**Safety of Chronic Anticoagulation Therapy After Endovascular Abdominal Aneurysm Repair (EVAR)**

De Rango P., Verzini F., Parlani G., Cieri E., Simonte G., Farchioni L., Isernia G., Cao P. Eur J Vasc Endovasc Surg 2014;47:296-303.

**Objective:** Current data supporting the effect of anticoagulation drug use on aneurysm sealing and the durability of endovascular abdominal aneurysm repair (EVAR) are conflicting. This study assessed the safety of chronic anticoagulation therapy after EVAR.

**Methods:** Records of 1409 consecutive patients having elective EVAR during 1997-2011 who were prospectively followed were reviewed. Survival, reintervention, conversion, and endoleak rates were analyzed in patients with and without chronic anticoagulants. Cox proportional hazards models were used to estimate the effect of anticoagulation therapy on outcomes.

**Results:** One-hundred and three (7.3%) patients were on chronic anticoagulation drugs (80 on vitamin K antagonists) at the time of EVAR. An additional 46 patients started on anticoagulants after repair were identified. Patients on chronic anticoagulation therapy at repair (mean age 73.6 years; 91 males) had more frequent cardiac disease (74.8% vs 44.2%; $P < .00001$), but no other differences in demographic and major baseline comorbidities with respect to the others. At baseline, mean abdominal aortic aneurysm (AAA) diameter was 56.43 mm vs 54.65 mm ($P = .076$) and aortic neck length 26.54 mm vs 25.21 mm ($P = .26$) in patients with and without anticoagulants, respectively. At 5 years, freedom from endoleak rates were 55.5% vs 69.9% ($P < .0001$), and freedom from reintervention/conversion rates were 69.4% vs 82.4% ($P < .0001$) in patients with (including those with delayed drug use) and without chronic anticoagulants, respectively. Controlling for covariates with the Cox regression method, at a mean follow-up of 64.3 ± 45.2 months after EVAR, use of anticoagulation drugs was independently associated with an increased risk of endoleak (odds ratio, OR 1.6; 95% confidence interval, CI: 1.23-2.07; $P < .0001$) and reintervention or late conversion rates (OR 1.8; 95% CI: 1.31-2.48; $P < .0001$).

**Conclusions:** The safety of anticoagulation therapy after EVAR is debatable. Chronic anticoagulation drug use risks exposure to a poor long-term outcome. A critical and balanced decision-making approach should be applied to patients with AAA and cardiac disease who may require prolonged anticoagulation treatment.

**A Randomised Controlled Trial of Supervised Exercise Regimens and Their Impact on Walking Performance, Skeletal Muscle Mass and Calpain Activity in Patients With Intermittent Claudication**

Delaney C.L., Miller M.D., Chataway T.K., Spark J.I. Eur J Vasc Endovasc Surg 2014;47:304-10.

**Objectives:** Supervised exercise training (SET) is recommended for patients with intermittent claudication (IC). The optimal exercise programme has not been identified, and the potential adverse effects of exercise on these patients warrant consideration. Calpain proteases have been linked with tissue atrophy following ischaemia–reperfusion injury. High calpain activity may therefore cause muscle wasting in claudicants undergoing SET, and skeletal muscle mass (SMM) is integral to healthy ageing. This study assesses the impact of (1) treadmill-based SET alone; and (2) treadmill-based SET combined with resistance training on pain-free walking distance (PFWD), SMM, and calpain activity.

**Methods:** Thirty-five patients with IC were randomised to 12 weeks of treadmill only SET (group A), or combined treadmill and lower-limb resistance SET (group B). PFWD via a 6-minute walking test, SMM via dual energy X-ray absorptiometry, and calpain activity via biopsies of gastrocnemius muscles were analysed.

**Results:** Intention-to-treat analyses revealed PFWD improved within group A (160 m to 204 m, $P = .03$), but not group B (181 m to 188 m, $P = .82$). There was no between group difference ($P = .42$). Calpain activity increased within group A ($1.62 \times 10^5$ fluorescent units [FU] to $2.21 \times 10^5$ FU, $P = .05$), but not group B. There was no between group difference ($P = .09$). SMM decreased within group A (−250 g, $P = .11$) and increased in group B (210 g, $P = .38$) ($P = .10$ between groups). Similar trends were evident for per protocol analyses, but, additionally, change in SMM was significantly different between groups ($P = .04$).

**Conclusions:** Neither exercise regimen was superior in terms of walking performance. Further work is required to investigate the impact of the calpain system on SMM in claudicants undertaking SET.