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sion, body mass index), baseline alcohol consumption were independently associated with AAA diagnosis ($P < .03$ for trend). There was a maximum hazard ratio of 1.21 (95% confidence interval [CI], 0.78 to 1.87) for ≥30.0 g (≥2 standard drinks) of daily alcohol consumption. The association for highest levels of alcohol intake was 1.65 (95% CI, 1.03 to 2.64).

Comment: This is the first report to document an association between alcohol consumption and AAA. The data convincingly demonstrate higher levels of alcohol consumption increase the risk of AAA, even in patients without pre-existing cardiovascular disease. Moderate alcohol consumption may have some benefit in lowering the risk of ischemic heart disease. However, higher levels of alcohol confer no benefit and actually appear harmful to the cardiovascular system.

A sustained mortality benefit from screening for abdominal aortic aneurysm

Kim LG, Scott AP, Ashton, HA. and the Multicentre Aneurysm Screening Study Group. Ann Intern Med 2007;146:699-706.

Conclusions: The early mortality benefit of screening ultrasonography in reducing abdominal aortic aneurysm (AAA)-related death is maintained over the long term. Cost effectiveness for screening improves over time.

Summary: The authors sought to estimate the benefits of ultrasound screening for AAA in terms of all-cause mortality and cost-effectiveness. They compared AAA mortality in a group of patients invited to undergo ultrasound screening vs a group that was not. Mean follow-up was 7 years. This was a randomized trial of AAA screening that was conducted in four centers in the United Kingdom (UK) using a population-based sample of 67,770 men aged 65 to 74 years. Patients with an AAA detected during screening were entered into a surveillance program and offered surgery when the aneurysm fulfilled predefined criteria. Cost analysis was performed using unit costs from large samples applied to individual data. Mortality data were obtained from the UK national database.

The hazard ratio for AAA mortality in the group invited for screening compared with those not invited for screening was 0.53 (95% confidence interval [CI], 0.42 to 0.68). In patients with normal results on initial ultrasonography, the rupture rate of AAA was 0.54 ruptures (95% CI, 0.25 to 1.02 ruptures) per 10,000 person-years. The observed hazard ratio for all cause mortality in the screened vs nonscreened men was 0.96 (95% CI, 0.93 to 1.00). Cost effectiveness analysis was estimated at US $19,500 (95% CI, $12,400 to $39,800) per life-year gained based on aneurysm-related mortality and $76,000 (95% CI, $33,000 to infinity) per life-year gained based on all cause death.

Comment: This is a late analysis of data from The Multicentre Aneurysm Screening Study (MASS) analyzing the effect of AAA screening on mortality in older men. It shows the short- and intermediate-term benefits of AAA screening continue in the longer term. An interesting side bit of data is that in patients whose initial aortic diameter is <3 cm, aneurysm-related death in the next 7 years is extraordinarily unusual. Additional screening after an initial ultrasound with a negative result is not warranted for at least up to 7 years after an initial negative result on a screening study.

The impact of postoperative discontinuation or continuation of chronic statin therapy on cardiac outcome after major vascular surgery

Le Manach Y, Godet G, Coriat P, et al. Anesth Analg 2007;104:1326-33.

Conclusions: Discontinuation of chronic statin therapy after major vascular surgery increases the risk of myocardial infarction. Statin therapy should be reinstituted on the first postoperative day after major vascular surgery.

Summary: Accumulating evidence indicates statins reduce perioperative cardiac events. It is unknown when patients who receive chronic statin therapy should resume statins after a major vascular procedure. The authors sought to compare cardiac outcomes in patients receiving chronic statin therapy who resumed statin therapy immediately postoperatively vs those where statin therapy was discontinued, at least temporarily, during the perioperative period. This was a retrospective analysis of a prospectively maintained database. Included were patients who underwent infrarenal aortic reconstruction for aneurysm or occlusive disease using endovascular or open techniques. Patients were studied from January 2001 to December 2004. Patients undergoing emergency procedures were excluded.

Blood was obtained for troponin I measurements when the patient arrived to the postanesthesia care unit and on the first, second, and third postoperative days. Patients not chronically treated with statins were considered controls and did not receive perioperative statin therapy. Patients chronically treated with statins were divided into two groups. The first was the discontinuation group. These were 491 patients from January 2001 to December 2003 in which the authors' institution did not have specific guidelines regarding postoperative statin re-administration. The continuation group consisted of 178 patients from January 2004 where guidelines were in place to continue statins up to the evening before surgery, with resumption on the first postoperative day using either nasogastric or oral administration. Intracohort (propensity score) and extracohort (Lee score) risk adjustments were performed to determine the significance of differences in elevated troponin levels in the continuation vs discontinuation groups. A troponin I level >99th percentile or >0.2 ng/mL was considered indicative of myocardial necrosis.

In the discontinuation group, median delay between surgery and resumption of statin therapy was 4 days, and in the continuation group, it was 1 day ($P < .001$). With propensity score matching for likelihood of preoperative treatment, the odds ratio associated with chronic statin treatment to predict myonecrosis for patients with vs without early postoperative statin resumption (continuation vs discontinuation groups) was 0.38 and 2.1 (relative risk reduction, 5.4; 95% confidence interval, 1.2 to 25.3, $P < .001$). The odds ratio for myocardial necrosis after adjustment for the Lee score was 0.38 in the continuation group and 2.1 in the discontinuation group ($P < .001$). Postoperative statin withdrawal was an independent predictor of postoperative elevated troponin levels (odds ratio, 2.9; 95% confidence interval, 1.6 to 5.5).

Comment: Although this was not a randomized trial and it used historic controls, the data suggest that early resumption of statin therapy in patients chronically receiving statin therapy will reduce the incidence of myocardial infarction. Although there was no difference in perioperative mortality in the continuation vs discontinuation groups, perioperative myocardial infarction in itself is an end point worth reducing. It has been associated with greater risk of long-term death and with increased duration of hospitalization. More and more evidence now suggests a deleterious cardiac effect of withdrawing statin therapy in the perioperative period.

Partial thrombosis of the false lumen in patients with acute type B aortic dissection

Tsai TT, Evangelista A, Nienaber CA. The International Registry of Acute Aortic Dissection. N Engl J Med 2007;357:349-59.

Conclusion: After hospital discharge, patients with acute type B aortic dissection and partial thrombosis of the false lumen are at increased risk of death compared with patients with complete patency of the false lumen.

Summary: The International Registry of Acute Aortic Dissection (IRAAD) tracks patients with acute aortic dissection at 22 aortic centers in 11 countries. This is a registry, and treatment during hospitalization is not standardized but is conducted at the discretion of each patient's physician and institution.

Patients with acute aortic type B dissection have low in-hospital mortality but survival rates after discharge range from 92% at 1 year to 48 to 82% at 5 years. It has been suggested that patients with complete thrombosis of the false lumen have improved outcomes compared with patients who have a patent false lumen, with the patent false lumen thought to place the patient at increased risk of aortic expansion and death (Am J Card 2001;87:1378-82). The authors sought to determine the influence on mortality of partial thrombosis of the false lumen. They defined partial thrombosis of the false lumen as both flow and thrombus present in the false lumen.

Between 1996 and 2003, 201 patients with type B aortic dissection were enrolled in IRAAD and survived to discharge from the hospital. The authors developed mortality curves according to the status of the false lumen (complete thrombosis vs partial thrombosis vs patent). False lumen status was determined during the initial hospitalization. They used Cox proportional hazard analysis to identify independent predictors of death on follow-up.

There were 114 patients (56.7%) who, during the index hospitalization, had a patent false lumen. Sixty-eight patients (33.8%) had partial thrombosis of the false lumen, and 19 (9.5%) had complete thrombosis of the false lumen. At 3 years, the mortality rate for patients with a patent false lumen was 13.7%. The mortality rate for patients with partial thrombosis of the false lumen was 31.6%, and mortality for those with complete thrombosis was 22.6% ± 22.6% (median follow-up, 2.8 years; $P = .003$, log-rank test). Postdischarge mortality was independently predicted by partial thrombosis of the false lumen (relative risk, 2.69; 95% confidence interval [CI], 1.45 to 4.98; $P = .002$), a history of atherosclerosis (relative risk, 1.87; 95% CI, 1.01 to 3.47; $P < .05$), and a history of aortic aneurysm (relative risk, 2.05; 95% CI, 1.07 to 3.93; $P = .03$).

Comment: The authors postulate their findings may reflect that a patent false lumen is perfused by a proximal entry tear and then decompressed through re-entry tears distally. When thrombus partially occludes the lumen, these distal re-entry tears may be occluded, impeding outflow and leading to an increase in mean arterial and diastolic pressure that results in increased wall tension in the false lumen and increases the risk of aneurysm expansion, with increased risk of rupture and redissection.

Sustained benefit at two years of primary femoral-popliteal stenting compared with balloon angioplasty with optional stenting

Schillinger M, Sabeti S, Dick P, et al. Circulation 2007;115:2745-9.

Conclusions: At 2 years, there is a sustained morphologic benefit for treatment of superficial femoral artery (SFA) obstructions using self-expanding nitinol stents inserted primarily vs balloon angioplasty with optional

stenting. There was no statistically significant clinical benefit in primary stenting vs optional stenting at 2 years.

Summary: The authors previously published data from the angioplasty vs stenting with nitinol stents in the superficial femoral artery (ABSOLUTE) trial. At 1 year, there was benefit from primary stenting vs optional stenting (N Engl J Med 2006;354:1879-88). The authors now report 2-year data on restenosis and clinical outcomes of the original patient group.

There were 104 patients with clinical limb ischemia and SFA occlusions originally entered into the study. At 2 years, 98 patients (94%) were available for assessment of restenosis (>50% by duplex ultrasound imaging) and for assessment of clinical and hemodynamic outcome using ankle-brachial index and treadmill walking distance. At 2 years, by an intention to treat analysis, restenosis rates were 45.7% (21 of 46) in the primary stenting group vs 69.9% (36 of 52) in the optional stenting group ($P = .031$). Primary and secondary (n = 63) stenting were both superior to plain balloon angioplasty (n = 35) with respect to the occurrence of restenosis (49% vs 74.3%; $P = .028$) using a treatment-received analysis. There was no statistical benefit in the primary stent group towards better treadmill walking (average 302 vs 196 m; $P = .12$) or any significant improvement in ankle-brachial index values in the primary stented vs optional stented group (average, 0.88 vs 0.78; $P < .09$). There was no difference in intervention rate in the primary stenting (17 of 46 [37.0%]) vs optional stenting group (28 of 52 [53.8%]; $P = .14$).

Comment: The authors put a favorable spin on their data by emphasizing a "trend" towards improved clinical benefit with primary vs optional stenting. Nevertheless, statistically insignificant is still statistically insignificant. Although larger numbers may have shown improvement in clinical benefit with primary stenting vs optional stenting, a 45% restenosis rate in stented patients at 2 years without clear clinical benefit indicates percutaneous treatment of SFA occlusive lesions is still not very good treatment of SFA occlusive lesions.