patients needed additional treatment. 30-day mortality was 1.4% (5/366). Follow-up is reported till 84 months (n = 24 at 84 months). 34% (123/366) patients died during follow-up. In five patients mortality was AAA related (2 ruptured). Kaplan Meier estimates revealed primary clinical success rates of 98% at 1 year, 93% at 2 years, 88% at 3 years, 79% at 4, 64% at 5 years, 51% at 6 years and 48% at 7 years, respectively. Secondary interventions had to be performed in 18% (66/366) of patients. Ten open conversions for failed endografts were performed. Life table yearly risk for AAA related reintervention was 6%, yearly risk for conversion 1.1%, and yearly risk for AAA-related mortality was 0.8%.

Conclusions: Initially technical success of EVAR using the Talent endograft is high, with low yearly risk for AAA-related mortality and conversion. However, a substantial amount of mainly endovascular reinterventions is necessary during long-term follow-up to achieve these results.

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C5: Poster Presentation I - Aortic Disease (2)

PS22.

Hardman Index and Glasgow Aneurysm Score in Predicting Survival Following Open Repair of Ruptured Abdominal Aortic Aneurysms (AAA): Are They Practical?

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Objectives: Despite advances in perioperative care, mortality after open repair of ruptured AAA remains high. The need to avoid futile surgery in frail patients led to the development of prognostic scoring systems. Literature regarding their clinical usefulness has been conflicting. This study aimed to assess the utility of 2 scoring systems in predicting survival following open repair of ruptured AAA.

Methods: 95 patients who had undergone open repair of a ruptured AAA between 2002 to 2007 were identified from a prospectively collected audit database. Parameter data for the Hardman Index (HI) and Glasgow Aneurysm Score (GAS) was collected retrospectively. Operative mortality, times of presentation, decision for surgery and availability of scoring results at time of decision-making was also documented. Receiver operating curves were constructed for both scoring systems.

Results: The median age of patients was 74 with 77 men and 18 women. The operative mortality was 48% (46/95). GAS was complete in 86 patients but only 44 had complete data for HI. However, at time of decision for surgery only 25 patients had all investigative parameters available to enable HI scoring while 78 patients had complete GAS results available. The area under the curve was 0.724 (95% CI, 0.575-0.874) and 0.695 (95% CI, 0.583-0.807) for HI and GAS respectively. The scoring systems were not significantly different in predicting perioperative death (p>0.05). However, in 6 patients who had HI > 3, one patient survived surgery and of 87 patients with GAS > 95, 15 lived.

Conclusions: Although the results suggest that both HI and GAS are moderately successful in predicting perioperative death, the scoring systems do not appear practical. A substantial proportion of the test results were not available to the clinician at the time of decision-making. This suggests that front-line clinicians are making subjective decisions that are later supported by prognostic scoring. More needs to be done to improve rapid access to investigation results.

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PS24.

Inhomogenous Wall Stress Distribution in the Normal Human Thoracic Aorta: A Potential Etiology of Type B Aortic Dissections

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Objectives: To determine the role peak aortic wall stress plays in the pathophysiology of type B aortic dissections.

Methods: Twenty-eight subjects with normal thoracic aortas underwent ECG-gated CTA. The aortic arch and descending thoracic aorta were segmented, reconstructed, and triangulated to create a mesh (Amira). Using a pressure load of 120 mm Hg and a uniform aortic wall thickness of 0.22 cm, finite element analysis was performed (Abaqus) to predict regional thoracic aortic wall stress.

Results: There were local maxima of regional wall stress just distal to the ostia of each arch vessel (Figure 1); the peak stress at the level of the left subclavian artery (LSA) was 0.20 ± 0.03 MPa. Peak wall stress in the proximal descending thoracic aorta (0.66 ± 0.01 MPa) was significantly lower than that of the arch vessels (p < 0.001). Patients with LSA arising from the top of the arch (n = 15, type I arch) did not have significantly different (p = 0.46) LSA peak wall stress than patients with LSA arising from below the horizontal line extending from the outer curvature of the arch (n = 13, type II or III arch).

Conclusions: Regardless of age or arch configuration, there is a peak in wall stress distal to the ostium of the LSA.