Endoscopic Lumbar Sympathectomy for Plantar Hyperhidrosis


Conclusion: In patients with severe plantar hyperhidrosis, endovascular lumbar sympathectomy is safe and effective.

Summary: Primary plantar hyperhidrosis is characterized by excessive secretions by eccrine sweat glands of the feet. The etiology is unknown and symptoms include cold cyanotic skin, skin maceration, bacterial and fungal infection, and bromidrosis (foul-smelling feet). Interrupting innervation of the sweat glands stops sweat secretion and has been used since the 1920s to treat other hyperhidrosis. The current study was designed to test the hypothesis that the benefits of endovascular repair may take longer than four years to become apparent.

In EVAR 2, 404 patients with AAAs ≤ 5.5 cm in diameter and considered ineligible for open repair were randomized to either undergo endovascular repair or no repair. The study was conducted in 23 hospitals in the United Kingdom from 1999 through 2004. The endovascular repair group consisted of 197 patients, and 207 were assigned to no intervention. Until the end of 2009, patients were followed for graft related complications, reinterventions, and rates of death.

Endovascular versus Open Repair of Abdominal Aortic Aneurysm


Conclusion: Endovascular repair of abdominal aortic aneurysm (AAA) has lower operative mortality than open repair. There is no difference in aneurysm-related or total mortality in the long term. There are increased rates of graft complications with endovascular repair, more reinterventions, and endovascular repair is more costly.


This report from the United Kingdom EVAR investigators presents long-term follow-up data of the EVAR 1 trial. In EVAR 1, from 1999 through 2004, there were 1252 patients with AAAs >5.5 cm who were randomly allocated to undergo either endovascular or open repair at 37 hospitals in the United Kingdom. There were 626 patients in each group. Resource use, graft-related complications, reinterventions, and rates of death were then determined until the end of 2009. In the endovascular repair group and open repair group, 30-day operative mortalities were 1.8% and 4.3%, respectively (adjusted odds ratio for endovascular repair compared to open repair, 0.39; 95% CI, 0.18 to 0.89; P = 0.2). Early benefit of endovascular repair with respect to aneurysm-related mortality was lost by the end of the study, at least partially, because of fatal endograft ruptures (adjusted hazard ratio, 0.92; 95% CI, 0.57 to 1.49; P = .73). There were higher rates of graft-related complications and reinterventions with endovascular repair. New complications occurred up to 8 years following randomization, contributing to overall higher cost of endovascular repair.

Comment: The value of the study is the long-term prospective nature of the data and excellent follow-up with very few patients lost to follow-up. The results of the study, in general, are not surprising in that they confirm previously published midterm findings of the EVAR 1 trial and other trials evaluating endovascular vs open repair of AAAs. Operative mortality with endovascular AAA repair is significantly lower than with open AAA repair and aneurysm-related mortality is reduced early on. Early benefit, however, is “completely lost in the longer-term” with no differences in long-term mortality and higher aneurysm-related mortality after 4 years in the patients treated with EVAR. The authors note secondary rupture after AAA repair was associated only with EVAR and “appeared” to explain the late aneurysm-related mortality in the EVAR patients. These data indicate EVAR for AAA is not as big a win for patients in the long term as was hoped and, with the current cost structure, is clearly a loss for the economics of health care system.

Endovascular Repair of Aortic Aneurysm in Patients Physically Ineligible for Open Repair


Conclusion: In patients ineligible for open repair, endovascular repair of abdominal aortic aneurysm (AAA) is associated with a lower rate of aneurysm-related mortality than no repair. Endovascular repair, however, is not associated with a reduction in the rate of death from any cause. Rate of graft-related complications and reinterventions were high and contributed to increased cost.

Summary: An early premise of endovascular repair of AAA was that it would be used in patients ineligible for open surgical repair with the expectation that life would be prolonged by eliminating the risk of fatal aneurysm rupture. Midterm results of EVAR 2 showed no benefit of endovascular repair on total or aneurysm-related mortality up to 4 years of follow-up (Lancet 2005;365:2187-92). In EVAR 2, operative mortality was higher than anticipated (9%) and there was a 68% mortality at 4 years. In addition, belated rupture of large, untreated aneurysms was lower than anticipated (9 ruptures per 100 person-years). The current study was designed to test the hypothesis that the benefits of endovascular repair may take longer than four years to become apparent.

In EVAR 2, 404 patients with AAAs ≤ 5.5 cm in diameter and considered ineligible for open repair were randomized to either undergo endovascular repair or no repair. The study was conducted in 33 hospitals in the United Kingdom from 1999 through 2004. The endovascular repair group consisted of 197 patients, and 207 were assigned to no intervention. Until the end of 2009, patients were followed for graft related complications, reinterventions, and rates of death.

The endovascular repair group 30-day operative mortality was 7.3%. Overall rate of AAA rupture in the no intervention group was 12.4 per 100 person-years (95% CI, 9.6 to 16.2). There was lower AAA-related mortality in the endovascular repair group (adjusted hazard ratio, 0.53; 95% CI, 0.32 to 0.89; P = 0.02). However, the advantage of decreased AAA-related mortality did not result in any benefit of total mortality (adjusted hazard ratio, 0.99; 95% CI, 0.78 to 1.27; P = 0.97). Of patients surviving endovascular repair, 48% had graft-related complications. Within the first 6 years, 27% required reintervention. Over the 8 years of follow up, endovascular repair was considerably more expensive than no repair (cost difference, U.S. $ 14,867; 95% CI, 11,556 to 18,176).

Comment: The midterm results of the EVAR 2 reported in 2005 did not support endovascular repair of AAAs in patients considered unfit for open repair. Now, with longer follow-up, the authors report benefit of endovascular repair in terms of aneurysm-related mortality but not in long term all-cause mortality. Patients in the study had a limited life expectancy, regardless of whether the aneurysm was managed with EVAR or no intervention was performed. There were few survivors beyond 8 years in either group. Many patients in the no intervention group crossed over to EVAR and these patients were, on post hoc analysis, found to be “more fit” than those that did not cross over. There were only two late ruptures in the EVAR group; perhaps related to the limited survival of the patients in general. One conclusion is that patients did not survive long enough to die of rupture following EVAR. Looking at both the late results of EVAR 1 and EVAR 2, one might conclude poor risk patients can, in terms of aneurysm related...
mortality, be managed with EVAR, while good to moderate risk patients are best managed with open repair.

**Growth Rate of Affected Aorta in Patients with Type B Partially Closed Aortic Dissection**


**Conclusion:** Patients with Type B aortic dissection, entirely patent false lumens have higher growth rates than partially-patent false lumens. Completely-occluded false lumens have the lowest false lumen growth rate.

**Summary:** Studies of patients with Type B aortic dissection have suggested that patients with complete thrombosis of the false lumen have improved outcomes compared to those where the false lumen remains patent with such patients having an increased risk of aortic enlargement and death (Ann J Card 2001;87:1378-82 and Lancet 1997;349:1461-4). A recent study suggested stratification of patients with patent false lumens into those where the false lumen is partially-patent with nonoccluding thrombus and those with a patent false lumen without associated thrombus. That study found an increased risk of death in the patients with partial thrombosis of the false lumen (N Engl J Med 2007;357:349-59). This later study did not describe the natural history of the affected aorta or the cause of death in patients with partially-closed false lumens

In the current study, the authors evaluated the evolution of the dissected aorta and the growth rate of Type B aortic dissections using computed tomography scanning. Patients were divided into 4 groups. Group A consisted of those with completely-closed false lumens. Group B consisted of patients with a patent false lumen with continuous blood flow from entry to reentry without regard to presence of thrombus in the false lumen. Group C consisted of patients with partially-closed false lumens defined as a false lumen partially-closed by a thrombus in any part of the false lumen. Group D was a subset of group C when the partially-closed false lumen was in the total portion of the false lumen with the presence of the portion of the false lumen patent. The mean fastest growth rates for patients with completely closed false lumens, partially-closed false lumens, and patent false lumens without associated thrombus. That study found an increased risk of death in the patients with partial thrombosis of the false lumen (N Engl J Med 2007;357:349-59). This later study did not describe the natural history of the affected aorta or the cause of death in patients with partially-closed false lumens

The article suggests a discrepancy between causes of death and the natural history of the aorta in patients with completely patent versus partially-patent false lumens following Type B aortic dissection. The current study indicates the natural history of a false lumen that is completely patent appears worse, in terms of growth rates, than the partially-patent false lumen. Certainly it would be useful to know whether the partially-patent false lumen has a higher risk of rupture. The walls of the dissected aorta with partially-patent false lumens may have a combination of diminished strength and relative hypoxia of the arterial wall adjacent to the internal luminal thrombus that results in increased local inflammation and wall weakening (JVS 1994;20:329-35 and Circulation 2005;111:1063-71). The implication is there may be factors other than simple expansion of the luminal diameter that predispose to rupture of the false lumen in patients with Type B aortic dissection.

**Incidence of Moderate to Severe Cognitive Dysfunction in Patients Treated with Carotid artery Stenting**


**Conclusion:** Carotid artery stenting is associated with cognitive performance decline of modern severity one day after stenting.

**Summary:** Neuropsychometric test are not routinely used to evaluate the effects of carotid interventions. Comparison of primary cognitive dysfunction in patients undergoing carotid endarterectomy (CEA) versus those undergoing carotid artery stenting (CAS) suggests patients treated with CAS tend to perform slightly worse with selective cognitive tests. There is, however, little prospective information on cognitive function using controls subjects, those not undergoing a cerebrovascular intervention. In this prospective, observational study, they compared the incidence of early cognitive dysfunction in patients undergoing CAS with embolic protection devices using a control population consisting of patients undergoing percutaneous coronary artery procedures.

There were 24 patients who underwent elective CAS and 23 patients who underwent a coronary procedure. Coronary procedures were all catheter-based and performed by members of the cardiology service. All CAS procedures were performed by either the Neurovascular service or the vascular surgical service at the authors’ institution. Subjects underwent one of six neuropsychometric tests chosen to represent a range of cognitive demands. Mean changes and standard deviations for patients undergoing coronary stenting procedures were used to calculate Z scores for each test. A negative value indicated a cognitive performance worse than that of the mean of controls. Negative Z scores were then transformed into injury points for each neuropsychometric test. An average deficit score (ADS) was calculated by dividing the sum of injury points by the total number of completed tests. Patients were considered to have cognitive dysfunction if their ADS was greater to severe cognitive dysfunction if their ADS was greater than the control mean by a minimum of 1.5 standard deviations and/or Z score of ≥ 1.5 on 2 or more tests. Severe cognitive dysfunction was defined as ADS greater than the control mean by a minimum of 2 standard deviations and/or Z scores of ≥ 2 on 2 or more tests.

The controlled patients tend to be younger and had a lower incidence of stroke or previous transient ischemic attack. On the first day after surgery, 41% of patients treated with CAS developed moderate to severe cognitive dysfunction (P = 0.022). Average deficit scores were also significantly higher in the CAS group at one day (P = 0.026). Differences in the mean difference in the history of stroke, transient ischemic attack, or age. At one month, one of 11 patients assessed (9%) who had been treated with CAS still exhibited cognitive dysfunction.

**Comment:** The effect of carotid interventions on cognitive function is an area of research that needs more long-term data. It would also be very interesting to correlate cognitive function with postprocedure MRI diffusion weighted imaging (DWI) studies. At this point we really don’t know whether DWI lesions have any actual short-term or long-term effects. We do know, however, that DWI lesions are considerably more common after CAS compared to CEA. This paper is of value primarily to bring this line of quantitative cognitive research to our attention and in demonstrating the techniques used to generate the information.

**Mortality and Vascular Morbidity in Older Adults with Asymptomatic Versus Symptomatic Peripheral Artery Disease**


**Conclusion:** In patients with peripheral arterial disease (PAD), the risk of mortality is similar in those patients where PAD is asymptomatic and in those patients where it is asymptomatic. In both groups, mortality is higher than in those without PAD.

**Summary:** PAD is widespread, especially in elderly patients and those with diabetes mellitus or with clusters of cardiovascular risk factors. PAD is known to be associated with increased risk of premature mortality and cardiovascular and cerebrovascular events (Circulation 2006;114:688-99). Studies regarding relative mortality of patients with symptomatic versus asymptomatic PAD are a bit conflicting. One described progressive increase in mortality as patients moved from asymptomatic to symptomatic or severely symptomatic status (N Engl J Med 1992;326:385-86). Another study demonstrated asymptomatic PAD patients had higher rates of morbidity events than symptomatic patients (Intern J Epid 1996;25:1172-81). Another study indicated patients with asymptomatic PAD have poorer quality of life and functional performance than patients with intermittent claudication (Circulation 2005;117:2484-91). The authors, therefore, thought to describe in a German population the prevalence of asymptomatic and symptomatic PAD and to relate symptomatic status to clinical events.

The patients are part of the German Epidemiologic Trial on Ankle Brachial Index (getABI). This is an ongoing, prospective, observational, cohort study that began in October 2001. Three hundred forty-four general practice physicians in Germany were trained in identification of patients with PAD. Patients were then identified in their practices during a prespecified week in October 2001. Approximately 20 eligible patients per practice met the inclusion criterion (age ≥ 65 years, legally competent, and able to cooperate appropriately and provide consent with life expectancy ≥ 24 months). ABI’s were determined and patients were followed under a standardized protocol for development of cardiovascular events. There were 6880 patients followed for over 5 years. Of these, 5392 were felt to have asymptomatic PAD (ABI> 0.9 without symptoms). A total of 593 were felt to have symptomatic PAD (lower extremity revascularization, amputation secondary to PAD) or symptomatic consistent with intermittent claudication regardless of ankle brachial index).

For the composite endpoint of all-cause death or severe vascular event (myocardial infarction, stroke, carotid revascularization, coronary revascularization, lower extremity peripheral vascular events) risk was increased in patients with symptomatic compared to asymptomatic PAD (HR, 1.48; 95% CI, 1.21 to 1.80). There were no differences between patients with symptomatic and asymptomatic PAD for cardiovascular events alone (HR, 1.20; 95% CI, 0.89 to 1.43), all-cause death/myocardial infarction/stroke (HR, 1.18; 95% CI, 0.92 to 1.20), or for cardiovascular events alone (HR, 1.20; 95% CI, 0.89 to 1.57), or cerebrovascular events alone (HR, 1.17; 95% CI, 1.00 to 1.18). Risk was increased in lower ABI categories.

**Comment:** Overall, in the primary practice setting, there was a high prevalence of PAD (12.2% asymptomatic, 8.7% symptomatic). The composite end point of all-cause mortality or severe cardiac cerebral or peripheral vascular events occurred in 104.7 cases per 1000 patient years in patients with symptomatic PAD, 60.4 cases per 1000 patient years in patients with asymptomatic PAD, and in 27.2 cases per 1000 patient years in patients without PAD. However, for the individual end points of all-cause death, all-cause death/myocardial infarction/stroke, and for cardiovascular