

REVIEW

Aneurysm Sac Pressure after EVAR: The Role of Endoleak

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Objective. The relation between endoleak and aneurysm sac pressure is not completely clear. This review evaluates the effect of endoleaks on aneurysm sac pressure and summarizes the present knowledge regarding aneurysm sac pressure after EVAR.

Methods. A systematic search of literature was carried out using MEDLINE, EMBASE and Web of Science. Studies were included if aneurysm sac pressure measurements as well as systemic pressure measurements were performed during or after EVAR. Mean pressure indices (MPI), ratio mean aneurysm sac pressure to mean systemic pressure, in the absence of endoleaks and in the presence of different type of endoleaks were compared.

Results. Stent-graft deployment does not seem to result in immediate reduction of aneurysm sac in the absence of an endoleak. Aneurysm sac pressure is elevated in the presence of an endoleak. However, the MPIs differ widely between studies both in the absence and presence of an endoleak.

Conclusion. MPI is not specific to the type of endoleak. This implies that the same type of endoleak does not necessarily pose the same MPI and by this the same hazard of aneurysm rupture, because the aneurysm sac pressure is directly related to the aneurysm wall stress.

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Keywords: EVAR; Endoleak; Endotension; Abdominal aortic aneurysm; AAA; Pressure; Follow-up.

Introduction

Endovascular aneurysm repair (EVAR) was introduced in 1991 as a less-invasive alternative for abdominal aortic aneurysm (AAA) therapy.^{1,2} EVAR aims at prevention of aneurysm rupture with exsanguinations and acute death. Endoleaks are the Achilles heel of EVAR. An endoleak is defined as persistence of blood flow outside the stent-graft, but within the aneurysm sac. Endoleaks occur approximately in 20% of the patients treated by EVAR.³

The absence of an endoleak on conventional imaging tools, such as Computer tomography (CT) or angiography, does not exclude the possibility of high pressure in the aneurysm sac and the persistent risk of rupture.⁴ Therefore the concept of endotension is formulated as persistent or recurrent pressurization of the aneurysm sac following endovascular repair.⁵

The success of EVAR relies on the extent of isolation of the aneurysm sac from systemic blood flow

and systemic pressure. The evaluation of this extent of isolation is difficult. Firstly, it is not known what happens to the aneurysm sac pressure after EVAR without detectable endoleak nor is it known how much pressure is required to cause rupture. Secondly, it is difficult to predict whether and when re-intervention is justified in the presence of endoleaks, solely based on imaging. Graft related endoleaks (Type I and III) are associated with a risk of late rupture and it is assumed but not proven that this is because such endoleaks are associated with significant pressurization of the aneurysm sac.⁴ The treatment of endoleaks from collateral back-flow (Type II) remains controversial. Many state that Type II endoleaks will seal.^{4,6} It has been proposed that most Type II endoleaks that seal are those detected at the original procedure.⁴ Little is known about their impact on the aneurysm sac pressure and the risk of aneurysm rupture. The clinical significance of Type V endoleaks, defined as aneurysm growth without detectable endoleaks, also remains uncertain.

Elucidation of the relationship between endoleak and pressure may help clinical decision making. This review evaluates the effect of endoleaks on aneurysm

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sac pressure and summarizes the present knowledge regarding aneurysm sac pressure after EVAR.

Methods

Search strategy for identification of studies

A systemic search of literature was conducted until December 2006 using PubMed, EMBASE and Web of Science. Our search strategy is given in Table 1 (PubMed), Table 2 (EMBASE) and Table 3 (Web of Science). There was no restriction on language.

Criteria for considering studies for this review

Articles of in-vitro, animal and patient-studies were selected by Pubmed, EMBASE and Web of Science, respectively. The abstracts of each article were studied after checking for duplication between the databases. If it appeared that aneurysm sac pressure measurements was concurrently performed with systemic pressure measurements during or after EVAR the full text was studied. Additional articles were sought by checking the reference lists of the relevant articles.

Data extraction

Particular attention during evaluation of the selected studies was paid to the type of study (in-vitro, animal or patient), the pressure measurement technique, the presence or absences of endoleaks, the time of pressure measurement and the used analysis of pressure measurements. The endoleak classification is given in Table 4.⁶

The interval between pressure measurement and EVAR might be relevant for the interpretation and

Table 1. Search strategy used for PubMed

Search
#1 "aortic aneurysm" [MeSH] OR aortic aneurysm* [Text word]
#2 "stents" [MeSH] OR stent* [Text Word]
#3 "blood vessel prosthesis" [MeSH] OR "blood vessel prosthesis implantation" MeSH] OR blood vessel prosthesis* [Text Word]
#4 evar [All Fields] OR "endovascular therapy" [All Fields]
#5 #2 OR #3 OR #4
#6 #1 AND #5
#7 endovascular [All Fields] AND ("aneurysm" [MeSH Terms] OR aneurysm* [Text Word]) AND (repair* [Text Word] OR treat* [Text Word] OR therap* [Text Word] OR explode therapy [Subheading] OR "therapeutics" [MeSH])
#8 "pressure" [MeSH] OR pressure* [Text Word] OR "manometry" [MeSH] OR manomet* [All fields] OR leak* [All Fields] OR endoleak* [All Fields] OR tension [All fields] OR endotension [All Fields]
#9 #8 AND (#6 OR #7)

Table 2. Search strategy used for EMBASE (OVID)

Search
1 exp aorta aneurysm/OR aortic aneurys\$.ft
2 stent/OR stent\$.ft
3 exp blood vessel prosthesis/OR blood vessel prosthe\$.ft
4 endovascular therap\$.ft
5 evar.ft OR endovascular aneurysm repair.ft
6 endovascular aneurysm treat\$.ft OR endovascular aneurysm therap\$.ft
7 (1 AND (2 OR 3 OR 4)) OR (5 OR 6)
8 pressure/OR pressure measurement/OR pressure\$.ft
9 manometry/OR manomet\$.ft
10 tension/OR tension.ft
11 leakage.ft OR endotens\$.ft OR endoleak\$.ft
12 exp prosthesis failure/OR prosthesis fail\$.ft
13 8 OR 9 OR 10 OR 11 OR 12
14 7 AND 13

comparison of measurements. Therefore, we categorized the studies, in which an endoleak was absent, in 4 groups (Group 1: < 1 week after EVAR, Group 2: 1 week to 1 year after EVAR, Group 3: 1 to 2 years after EVAR, Group 4: 2 to 4 years after EVAR). We divided the studies, in which a Type II endoleak was present, in 2 groups (Group 1: 0 to 1 month after EVAR, Group 2: > 1 month after EVAR). Time of pressure measurement is probably less relevant in the presence of a Type I and III leak, because there is a direct connection between the systemic circulation and the aneurysm sac. Consequently, it is not likely that the ratio between aneurysm sac pressure and systemic blood pressure changes over time. Type IV and V endoleak studies were not categorized in moment of pressure measurement, because these endoleaks are investigated only in a small number of studies.

Analysis of data

Peripherally measured systolic and diastolic pressure does not always reflect corresponding pressures in the aorta. The systolic pressure in the brachial artery over-estimates the central aortic systolic pressure.⁷ So the comparison of the ratios between the systolic or

Table 3. Search strategy used for Web of Science

Search
#1 TS = (pressure* or manomet* or leak* or endoleak* or tension or endotension)
#2 TS = aortic aneurysm*
#3 TS = (stent* or blood vessel prosthe* or evar or endovascular therap*)
#4 TS = endovascular
#5 TS = (repair* or treat* or therap*)
#6 #3 AND #2
#7 TS = aneurysm*
#8 #7 AND #5 AND #4
#9 #8 OR #6
#10 #9 AND #1

Table 4. Classification of endoleaks

Endoleaks (type)	Description
I	Attachment site
II	Collateral vessel
III	Failure of graft
IV	Porosity of graft wall
V	Endotension

pulse aneurysm sac and the systolic or pulse systemic pressure between studies, in which the systemic blood pressure is measured centrally, and studies, in which this pressure is measured peripherally, is biased. However, the mean pressure is virtually identical in central and peripheral arteries.⁸ Therefore, we chose to evaluate the mean pressure index (MPI) to enable meaningful comparisons. The MPI is the ratio between the mean aneurysm sac pressures and the mean systemic blood pressure (MPI). If no single aneurysm sac and systemic pressure were given, we depicted the ratio between the reported mean or median aneurysm sac pressure and the mean or median systemic blood pressure of the experiments.

Data were stored in a database for analyses (Microsoft Excel 2002, Redmond, USA). The mean pressure

indices (MPIs) were compared between studies. MPIs were depicted in Box & Whisker plots.

The effect of stent-graft deployment and the occurrence of endoleaks on the pulse pressure in the aneurysm sac were evaluated as well. Because of the difference between centrally and peripherally measured blood pressures, no quantitative analysis of the pulse pressure was performed.

Results

Aneurysm sac pressure without endoleak

Dampening of pulse pressure after stent-graft deployment without endoleak was observed in all studies. Pulse pressure in the aneurysm sac was never totally eliminated.^{9–35}

Patient-studies

In literature, the mean pressure index (MPI) in patients after EVAR without detectable endoleak ranges from 0 to 1.2 (Fig. 1A–D, red lines).^{9–36} Patient-studies demonstrate that stent-graft deployment

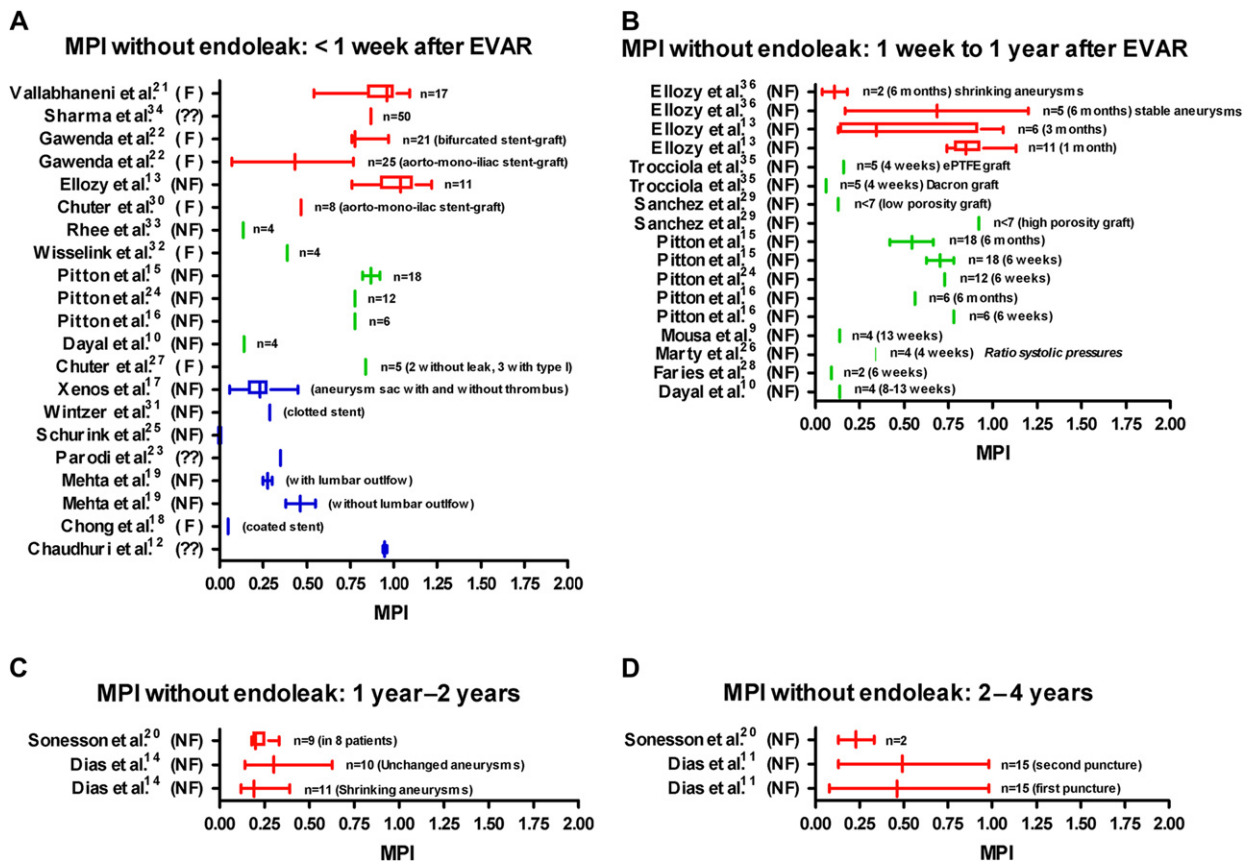


Fig. 1. MPI without endoleak in patient-studies (red), animal-studies (green) and in-vitro studies (blue). Type of pressure measurement device is given by (??) unknown, (F) fluid filled or (NF) non-fluid filled.

does not usually result in immediate decrease of intra-sac pressure. However, Chuter *et al.* and Gawenda *et al.* recorded a lower MPI immediately after aneurysm exclusion than other patient-studies (Fig. 1A, red lines).^{22,30} They deployed aorto-mono-iliac stent-grafts instead of bifurcated. Other patient-studies confirm (Fig. 1A–D, red lines) the suggestion that time is required before pressure reduction in the aneurysm sac takes place. Ellozy *et al.* performed experiments with wireless pressure sensors to monitor aneurysm sac pressure continuously. They demonstrated that after EVAR the ratio of aneurysm sac pressure to systemic pressure decreased, except in 2 patients, from the time of implantation to 1 month and 3 months, respectively (Fig. 1B, red lines).¹³

Animal-studies

In animal studies, it has also been reported that the intra-aneurysm sac pressure after EVAR without endoleak is not immediately eliminated. Pitton *et al.* reported a pressure reduction during the period from 6 weeks to 6 months after EVAR (Fig. 1B, green lines).^{15,16}

In-vitro studies

In in-vitro studies, recorded MPIs are consistently lower than those reported in in-vivo studies. All in-vitro studies, except the study by Chaudhuri *et al.*,¹² reported a MPI less than 0.55 after successful exclusion of the aneurysm sac (Fig. 1A, blue lines).

Aneurysm sac pressure with type I endoleak

All studies, in which the effect of type I endoleak on the pulse pressure was investigated, demonstrated that aneurysm sac pulse pressure is less reduced in the presence of type I endoleaks than in the absence of type I endoleaks.^{12,18,21,30,37}

Patient-studies

The MPI in patients with type I endoleak ranges from 0.76 to 1.08.^{21,30,37} (Fig. 2, red lines).

Animal-studies

Criado *et al.* investigated only the aneurysm sac pressure in animals in the presence of a type I endoleak. The pressure differential between the aneurysm sac pressure and systemic pressure was in 3 dogs <5 mmHg (MPI 1) (Fig. 2, green line).³⁸

In-vitro studies

The MPI in in-vitro studies, in which type I endoleaks are investigated, varies from 0.26 to 1.07.^{12,17,18} The

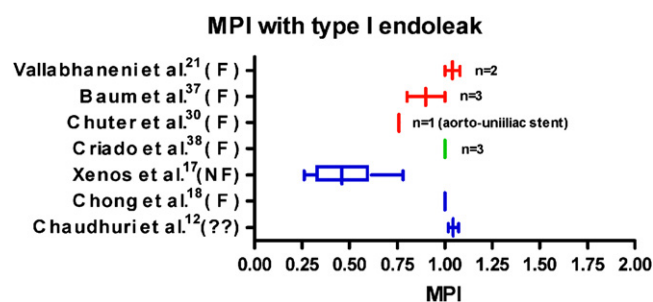


Fig. 2. MPI with type I endoleak in patient-studies (red), animal-studies (green) and in-vitro studies (blue). Type of pressure measurement device is given by (??) unknown, (F) fluid filled or (NF) non-fluid filled device.

MPIs reported by Xenos *et al.* are lower than those reported by other studies (Fig. 2, blue lines).¹⁷

Aneurysm sac pressure with type II endoleak

In spite of the presence of a type II endoleak, the pulse pressure in the aneurysm sac is reduced when compared to the systemic pulse pressure.^{9,10,12–16,21,33,37,39,40}

Patient-studies

MPIs in the first month after EVAR, in patients with type II endoleaks, vary between 0.5 and 1.0. However, aneurysm sac pressure during this period appears similar to systemic pressure in most patients with type II endoleak (Fig. 3A, red lines).^{12,13,15–17,21,33,36,37,39,41–43}

MPIs, measured at more than 1 month after EVAR, in patients with type II endoleaks, range from 0.2 to 1.0.^{9,10,13–16,33,44} Ellozy *et al.* measured aneurysm sac pressure immediately after EVAR in a patient with a type II endoleak, at 1 month and at 3 months after EVAR. The MPI decreased from 1.0 to 0.95 and 0.72, respectively.¹³ MPIs, measured by Dias *et al.* more than 1 month after EVAR (Fig. 3B), are generally lower than those measured in the first month after EVAR (Fig. 3A). These findings imply that aneurysm sac pressure also decreases after a time interval in spite of the presence of a persisting type II endoleak. However, aneurysm sac pressure does not always decrease in the presence of type II endoleak (Fig. 3B).⁴⁴

Animal-studies

The MPIs in animals in the first month after EVAR ranges from 0.33 to 0.85 (Fig. 3A, green lines) and at more than 1 month after EVAR from 0.42 to 0.94 (Fig. 3B, green lines).^{9,10,15,16,33,40–42} Pitton *et al.*

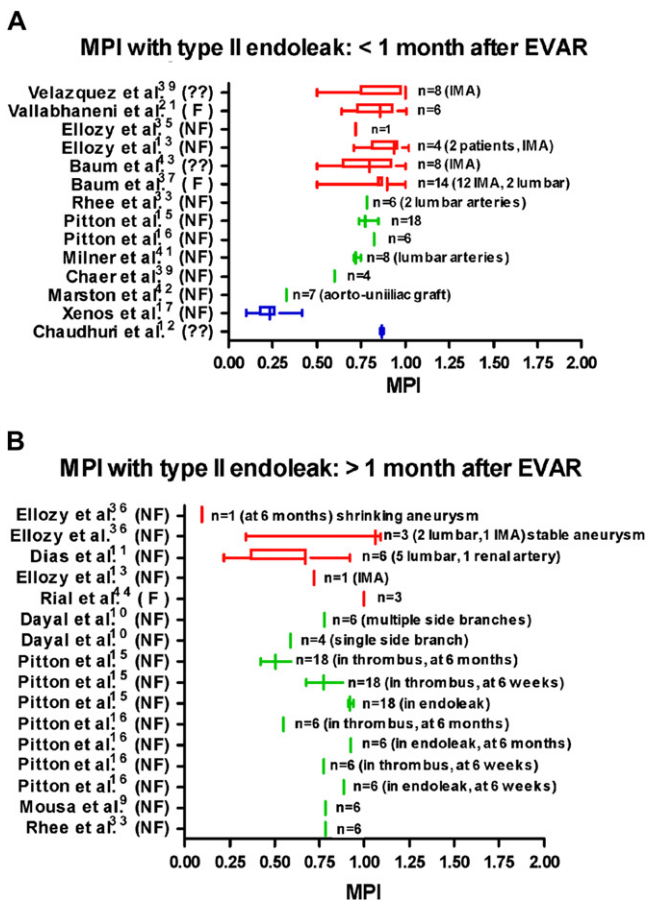


Fig. 3. MPI with type II endoleak in patient-studies (red), animal-studies (green) and in-vitro studies (blue). Type of pressure measurement device is given by (??) unknown, (F) fluid filled or (NF) non-fluid filled device.

demonstrated also a pressure reduction during the time. The MPI after 6 months was lower than after 6 weeks (Fig. 3B).^{15,16}

In-vitro studies

The MPIs in in-vitro studies, in which type II endoleaks are investigated, ranges from 0.10 to 0.88 (Fig. 3A, blue lines).^{12,17} Xenos *et al.* did not demonstrate in their in-vitro model a significant increase in the aneurysm sac pressure in the presence of flow through a type II endoleak (Fig. 3A).¹⁷

Aneurysm sac pressure with type III endoleak

The pulse pressures in the aneurysm sacs with type III endoleaks were reduced, but to a lesser extent than without endoleak.^{19,23,25,26,31} Aneurysm sac pulse pressure was also depended on the presence or absence of outflow from the aneurysm sac.^{19,23}

Animal-studies

The MPIs in animal-studies, in case of type III endoleak, range from 0.27 to 1.00 (Fig. 4, green lines).^{26,45}

In-vitro studies

In-vitro studies with type III endoleaks demonstrated a MPI from 0 to 1.96 (Fig. 4, blue lines).^{19,23,25,31} Wintzer *et al.* investigated aneurysm sac pressure in an in-vitro model. They demonstrated that the MPI depended on the presence of outflow from the aneurysm sac and on the pressure inside this outflow channel. In the absence of outflow through collateral vessels the mean pressure in the aneurysm will increase to mean pressure of the systemic circulation. However, type III endoleak with free outflow without resistance through the open inferior mesenteric artery (IMA) resulted in an aneurysm sac pressure of 0 mmHg. In the presence of the IMA, with a pressure of 100 mmHg, the aneurysm sac pressure appeared to be 96 mmHg.³¹ All these conditions are illustrated in Fig. 4.

Furthermore, Mehta *et al.* demonstrated that the aneurysm sac pressure is equivalent to that of the systemic circulation if a type III endoleak and lumbar outflow are present. In the presence of type III endoleak without lumbar branch outflow, the aneurysm sac pressure was higher than that of the systemic circulation.¹⁹ This corresponds with the findings by Parodi *et al.* (Fig. 4).²³

Aneurysm sac pressure with type IV endoleak

Three studies, one animal (Fig. 5, green line) and two in-vitro studies (Fig. 5, blue lines), are performed to investigate the effect of stent-graft porosity on the

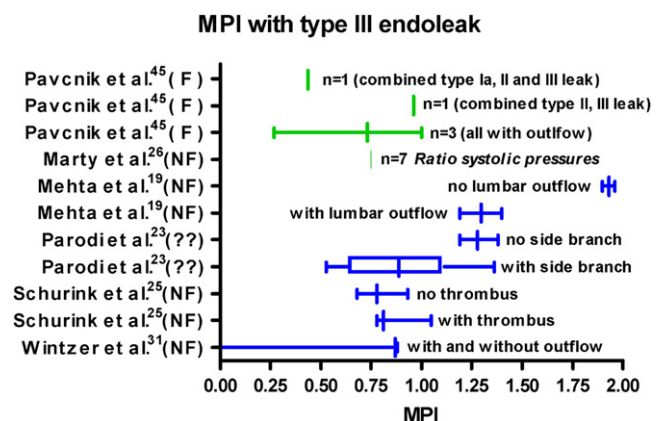


Fig. 4. MPI with type III endoleak in patient-studies (red), animal-studies (green) and in-vitro studies (blue). Type of pressure measurement device is given by (??) unknown, (F) fluid filled or (NF) non-fluid filled device.

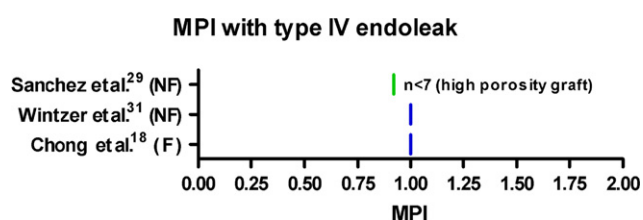


Fig. 5. MPI with type IV endoleak in patient-studies (red), animal-studies (green) and in-vitro studies (blue). Type of pressure measurement device is given by (F) fluid filled or (NF) non-fluid filled device.

aneurysm sac pressure. Although a type IV endoleak was present, the pulse pressure in the aneurysm sac was reduced in all studies.^{18,29,31} These studies demonstrate that graft porosity causes systemic pressure in the aneurysm sac (MPI > 0.9) (Fig. 5).

Aneurysm sac pressure with type V endoleak

Patient-studies

The MPIs in patients with expanding aneurysms without detectable endoleak vary from 0.27 to 1.12 (Fig. 6, red lines).^{14,46–50}

Dias *et al.* measured MPIs in patients with shrinking, stable and expanding aneurysms after EVAR without detectable endoleak. Although the number of patients was too small for definitive conclusion, they associated a MPI above approximately 0.35 with a subsequent AAA expansion.¹⁴ This corresponds with findings of other studies (Fig. 6).

Animal-studies

The MPIs with type V endoleak in the animal-study by Skillern *et al.* vary from 0.91 to 1.01 (Fig. 6, green line).⁵¹

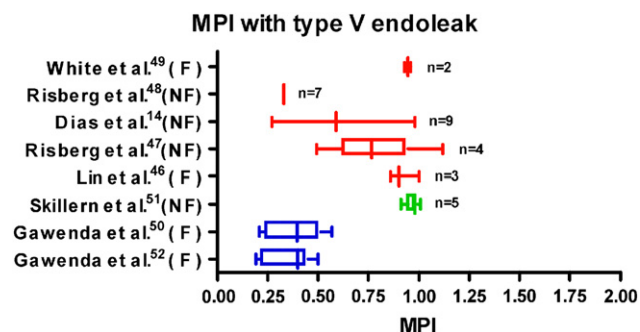


Fig. 6. MPI with type V endoleak in patient-studies (red), animal-studies (green) and in-vitro studies (blue). Type of pressure measurement device is given by (F) fluid filled or (NF) non-fluid filled device.

In-vitro studies

Gawenda *et al.* investigated determinant of endotension in an in-vitro study. The MPIs range from 0.19 to 0.57.^{50,52}

Discussion

This review demonstrates that the MPI after EVAR without detectable endoleak differ widely between studies. This can be understood since the aneurysm sac pressure is multifactorial. The presence of efferent side branches, the size of endoleak, the type of graft, the mechanical properties of the aneurysm wall and the aneurysm volume have been investigated as determinants of aneurysm sac pressure.^{18,31,50,52,53} Stent-graft deployment, in absence of an endoleak, does not result in immediate reduction of intra-sac pressure of many patients (Fig. 1A).

Since aneurysm sac pressure is a direct determinant of aneurysm wall stress, the risk of aneurysm rupture is not immediately reduced after successful EVAR. Pressure reduction in the aneurysm sac takes some time varying from 1 week to two years in most cases whereas sometimes pressure reduction never takes place (Fig. 1A–D). Theoretically, the aneurysm sac pressure decreases only if there is an outflow or resorption since the aneurysm sac is filled with incompressible material. The rate of pressure reduction depends on the outflow resistance in the efferent vessels and the rate of resorption which is influenced by biochemical factors. Hence it is understandable that time is needed before pressure reduction will take place.

The MPIs in in-vitro studies are lower. This might be explained by misdiagnosing endoleaks in in-vivo studies. However, differences between the in-vitro experimental set-up and the in-vivo situation also contribute to the low MPI in in-vitro studies. In-vitro models are only appropriate to evaluate aneurysm sac pressure after successful EVAR if the stent-graft is deployed in a running artificial circulation. During some in-vitro studies the aneurysm sac was separately filled after deployment of a stent-graft.^{12,23,25} Of course, the aneurysm sac pressure in this set-up depends on the amount of liquid injected in the aneurysm sac and misrepresents the in-vivo aneurysm sac pressure after successful stent-graft deployment.

Pulse pressure is reduced after EVAR. Persistence of systemic pulse pressure in the aneurysm sac and aneurysm pulsatility have been considered as an important guide to evaluate the success of EVAR.^{21,54} However, Mehta *et al.* demonstrated that the

aneurysm pulsatility did not correlate with the absolute aneurysm sac pressures, but appeared to be dependent on the presence or absence of side-branch outflow. Therefore, they concluded that aneurysm pulsatility is an unreliable guide to predict aneurysm sac pressurization.¹⁹

Endoleak

The type of endoleak does not directly correlated to the aneurysm sac pressure, because great differences exist between studies in which identical type of endoleaks are investigated. We will discuss the results of various types of endoleaks separately.

Type I and III endoleak

Type I and III endoleaks are considered as the most dangerous, even if sealing appears to have occurred.⁴ Ruptures have been reported with EVAR associated with these types of endoleaks.³ The level of MPIs in Figs. 2 and 4 are probably high enough to contribute to aneurysm sac rupture. These endoleaks are often associated with "systemic" pressure in the aneurysm sac. However, even in the presence of these endoleaks, not every study demonstrates MPIs of 1.0 (Figs. 2 and 4). Differences between studies are probably caused by the presence or absence of outflow from the aneurysm sac. The levels of the MPI are less in the presence of an outflow channel (Fig. 4).^{19,23,31} Theoretically, the pressure in the outflow channel influences the aneurysm sac pressure since it determines the outflow resistance. Low outflow resistance enhances its ability to depressurize the aneurysm sac (also in the presence of an endoleak).

Type IV

If the stent-graft is permeable for blood (type IV endoleak), the MPI will be around 1.0 (Fig. 5).^{18,29,31} The pressure reduction in the aneurysm sac after stent-graft placement was inversely correlated with the porosity of the graft material.²⁹

Type V

The mechanism of type V endoleaks is still unclear. Endotension may represent a very low flow endoleak that is not visualized with conventional imaging. Risberg *et al.* described a theory that degradation of aneurysm sac thrombus with formation of proteins and protein particles may cause osmotic pressure.⁴⁷ Ultrafiltration through a PTFE graft (persistent type IV) has also been discussed as cause of endotension.^{47,55} Although the pathogenesis of endotension (AAA growth without detectable endoleak) remains unclear, it is

hypothesized that an elevated aneurysm sac pressure is a contributory factor.⁴⁹ Several studies demonstrated significant aneurysm sac pressures in the presence of endotension (Fig. 6).^{14,46–49} Pressure transmission through thrombosed attachment site failure (thrombosed type I) has also been considered as cause of endotension.⁵¹ However, elevated aneurysm sac pressure itself is probably not the only explanation for endotension since an increase in sac volume cannot occur without accumulation of more luminal content.⁴⁹

Type II

In the presence of a type II endoleak, all patient studies demonstrated a significant pressurization of the aneurysm sac in the first month after EVAR with type II endoleaks (Fig. 3A). However, in view of the fact that the aneurysm sac is not depressurized immediately after EVAR in *absence* of any endoleak (Fig. 1A), it is not absolutely certain that "systemic" aneurysm sac pressure is caused by the *presence* of a type II endoleak.

Side-branches can act as afferent (feeding) and efferent (outflow) vessels depending on the pressure gradient between aneurysm sac and side branch.^{16,18}

Vallabhaneni *et al.* measured pressures within patent lumbar arteries after restoration of iliac blood flow during open aneurysm surgery.²¹ The pressure did not rise to the levels recorded within the aneurysm after endograft deployment. They suggest that retro-grade perfusion via patent side-branches cannot be the only explanation for maintenance of pressure within the aneurysm sac.

Since the mean aneurysm sac pressure immediately after *successful* EVAR is similar to the systemic mean pressure (Fig. 1A), the aneurysm sac pressure level is higher than the pressure level in the side branches.²¹ This means that the non-thrombosed side branches of the aneurysm will be efferent vessels immediately after EVAR. The level of aneurysm sac pressure will decrease to the pressure level in the patent side branches.^{18,31,56} This pressure reduction probably takes time, as can be derived from literature findings that the MPIs of aneurysms with a type II endoleak more than 1 month after EVAR^{13,14} are generally lower than MPIs measured at an earlier date (Fig. 3A). This is supported by data of the animal studies by Pitton *et al.*, in which is demonstrated that the MPI decreases during the period from 6 weeks to 6 months (Fig. 3B).^{15,16}

Considering these findings, the situation may be comparable immediately after EVAR without endoleak and with type II endoleak; a time interval in both situations is needed before the depressurization of the aneurysm sac takes place. The extent of

pressure reduction will be less in the presence of a type II endoleak, because in this situation the aneurysm sac pressure will not decrease beneath the pressure level in the side branch.^{18,31,56} Hence, the risk of type II endoleaks depends on the side branch pressure level. Furthermore, pressure level in side branches differs individually from patient to patient which results in different MPIs.

We accept that direct comparison of studies is hampered by different methods of reporting, different measurements of systemic pressure (peripheral or central), different time of measurement and different measuring techniques, but comparison of the material is needed to determine the value of aneurysm sac pressure monitoring during follow-up after EVAR. We took these factors into account to compare the results of different studies in the best possible way.

As mentioned, this review demonstrates that the MPI after EVAR differs between studies in which same type of endoleaks are investigated. Hence the risk of aneurysm rupture and therefore the need for re-intervention could differ per patient. This explains why identical types of endoleaks, as detected by imaging techniques, probably do not have identical clinical relevance. Therefore, continuous aneurysm sac pressure monitoring by wireless pressure sensors could be a valuable tool to estimate the risk of aneurysm rupture.⁵⁷ Unfortunately, aneurysm sac pressure monitoring is probably not that straightforward, because pressure measurements are often performed in a thrombosed aneurysm sac. Differences in pressure readings under identical pressure conditions could occur caused by the thrombus.^{58,59} Future research is needed to develop accurate methods of aneurysm sac pressure measurements and to evaluate the pitfalls of aneurysm sac pressure monitoring. Furthermore, patient studies with continuous aneurysm sac pressure monitoring are needed to determine the threshold of MPI when re-intervention is necessary to prevent aneurysm rupture.

In conclusion, MPIs differ widely in the presence as well as in absence of an endoleak. MPIs are not specific to the type of endoleak, because the determinants of aneurysm sac pressure are multi-factorial. This implies that the same type of endoleaks, detected by imaging, probably does not cause an identical risk of aneurysm rupture. Wireless aneurysm sac pressure monitoring could be valuable tool during follow-up after EVAR. However, further research is necessary to investigate the pitfalls of aneurysm sac pressure measurements before the clinical relevance of aneurysm sac pressure monitoring can be evaluated.

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