INVITED COMMENTARY

# Commentary on "Incidence and Clinical Impact of Stent Fractures after Primary Stenting for TASC C and D Femoropopliteal Lesions at 1 Year"

**A. Chaudhuri** *

Bedford Hospital NHS Trust, Bedfordshire Vascular Unit, Kempston Road, Bedford, United Kingdom

Femoropopliteal stents fracture, period. There are too many forces — flexion, extension, axial torsion, elongation, shortening, compression — in the femoropopliteal segment that eventually exert their toll on stents deployed therein, notwithstanding changes due to atherosclerosis itself. This possibly relates to higher fracture rates in the distal SFA-P1 segments, as indicated by Davaine et al.,[1] and the biomechanical association with the adductor hiatus (being a relatively fixed part of that segment), and proximity to the inter-epicondylar line (the axis for knee flexion), is worth considering.[2]

Recent meta-analyses cautiously support a primary stenting approach, rather than as a bail-out option, but suggest bare metal stent (BMS) fracture rates initially close to 25%.[2] Recent change-over from stainless steel to nitinol in this area is relevant, reflected in the reduced fracture rates of <5% in the Resilient and Absolute trials, examining the Lifestent (Bard Peripheral Vascular, USA) and Absolute stents (Abbott Vascular, USA), and the design proviso that stents with more flexible cell junctions do better.[3]

This study examines the significance of BMS fracture after deploying the Lifestent for TASC C/D femoropopliteal lesions, using a standardised classification system, and is thoroughly detailed. Optimal stent overlaps and numbers deployed are indicated, which have a bearing on minimising junction fractures and distraction, and the authors have taken care to treat inflow/outflow diseases that may have confounded the results. Under-ballooning of the devices is described; saying that a 6 mm balloon dilatation of a 7 mm stent causes less damage than a 7 mm balloon is rather far-fetched. This is particularly so as the stent should supposedly expand to its designated size from its shape-memory properties. I cannot see a valid argument here for "reducing medial damage" — damage already done by extensive stenting/angioplasty itself — as this is precisely why we have aggressive antiplatelet and lipid-lowering therapy in place. The IFU states stents should be very slightly oversized; therefore, for example, if a reference vessel of 6.5 mm is appropriate for a 7 mm stent, a 6 mm balloon would be inappropriate, as would be coronary analogies. In general, a 1:1 stent:balloon ratio is more appropriate. The (seemingly minor) question that then arises is does under-ballooning leave residual imperceptible kinks more prone to fracture, given the stent fracture rate of 17.7% is much higher than that reported in the Absolute trial? Also, why were bigger stents associated with more fractures? The comparison to large trees breaking and smaller plants bending is tenuous, with only 1 mm difference between stents.

Diagnostic angiography — using the rhetorical "time is tissue" mantra (this is not a cerebrovascular event) — is obsolescent practice in my view, as most centres should provide urgent computed tomography angiography (CTA), if not duplex/magnetic resonance angiography (MRA) (which provides excellent below-knee vessel images when done correctly), which reduce contrast and radiation doses.[4] In the 21st century, poor lower limb vascular imaging using MRA/CTA is largely operator-dependent.

The authors imply BMS fracture is not really a problem (despite limbs without stent fractures doing better), though this contradicts recent evidence suggesting fractures relate directly to loss of patency,[3] also indicating that duplex surveillance alone (generally not undertaken) is possibly enough — What does one believe now, given the paucity of robust trials in any case?[3]

The message perhaps is one should not insert BMS just because one can (a questionable one noted in the femoral artery in this series), especially with drug-eluting stents, drug-coated balloons, and stent-grafts already in current practice. The authors have no experience with stent-grafts such as the Viabahn — shown to be clearly superior to BMS in the Viastar trial, presented recently and currently awaiting publication — which has extremely low fracture rates, even in this segment.

In that sense, the article represents a time-warp that the endovascular community is already moving away from, perhaps with good reason, but the core message that we should not worry too much about femoropopliteal stent fracture is worth pondering over.

## REFERENCES

1 Davaine JM, et al. Incidence and the Clinical Impact of Stent Fractures after Primary Stenting for TASC C and D Femoropopliteal Lesions at 1 Year. *Eur J Vasc Endovasc Surg* 2013;**46**(2):201—12.

2 Smidt J. Biomechanical analysis of knee flexion and extension. *Biomech* 1973;**6**(1):79—80.

3 Acin F, de Haro J, Bleda S, Varela C, Esparza L. Primary nitinol stenting in femoropopliteal occlusive disease: a meta-analysis of randomized controlled trials. *J Endovasc Ther* 2012;**19**(5):585—95.

4 Chaudhuri A. Comprehensive MRA of the lower limbs including high-resolution extended-phase infra-inguinal imaging with gadobenate dimeglumine: initial experience with inter-individual comparison to the blood-pool contrast agent gadofosveset trisodium. *Clin Radiol* 2013. http://dx.doi.org/10.1016/j.crad.2013.04.013.

DOI of original article: http://dx.doi.org/10.1016/j.ejvs.2013.05.010

*E-mail address:* a.chaudhuri@ntlworld.com (A. Chaudhuri).



http://dx.doi.org/10.1016/j.ejvs.2013.05.016