Regarding “Endoleaks after endovascular aneurysm repair lead to nonuniform intra-aneurysm sac pressure”

I read with interest the article by Dias et al describing the measurement on intra-aneurysm sac pressure in patients with endoleaks post-endovascular aneurysm repair (EVAR) and congratulate the authors for their pioneering work in the area of sac pressure measurement. However, I believe it is necessary to challenge certain statements in the manuscript that apply to alternative technologic approaches to long-term surveillance of EVAR patients.

In their article, the authors state that “the results of this study show that the endoleak nidus (channel) has consistently higher pressure than the intra-sac thrombus”. Their observations that the pressure within the sac is nonuniform and that the pressure by the endoleak nidus is higher than it would be within the thrombus are consistent with previous clinical and experimental studies examining distribution of pressure resulting from an endoleak. It is important to point out that attenuation of the pressure waveform as it moves away from the source is a different physical phenomena than what has been described as “compartmentalization”, which suggests that there exist areas within the sac that are isolated from the source of increased pressure. I believe the authors are blurring this distinction and using this position to incorrectly conclude that “this varying distribution of sac pressure in patients with endoleaks, although consistently higher in expanding AAAs, may question the reliability of systems based on pressure measurements in a single spot, such as with implantable pressure sensors”.

I have been closely involved with the development of implantable pressure sensors, which are being actively studied in multiple areas of the body. Although the long-term data is still being assembled, I strongly believe that Dias et al and Carpenter JP are failing to note that the fundamental difference between the work presented in this manuscript and the use of permanently implanted sensors is that the measurements performed by the physicians in Malino represent a single moment, whereas the implantable sensors allow multiple pressure readings to be taken over time, thus providing a history of the sac’s pressure environment. The significance of this point is that since, as Dias points out, sac pressure is elevated even in the thrombus in the presence of an endoleak, serial measurements of the sac, irrespective of the exact position of the sensor within the sac, should allow the physician to prospectively see changes in pressure that signal the stent graft may be in the process of failing. This same conclusion could not be reached retrospectively with a single measurement.

I believe that the use of implantable pressure measurement systems will become an important tool in management of the post-EVAR patient. Clinical trials to prove the long-term efficacy of implantable pressure sensors are currently being designed and I actively look forward to future reports detailing the chronic use of these devices.

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plantable pressure sensors. Our study is the first report on late pressure measurements in the endoleak nidus (flow channel) and the thrombus with the same technique and the same a differing pressure gradient between these two locations. The previously reported association of aneurysm shrinkage with intra-sac depressurization in the absence of endoleaks seems also to be present with endoleaks. The degree of depressurization seems nevertheless to be different. Pressure measurements in the presence of an endoleak need, therefore, to be assessed cautiously, no matter how the measurement was obtained.

Implantable pressure sensors have the advantage of allowing repeated measurements over time.5,6 We share the view that pressure sensors may eventually become useful in the follow-up of patients after EVAR. Before that happens, the aforementioned issues common to all pressure measurement systems in the presence of endoleaks need to be solved and the long-term accuracy of implantable sensors needs to be established. Implantable devices have been validated in the immediate period after EVAR, but thrombus can change with time acquiring a non-uniform structure7-9 that can influence transmission of pressure.8,9 Some of these issues are expected to be answered by ongoing trials, which will hopefully include a validation against the validated direct intra-aneurysm sac pressure measurements (DISP) with tip-pressure sensors.

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Regarding “Does simvastatin save lives? If so, when and in whom?”

In the Heart Protection Study,1 it was suggested that cholesterol-lowering therapy with simvastatin can reduce the risk of major vascular events among people with peripheral artery disease (PAD). However, a clearly defined effect on total mortality was not reported. In 1992, four of the initiators of the Heart Protection Study (HPS) called for larger total mortality trials to generate data in six named patient subgroups.2 In 2001, after their study was completed, the press release started with the words “LIFE-SAVER” but it gave no data on deaths.3 Similarly, the current HPS report avoided the subject by curiously combining deaths with aneurysm repairs, only to find a small increase on simvastatin for this combined endpoint.

Furthermore, HPS found no significant mortality benefit in women, and it now seems that this could also be true for patients with baseline PAD.1 The authors admitted that much of their combined endpoint benefit in PAD patients was in revascularizations, and after they retrospectively redefined “peripheral vascular events” to include all noncardiac revascularizations including carotid procedures.

Revascularizations are procedures that may or may not affect mortality or future cardiovascular events,4 and it is, thus, important to report deaths and true disease endpoints separately. Regardless, despite the absence of noncombined endpoint numbers and numbers needed to treat, the article and its debating author, Dr Bulbulia, stated repeatedly that patients with PAD “should be” on statin (presumably for life) and that there “should be no threshold for initiation of statin therapy”.5 We believe that this is not supported by the grouped endpoint data presented, especially since total deaths are not clearly reported for all participant groups.

Interestingly, HPS, 4S and LIPID6 are the only three large statin trials to show a brief period of mortality benefit, almost certainly in some men only. Such benefit appeared after about 1.5 years of use and ended about 2 or 3 years later. Such time-dependent and time-limited mortality effect can be shown by releasing the relevant disease and group specific time curves individually.

The authors of HPS should therefore finally release a table for total deaths, heart attacks, amputations, and other disease endpoints and related numbers needed to treat, with confidence levels, in women, men, and diabetics, and in this case for PAD patients for 1, 3, and 5 years of simvastatin treatment regarding these endpoints. Without such curves and year-by-year disease and group specific numbers needed to treat, prescribers lack crucial data relevant to their patients. Patients deserve to be told their odds of avoiding death and each of the various lesser disease endpoints and for how long they need to take statin to attain these results, and when the effect may no longer exist or be incremental.

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