





DISCLOSURES

Proctor for WL Gore & Cook



 The primary aim of elective AAA repair is to increase life expectancy by preventing aneurysm rupture.

1st aim: not to die on operation

• 2nd aim: not to die on AAA rupture on the long run



Is the game really over?

VASCULAR DISEASE

Repair of infrarenal aortic aneurysm—the debate is OVER

Alan Karthikesalingam and Matthew M. Thompson

Randomized trials of open versus endovascular repair (EVR) of abdominal aortic aneurysm have demonstrated the short-term superiority of EVR, but have raised questions about the long-term durability of this approach. The final report from the OVER trial now demonstrates that EVR is the treatment of choice for morphologically suitable aortic aneurysms.

Karthikesalingam, A. & Thompson, M. M. Nat. Rev. Cardiol. 10, 122–124 (2013); published online 29 January 2013; doi:10.1038/nrcardio.2013.4

The primary goal of abdominal aortic aneurysm (AAA) repair is to prolong the life of patients whose risk of premature death from aneurysm rupture would otherwise be substantial. The safety of operative intervention assumes critical importance as

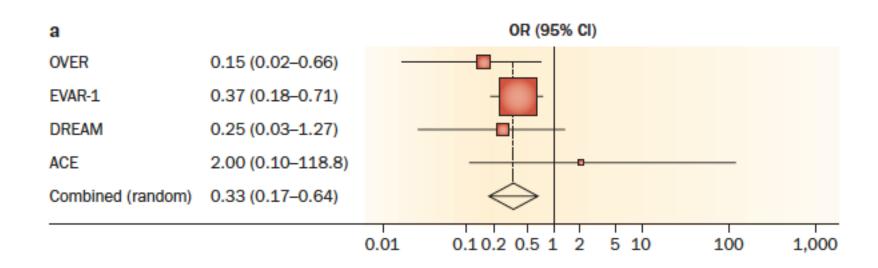
it must be weighed against the risk of fatal rupture without surgery, which has proved stubbornly resistant to reduction by medical therapy. For this reason, clinical practice has been revolutionized by the advent of endovascular repair (EVR), a minimally invasive

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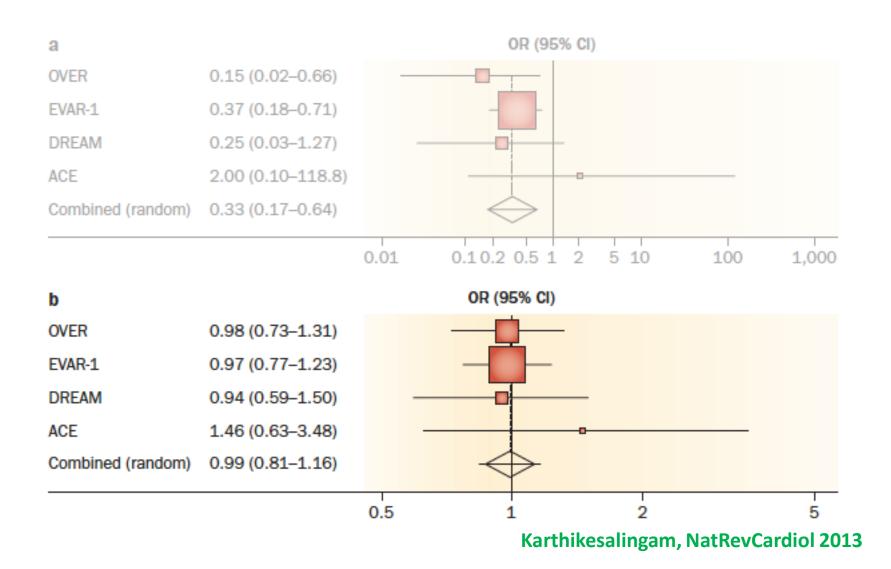


Perioperative & Longterm mortality





Perioperative & Longterm mortality





Perioperative & Longterm mortality

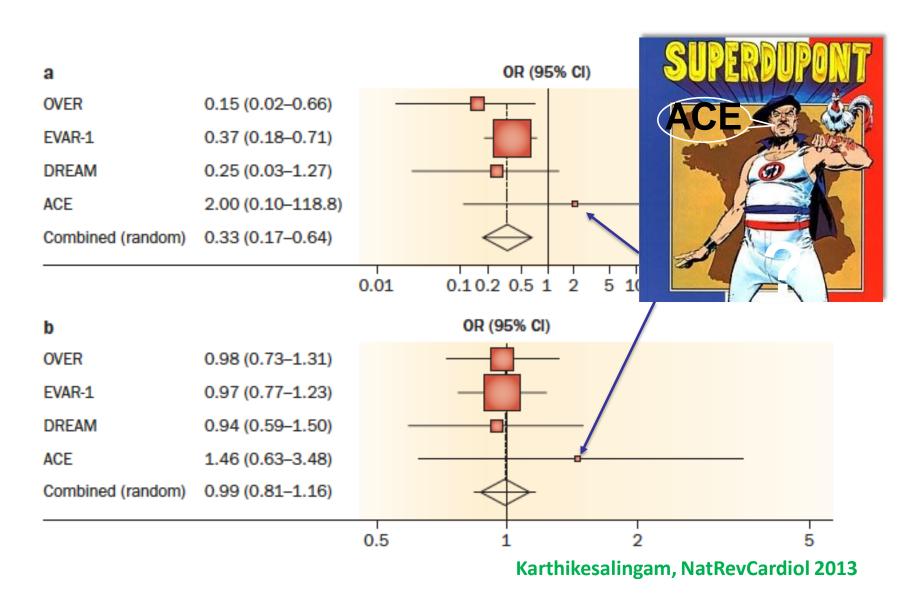




Table 3. Summary of factors identified in this review that influence long-term survival following abdominal aortic aneurysm repair.

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Factor	Number of patients	Number of studies	HR (95% CI)	l ² (%)	Overall Z-Test effect	p
Demographic						
Age (continuous)/year	31,100	21	1.05 (1.04-1.06)	81	9.74	<.00001
Age category						
Up to 75 years old	22,047	8	1.77 (1.36-2.30)	77	4.24	<.0001
>75 years old	24,492	5	2.32 (1.93-2.80)	57	8.79	<.00001
Females	49,653	16	1.15 (1.07-1.27)	45	3.42	<.0006
Clinical assessment						
ASA	3,374	3	1.30 (1.16-1.47)	0	4.32	<.0001
Comorbidity						
IHD	31,441	18	1.29 (1.18-1.48)	46	5.58	<.00001
MI	5,433	7	1.52 (1.32-1.73)	0	6.04	<.00001
Cardiac failure	35,525	14	1.91 (1.58-2.30)	70	6.77	<.00001
Hypertension	17,927	9	0.90 (0.79-1.03)	60	1.55	0.12
LVH on ECG	1,308	3	2.25 (1.66-3.04)	0	5.28	<.00001
COPD	43,953	18	1.53 (1.37-1.70)	70	7.58	<.00001
COPD on O ₂ supplement	4,142	3	3.05 (1.93-4.80)	63	4.8	<.00001
Renal impairment						
Creatinine	26,974	16	1.54 (1.43-1.67)	11	10.8	<.00001
(>150-200 μmol/L)						
Dialysis or ESRF	4,744	5	3.15 (2.45-4.04)	0	8.98	<.00001
Cerebrovascular disease	7,726	9	1.57 (1.40-1.77)	0	7.49	<.00001
Carotid disease	9,578	2	1.27 (0.93-1.73)	0	1.5	0.13
PVD	2,646	3	1.36 (1.18-1.58)	0	4.17	<.0001
Diabetes	44,211	14	1.34 (1.20-1.49)	26	5.35	<.00001



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AAA repaired patients life expectancy

Eur J Vasc Endovasc Surg (2015) 50, 320-330

REVIEW

Systematic Review and Meta-analysis of Long-term survival After Elective Infrarenal Abdominal Aortic Aneurysm Repair 1969-2011: 5 Year Survival Remains Poor Despite Advances in Medical Care and Treatment Strategies

S.S. Bahia ^{a,*}, P.J.E. Holt ^a, D. Jackson ^b, B.O. Patterson ^a, R.J. Hinchliffe ^a, M.M. Thompson ^a, A. Karthikesalingam ^a

WHAT THIS PAPER ADDS

The key findings of this study are that 5 year survival after elective infrarenal AAA repair is 69% and that this value has not improved over the period 1969-2011. A larger aneurysm diameter at the time of surgery was associated with poorer 5 year survival. This 5 year survival figure is disappointingly poor; patients diagnosed with a Dukes B colorectal cancer can expect better 5 year survival. More needs to be done to address the shortfall in survival to ensure that patients who can now reasonably expect to survive major aortic surgery live long enough to justify what remains a significant intervention.

^a St George's Vascular Institute, London, UK

^b MRC Biostatistics Unit, Cambridge, UK



AAA repaired patients life expectancy

Trial arm Survival Probability Bartoli (No secondary intervention) Bartoli (Secondary Intervention) 0.46 | 0.34 | 0.58 | 0.70 | 0.48 | 0.86 | 0.77 | 0.99 | 0.97 | 0.52 | 1.00 | 0.82 | 0.47 | 0.96 | 0.67 | 0.41 | 0.86 | 0.71 | 0.59 | 0.80 | 0.78 | 0.67 | 0.86 | 0.78 | 0.67 | 0.86 | 0.75 | 0.63 | 0.84 | 0.67 | 0.64 | 0.70 | 0.52 | 0.47 | 0.57 | 0.47 | 0.54 | 0.91 | 0.77 | 0.77 | 0.47 | 0.66 | 0.80 | 0.77 | 0.77 | 0.66 | 0.82 | 0.80 | 0.73 | 0.85 | 0.75 | 0.76 | 0.66 | 0.85 | 0.75 | 0.76 | 0.66 | 0.85 | 0.75 | 0.77 | 0.54 | 0.91 | 0.57 | 0.79 | 0.64 | 0.63 | 0.65 | 0.65 | 0.66 | 0.65 | 0.65 | 0.66 | 0.65 | 0.65 | 0.66 | 0.65 | 0.65 | 0.66 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.65 | 0.74 | 0.70 | 0.66 | 0.74 | 0.70 | 0.66 | 0.74 | 0.73 | 0.89 | 0.77 | 0.75 | 0.77 | 0.77 | 0.78 | 0.77 | 0.77 | 0.78 | 0.77 | 0.79 | 0.77 | 0.78 | 0.77 | 0.79 | 0.77 | 0.78 | 0.77 | 0.79 | 0.77 | 0.78 | 0.77 | 0.79 | 0.78 | 0.77 | 0.79 | 0.79 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.79 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0.77 | 0. Bayazit (>5cm) Bayazit (<5cm) Bayazti (<5cm)
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Figure 2. Pooled 5 year survival after elective IR AAA.



Vascular Medicine

Predictors of Abdominal Aortic Aneurysm Sac Enlargement After Endovascular Repair

Andres Schanzer, MD; Roy K. Greenberg, MD; Nathanael Hevelone, MPH; William P. Robinson, MD; Mohammad H. Eslami, MD; Robert J. Goldberg, PhD; Louis Messina, MD

Background—The majority of infrarenal abdominal aortic aneurysm (AAA) repairs in the United States are performed with endovascular methods. Baseline aortoiliac arterial anatomic characteristics are fundamental criteria for appropriate patient selection for endovascular aortic repair (EVAR) and key determinants of long-term success. We evaluated compliance with anatomic guidelines for EVAR and the relationship between baseline aortoiliac arterial anatomy and

Methods and Results—Patients with pre-EVAR and at least 1 post-EVAR computed tomography scan were identified from the M2S, Inc. imaging database (1999 to 2008). Preoperative baseline aortoiliac anatomic characteristics were reviewed for each patient. Data relating to the specific AAA endovascular device implanted were not available. Therefore, morphological measurements were compared with the most liberal and the most conservative published anatomic guidelines as stated in each manufacturer's instructions for use. The primary study outcome was post-EVAR AAA sac enlargement (>5-mm diameter increase). In 10 228 patients undergoing EVAR, 59% had a maximum AAA diameter below the 55-mm threshold at which intervention is recommended over surveillance. Only 42% of patients had anatomy that met the most conservative definition of device instructions for use; 69% met the most liberal definition of device instructions for use. The 5-year post-EVAR rate of AAA sac enlargement was 41%. Independent predictors of AAA sac enlargement included endoleak, age ≥80 years, aortic neck diameter ≥28 mm, aortic neck angle >60°, and common iliac artery diameter >20 mm.

Conclusion—In this multicenter observational study, compliance with EVAR device guidelines was low and post-EVAR aneurysm sac enlargement was high, raising concern for long-term risk of aneurysm rupture. (Circulation. 2011;123:2848-2855.)

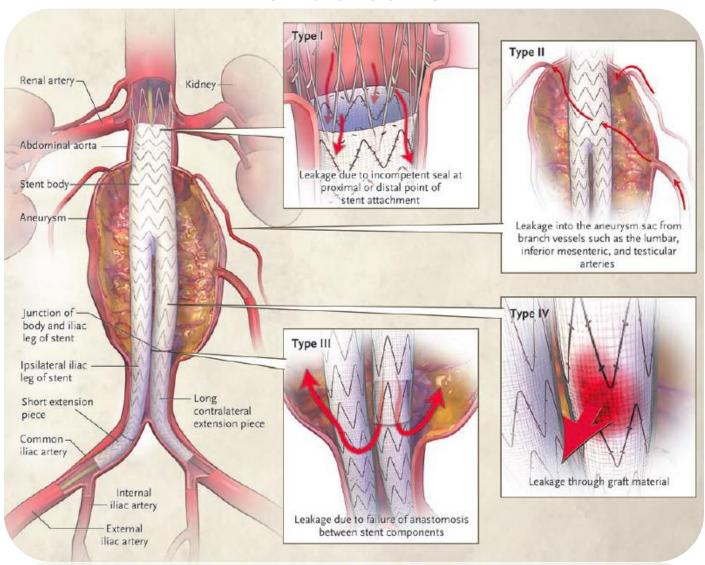
Key Words: abdominal aortic aneurysm ■ endovascular procedures ■ graft



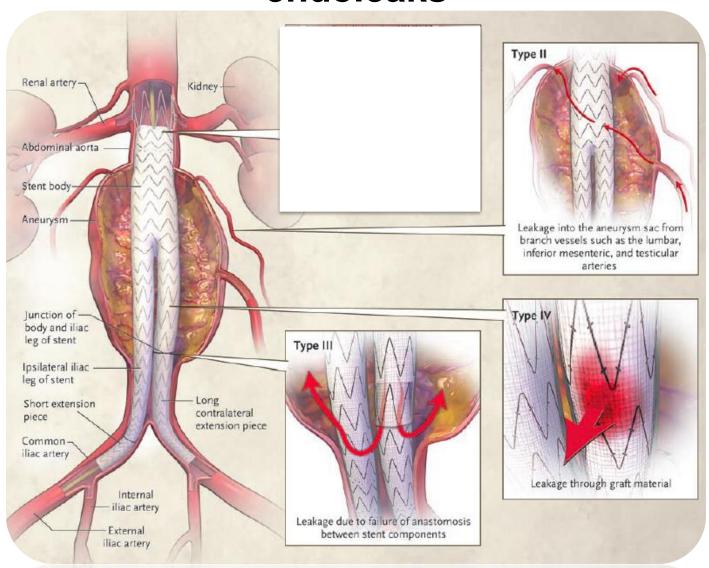
Respect the IFU



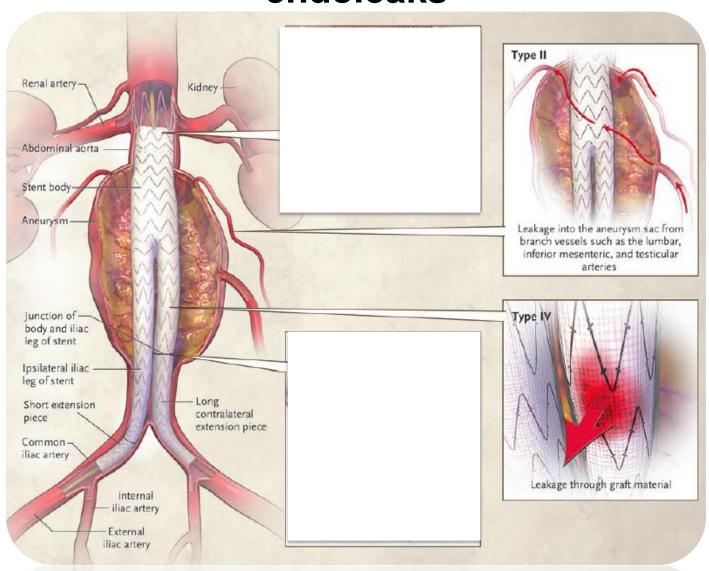




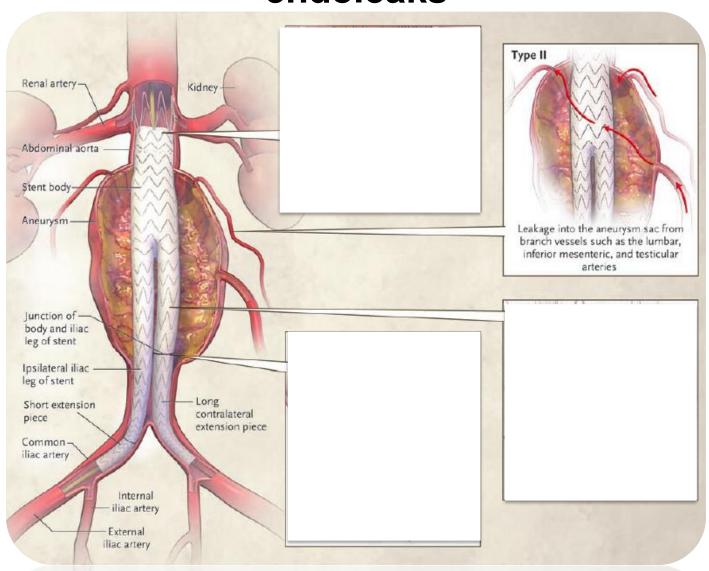




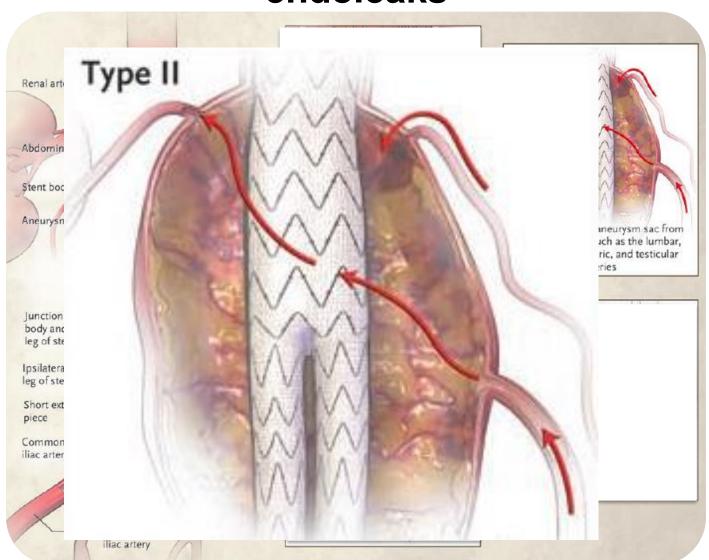




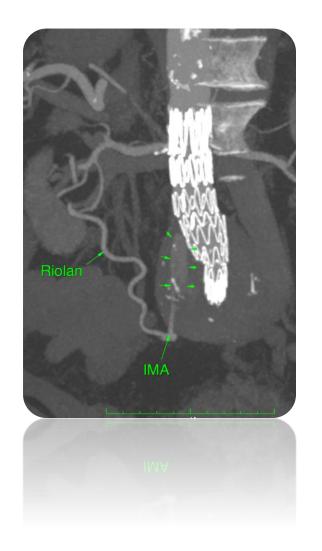


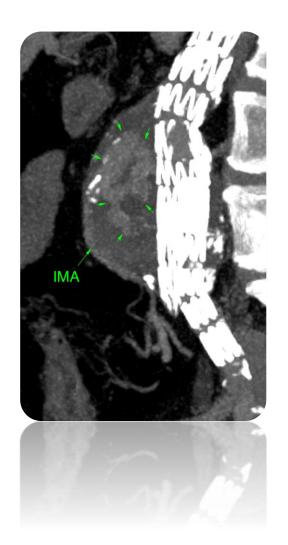


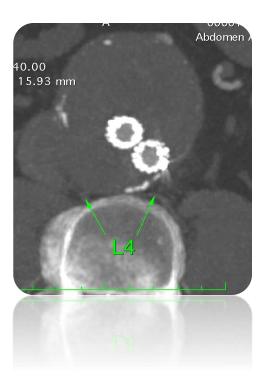




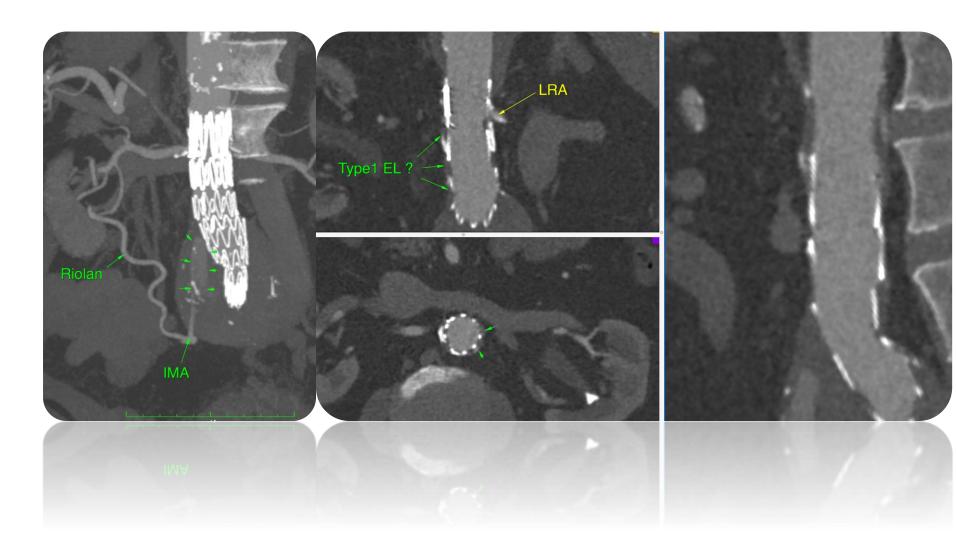




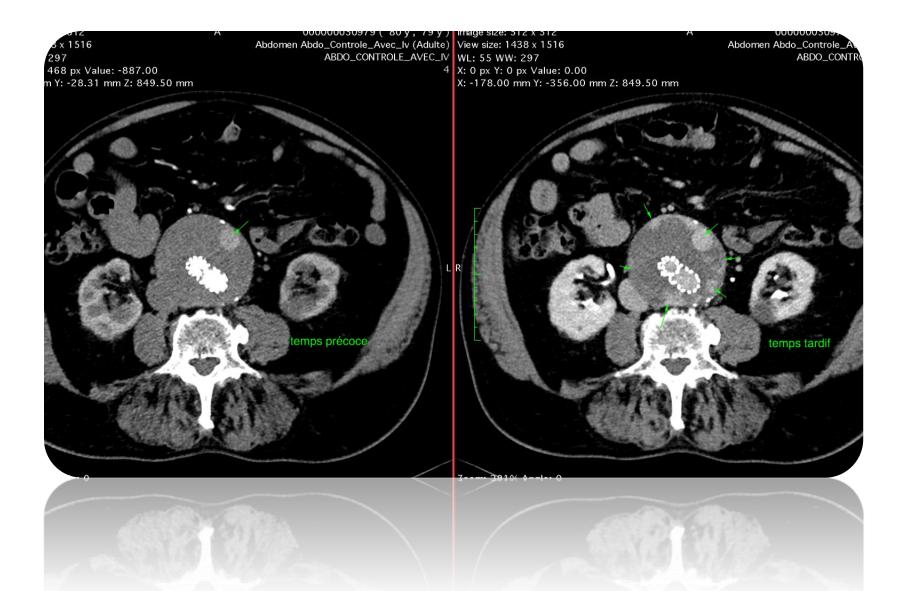




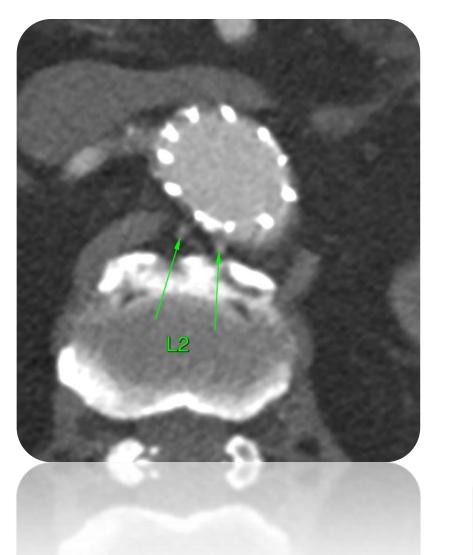


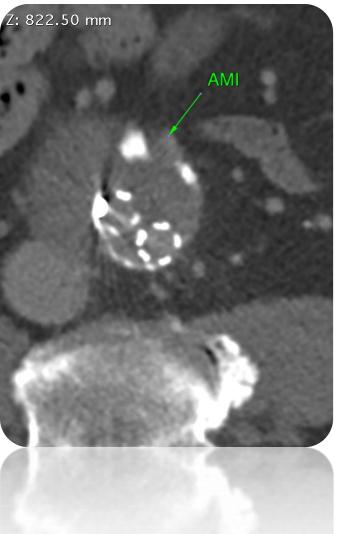




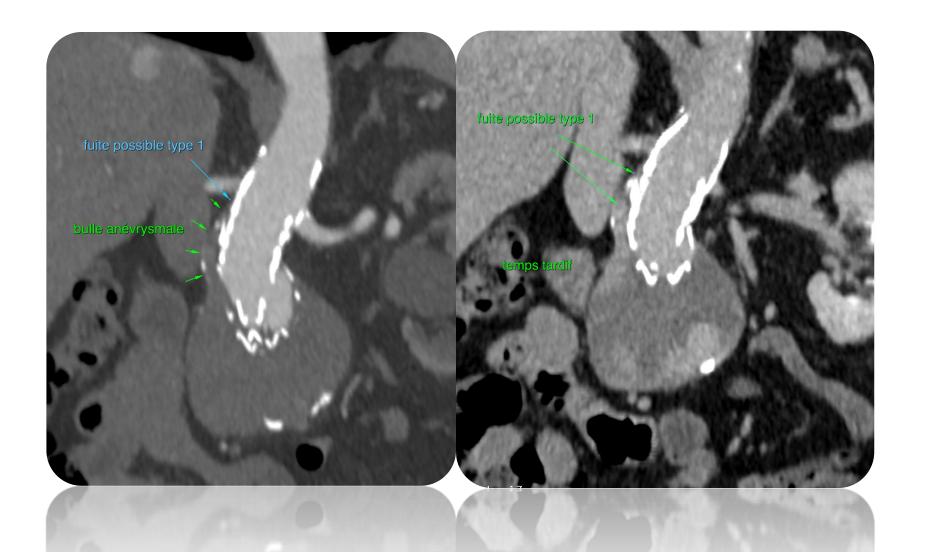














Type II endoleak with or without intervention after endovascular aortic aneurysm repair does not change aneurysm-related outcomes despite sac

Joy Walker, MD, Lue-Yen Tucker, BA, Philip Goodney, MD, Leah Candell, MD, Hong Hua, MD, Steven Okuhn, MD, Bradley Hill, MD, and Robert W. Chang, MD, San Francisco, Oakland, Santa Clara,

Objective: There is considerable controversy about the significance and appropriate treatment of type II endoleaks (T2Ls) after endovascular aneurysm repair (EVAR). We report our long-term experience with T2L management in a large

Methods: Between 2000 and 2010, 1736 patients underwent EVAR, and we recorded the incidence of T2L. Primary outcomes were mortality and aneurysm-related mortality (ARM). Secondary outcomes were change in aneurysm sac size,

Results: During the follow-up (median of 32.2 months; interquartile range, 14.2-52.8 months), T2L was identified in 474 patients (27.3%). There were no late abdominal aortic aneurysm ruptures attributable to a T2L. Overall mortality (P =.47) and ARM (P = .26) did not differ between patients with and without T2L. Sac growth (median, 5 mm; interquartile range, 2-10 mm) was seen in 213 (44.9%) of the patients with T2L. Of these patients with a T2L and sac growth, 36 (16.9%) had an additional type of endoleak. Of all patients with T2L, 111 (23.4%) received reinterventions, including 39 patients who underwent multiple procedures; 74% of the reinterventions were performed in patients with sac growth. Reinterventions included lumbar embolization in 66 patients (59.5%), placement of additional stents in 48 (43.2%), open surgical revision in 14 (12.6%), and direct sac injection in 22 (19.8%). The reintervention was successful in 35 patients (31.5%). After patients with other types of endoleak were excluded, no difference in overall all-cause mortality (P = .57) or ARM (P = .09) was observed between patients with T2L-associated sac growth who underwent reintervention and those in whom T2L was left untreated.

Conclusions: In our multicenter EVAR registry, overall all-cause mortality and ARM were unaffected by the presence of a T2L. Moreover, patients who were simply observed for T2L-associated sac growth had aneurysm-related outcomes similar to those in patients who underwent reintervention. Our future work will investigate the most cost-effective ways to select patients for intervention besides sac growth alone. (J Vasc Surg 2015; 1:1-11.)

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Early and delayed rupture after endovascular abdominal aortic aneurysm repair in a 10-year

Leah Candell, MD, Lue-Yen Tucker, BA, Philip Goodney, MD, Joy Walker, MD, Steven Okuhn, MD, Bradley Hill, MD, and Robert Chang, MD, Oakland, San Francisco, Santa Clara, and South San Francisco,

Objective: Rupture after abdominal endovascular aortic ancurysm repair (EVAR) is a function of graft maintenance of the

Methods: From 2000 to 2010, 1736 patients with abdominal aortic ancurysm (AAA) from 17 medical centers underwent EVAR in a large, regional integrated health care system. Preoperative demographic and clinical data of interest were collected and stored in our registry. We retrospectively identified patients with postoperative rupture, characterized as "early" and "delayed" rupture (≤30 days and >30 days after the initial EVAR, respectively), and identified predictors

Results: The overall follow-up rate was 92%, and the median follow-up was 2.7 years (interquartile range, 1.2-4.4 years) in these 1736 EVAR patients. We identified 20 patients with ruptures; 70% were male, the mean age was 79 years, and mean AAA size at the initial EVAR was 6.3 cm. Six patients underwent initial EVAR for rupture (n = 2) or symptomatic presentation (n = 4). Of the 20 post-EVAR ruptures, 25% (five of 20) were early, all occurring within 2 days after the initial EVAR. Of these five patients, four had intraoperative adverse events leading directly to rupture, with one type I and one type III endoleak. Of the five early ruptures, four patients underwent endovascular repair and one received repair with open surgery, resulting in two perioperative deaths. Among the remaining 15 patients, the median time from initial EVAR to rupture was 31.1 months (interquartile range, 13.8-57.3 months). Most of these delayed ruptures (10 of 15) were preceded by AAA sac increases, including three patients with known endoleaks who underwent reintervention. At the time of delayed rupture, nine of 15 patients had new endoleaks. Among all 20 patients, six patients did not undergo repair (all delayed patients) and died, nine underwent repeated EVAR, and five had open repair. For patients who underwent repair for delayed rupture, mortality at 30 days and 1 year were 44.4% and 66.7%, respectively. Multivariable Cox regression analysis identified age 80 to 89 (hazard ratio, 3.3; 95% confidence interval, 1.1-9.4; P = .03), and symptomatic or ruptured initial indication for EVAR (hazard ratio, 7.4; 95% confidence interval, 2.2-24.8; P < .01) as significant predictors of delayed rupture.

Conclusions: Rupture after EVAR is a rare but devastating event, and mortality after repair exceeds 60% at 1 year. Most delayed cases showed late AAA expansion, thereby implicating late loss of seal and increased endoleaks as the cause of rupture in these patients and mandating vigilant surveillance. (J Vasc Surg 2014;60:1146-53.)

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conclusion

Is EVAR safe @ long term?

Where exactly are the frontiers of EVAR?

Debate remains open



French Paradox?

