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 confirms a difference in pressure gradient between these two locations. The previously reported association of aneurysm shrinkage with intra-sac depressurization in the absence of endoleaks[2] 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.[3,4] 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 structure[5-7] that can influence transmission of pressure.[8,9] Some of these issues are expected to be answered by on-going trials, which will hopefully include a validation against the validated direct intra-aneurysm sac pressure measurements (DISP) with tip-pressure sensors.

*Nuno V. Dias, MD, PhD*
*Krassi Ivancev, MD, PhD*
*Timothy A. Resch, MD, PhD*
*Martin Malina, MD, PhD*
*Björn Sonesson, MD, PhD*

UMAS, Malmo University Hospital
Malmo, Sweden

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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 event 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*".[1] 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[5] and LIPID[6] 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.

*Eddie Vos, M. Eng*

Sutton (QC), Canada

*Luca Mascitelli, MD*

Comando Brigata alpina "Julia"
Udine, Italy

*Colin P Rose, MD*

McGill University
Montréal (QC), Canada

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## Reply

Previously published results from the Heart Protection Study (HPS) show clearly that lowering LDL cholesterol by about 1 mmol/L (38 g/dL) with simvastatin produced a highly significant 13% (SE 4) relative reduction in all-cause mortality (1328 [12.9%] simvastatin-allocated vs 1507 [14.7%] placebo-allocated deaths; $P = .0003$) during the scheduled 5-year treatment period.[1] This very definite survival benefit reflected the combined impact of a highly significant 17% (SE 4) relative reduction in vascular deaths (781 [7.6%] vs 937 [9.1%]; $P < .00001$) and of a nonsignificant difference in nonvascular mortality (547 [5.3%] vs 570 [5.6%]; $P = .4$). A subsequent report showed that there were similar relative reductions in vascular deaths (and nonfatal major vascular events), with no evidence of any adverse effects on nonvascular deaths (or cancers), in a range of different circumstances (including among women).[2] The reduction in vascular mortality started to emerge during the first year of statin treatment and increased during each subsequent year of treatment, with no adverse effect on nonvascular mortality emerging during or after the scheduled treatment period.[1-3]

Meta-analyses of individual patient data from large randomized trials (including HPS) have reliably demonstrated that statin therapy reduces vascular mortality substantially, while producing little or no effect on nonvascular mortality.[4] Consequently, the relative reduction in all-cause mortality in some particular circumstance is determined not only by the size of the relative reduction in vascular mortality with statin therapy but also by the ratio of vascular to nonvascular deaths. Moreover, separate assessment of the effects of statin therapy on vascular mortality and on nonvascular mortality in such circumstances (considered in the context of the overall findings for cause-specific mortality and for the much larger numbers of nonfatal vascular and nonvascular events) is likely to provide a more sensitive assessment of any benefits and hazards than would direct comparisons of deaths from all-causes.

With regard to the subgroup of patients in HPS with peripheral arterial disease (PAD), the observed 10% (95% CI -5-12) relative reduction in vascular mortality (10.2% simvastatin vs 11.2% placebo) was not significantly different from the 23% (12-32) relative reduction observed among the other high-risk patients studied (heterogeneity $P$ value = .1). Moreover, this lack of heterogeneity of benefit with statin therapy was reinforced by the similar (heterogeneity $P$ value = .5), and highly significant, relative reductions in major vascular events (MVE): (ie, vascular deaths, heart attacks, strokes, and revascularizations) among patients with PAD (22% [SE 4]; $P < .0001$) and the other high-risk patients (25% [SE 3]; $P < .0001$).[5] The apparent lack of effect on the small number of aneurysm deaths or repairs should be considered in the context of these large reductions in vascular events. In terms of the absolute benefits in HPS, allocation to statin therapy prevented 63 (11) first MVEs, and 116 (21) first and subsequent MVEs, per 1000 PAD patients. This corresponds to a "number needed to treat" to prevent a first MVE of 16 (SE 3), although this underestimates the benefit of actually taking a statin because only about two-thirds of patients complied with their allocated treatment during the 5-year study period. In terms of safety, as was observed overall in HPS, there was no apparent effect on nonvascular mortality among the patients with PAD (7.3% simvastatin vs 7.7% placebo; hazard ratio 0.94; 95% CI 0.79-1.12; $P$ = NS).

In conclusion, HPS has shown that 40 mg simvastatin daily safely reduces both vascular mortality and major vascular morbidity in patients with PAD and in other high-risk patients, without adverse effects on nonvascular mortality or major nonvascular morbidity. It is, therefore, entirely reasonable to conclude that vascular surgeons should consider statin therapy for all patients with proven PAD irrespective of age, gender, and baseline lipid levels.

*Richard Bulbulia*
*Louise Bowman*
*Karl Wallendszus*
*Sarah Parish*
*Jane Armitage*
*Rory Collins*

CTSU, University of Oxford
Oxford, United Kingdom

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## Regarding "Light assisted stab phlebectomy: Report of a technique for removal of lower extremity varicose veins"

In the article by Lawrence and Vardanian,[1] the authors reported on light assisted stab phlebectomy. I was amazed by how a simple procedure can be made so eloquently complicated. We have performed stab avulsion phlebectomies on more than 2000 patients in our office without sedation. We use 1% local lidocaine anesthetic injected only into the site of the 2 mm puncture over the previously marked vein. Tumescent anesthesia is not required or used since we find that it oozes from the incision making application of Steri-strip closure unreliable. Provided that the vein itself is removed, without any adjacent subcutaneous tissue, patients feel no pain. At most, they notice a pulling sensation. Simple finger pressure for a minute or two prevents bleeding even from a large varicosity.

No sutures are required to close the incision but simply Steri-strips. We do not wrap the leg but rather use a 30 to 40 mm