postoperative cardiovascular complications including cardiac events and strokes were statistically analysed.

**Results:** Out of the total of 300 patients, 204 had been taking statins on admission while the other 96 had not. Perioperative cardiovascular complications occurred in 10 out of 204 patients taking statins (4.9%), and 11 out of 96 patients (11.5%) not taking statins – a proportional risk reduction of 57% (p < 0.05). 1 patient taking statins (0.5%) suffered from preoperative cardiac event compared with 3 out of 96 (3.1%) in the counterpart – a proportional risk reduction of 84%. 4 patients taking statins (2%) had perioperative strokes in the stain group in contrast to 4 in 96 (4.2%) in the counterpart – a proportional risk reduction of 52%. Differences in the rates of cardiac events and strokes between the two groups of patients are significant (P < 0.05) on the Chi-square test.

**Conclusion:** The results showed statins to be beneficial in preventing cerebrovascular and particularly cardiac complications in the perioperative period of carotid endarterectomy. This is consistent with the developing evidence in literature, and is possibly related to the property of statins in stabilising vulnerable plaques during surgical procedure.

**Improving blood flow 0792**

**Platelet function in patients with end-stage renal failure undergoing haemodialysis**

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**Background:** The onset of thrombosis in the vascular access for dialysis has a significant impact on access survival, quality of life and health care cost. Controversy exists as to the role of platelet activation in vascular access thrombosis. We aimed to assess platelet activity in patients on haemodialysis (HD) compared with healthy volunteers.

**Methods:** Venous blood samples were taken from 55 patients immediately before (baseline) and 30 min after HD and from 72 resting healthy volunteers. Platelet function was assessed by: (1) Ultegra rapid platelet function assay (RPFA), using the agonists thrombin receptor activating peptide (TRAP) and arachidonic acid (ASA), and (2) flow cytometric measurement of P-selectin expression and fibrinogen binding both with and without ex vivo 10 uM ADP stimulation.

**Results:** At baseline, ASA-stimulated platelet aggregation was lower in patients on aspirin (n = 27) compared with volunteers (median (iqr) 471 (82) versus 650 (54), P < 0.001), but not significantly different from volunteers in patients not on aspirin (P > 0.05). TRAP-stimulated platelet aggregation was not significantly different between patients and volunteers. P-selectin expression on unstimulated platelets was lower in patients than volunteers (% positive platelets 0.79 (1.0) versus 1.68 (1.41), P < 0.001). Fibrinogen binding on resting platelets was higher in patients than volunteers [median (IQR) 1.95 (0.99) versus 1.39 (0.75) P < 0.001] but ADP-stimulated fibrinogen binding was lower [40.9 (25.8) versus 50.6 (19.8)]. Following HD, there were no significant changes in ASA aggregation, unstimulated P-selectin or fibrinogen binding. Changes in TRAP-aggregation, stimulated P-selectin and fibrinogen binding occurred post-dialysis. The effects of heparin could contribute to some of the post-HD changes.

**Conclusion:** This study suggests a complex picture of platelet function, but no overall evidence of increased platelet activation in patients on haemodialysis. Although aspirin might be required for its cardioprotective role, it may not aid the prevention of access thrombosis. Other causes and preventative medical therapies for vascular access thrombosis require to be investigated.