurrent TIA had an unsuccessful attempt. There was no in-hospital death, MI or need for surgery. The median hospital stay was 2 days. Mean stenosis was reduced to $6 \pm 8\%$ after PTA and stenting. There was one stroke (1/38, 2.6%) in a patient presenting with left hemiparetic TIA. Following rehabilitation, this patient was independent in daily living with mild left arm weakness only. Two patients with critical ICA stenosis experienced generalised convulsion following reperfusion, one at 3 hours after PTA and the other at day 14. Both were discharged uneventfully with no change in NIHSS. Follow-up CT and MRI brain showed no changes compared with baseline. At a mean follow-up of 16 ± 9 months, all patients are alive without further TIA or stroke. Angiographic follow-up (10 ± 4 months) was completed for 23/38 vessels (61%). Restenosis rate was 8.7% (2/23, 1 ICA, 1 SA).

Conclusion: PTA and stenting for large supra-aortic arch arteries is an alternative to surgery in the treatment of ICA, SA and VA stenosis. Even in this cohort of patients with high comorbidity and surgical risk, the complications and long-term results appears to compare favourably to that of surgery or medical treatment.

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Assessment of Regional Systolic and Diastolic Impairment on left ventricular function: A study with Tissue Doppler Imaging on Right Ventricular Pacing.

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Background: Right ventricular apical pacing (RVP) induces an abnormal cardiac activation sequence which may impair regional and global left ventricular (LV) function in long term. The abnormality can be assessed by Tissue Doppler Imaging (TDI), which allows high-amplitude, low-velocity signals of regional myocardial velocities to be quantified.

Methods: 11 patients (M:F=8:3, age= 67 ± 13 yrs) with sinus node disease were paced randomly in either AAI or DDD (atrioventricular delay=100ms) at 10 beats higher than the lower rate. Cardiac Output was measured by pulse Doppler at the aortic outflow tract. Mid and basal septal and lateral ventricular wall motions were studied at the four chamber view by TDI.

Results: a) Activation Sequence: During AAI pacing, LV activation starts from mid cavity (67% at mid lateral and 44% at mid septum), with the basal septum activated last, but is reversed in DDD pacing, at which 63% starts at basal septum, 50% lastly at mid lateral and basal lateral regions. b) Regional systolic/diastolic peaks: Peak E and peak A are higher at AAI pacing, indicating impaired diastolic function during ventricular pacing. c) Cardiac Output: AAI pacing contributes to better cardiac output than DDD pacing.

	Systolic Peak	Peak E	Peak A	Cardiac Output
At AAI	4.21cm/s	4.70cm/s	4.21cm/s	3.23L/min
At DDD	4.02cm/s	4.11cm/s	3.83cm/s	2.59L/min
p-value	0.34	0.01	0.02	0.03

Conclusion: The LV contraction pattern is changed by RVP, together with abnormal regional left ventricular relaxation, resulting in reduction of cardiac output.