The goal of endovascular repair of a thoracic or abdominal aortic aneurysm (TEVAR – EVAR) is – as with open surgical repair – to prevent enlargement and rupture of the aneurysm (1). A common complication of TEVAR and EVAR is an endoleak and this is seen in up to 30% of cases (2). Endoleak is defined as the persistence of blood flow outside the lumen of the endoprosthesis, but within the aneurysm sac (3). A classification system for endoleaks has been developed, organizing endoleaks into 5 categories, depending on the blood flow into the aneurysm sac (4).

**Endoleak categories**

**Type I endoleak**

In type I endoleak the blood flow passes alongside the proximal (IA) or distal (IB) attachment sites of the endoprosthesis, as a result of poor or insufficient apposition between the attachment site and the arterial wall (4, 5) (Fig. 1A-C, Fig. 2A,B). An endoleak through a non-occluded iliac artery in a patient with an aorto-uni-iliac endoprosthesis and a femoral-femoral bypass is a type IC endoleak (2). In type I endoleaks there is a direct communication between the aneurysm sac and the systemic arterial circulation and therefore the aneurysm sac is at high risk for rupture (5). Type I endoleak is the most common type of endoleak in TEVAR (1).

**Type II endoleak**

Retrograde filling of the aneurysm sac through side branches, mainly from intercostal/lumbar arteries and/or inferior mesenteric artery causes a type II endoleak (1, 2). A distinction is made between a type II endoleak involving a single side branch (IIA) (Fig. 3A-C) and a type II endoleak with more complex blood flow through 2 or more arteries (IIB). Type II endoleak is the most common type of endoleak after EVAR (1). An association with aneurysmal expansion and rupture is possible, but the risk is lower in comparison to type I and type III endoleaks (0.5 vs 3.4%) (2).
risk for rupture because of the aneurysm sac is subject to the arterial pressure (2, 5).

**Type IV endoleak**

Type IV endoleaks are caused by porosity of the endoprosthesis fabric and identified during implantation of the device, when the patient is fully anticoagulated. These endoleaks are
In the EUROSTAR registry imaging surveillance at 1, 6, 12, 18 and 24 month after TEVAR - EVAR and then yearly is suggested (7, 8). Other centers perform surveillance at 1 month, 6 months and 12 months and then annually (1).

For endoleaks imaging surveillance is used for the detection and (if possible) the classification of endoleaks and for demonstration of any problems or deformities of the endoprosthesis.

Several imaging techniques have been used for surveillance, but (multi-detector) CT angiography remains the most widely used.

**MR angiography**

Gadolinium-enhanced MR angiography can be used to detect endoleaks, but only if the stents of the endoprosthesis are suitable for MR imaging. Stainless steel stents cause large susceptibility artifacts resulting in a nondiagnostic study. Eligiloy stents may obscure the vessel lumen. An endoprosthesis with nitinol stents will cause little or no artifacts and are therefore suitable for MR imaging (1). Time-resolved MR imaging can be used for characterization of the endoleak (1) and showed a 97% concordance with the findings of digital subtraction angiography (DSA) in one study (9).

**Ultrasound**

Ultrasound (US) has well known advantages over (MD)CTA (safe, inexpensive, no radiation, no use of iodine), but remains operator and patient dependent. Measurement of the aneurysm size with US correlates well with (MD)CT (1). Sensitivity for detecting an endoleak ranges between 67% and 86% in comparison to (MD)CT. Specificity varies between 67% and 100%. Positive predictive value (PPV) and negative predictive value (NPV) was between 29-100% and 90-100% respectively (10, 11). In 6 to 25% of cases US resulted in an inconclusive exam (10, 11). In the study of Uthoff and colleagues, the use of US as imaging surveillance technique increased during follow-up (26.6% at 6 months, 35.5% at 3 years), especially in experienced centers (12).

**Type V endoleak**

A continued expansion of the aneurysm sac after TEVAR – EVAR, without a radiological evidence of an endoleak is called type V endoleak or endotension (5, 6). The exact cause is unknown, but may be an undiagnosed or occult endoleak (because of very slow flow or suboptimal imaging) or ultrafiltration through the device fabric (4-6). Others suggested that in some patients the thrombus in the aneurysm sac provides an ineffective barrier to transmission of arterial pressure and that these significant forces continue to affect the sac (4).

**Surveillance**

Because of the potential complications after TEVAR – EVAR, such as an endoleak, a life-long imaging surveillance is necessary (1). The ideal frequency and imaging technique has however not yet been determined (1). In the EUROSTAR registry imaging surveillance at 1, 6, 12, 18 and 24 month after TEVAR - EVAR and then yearly is suggested (7, 8). Other centers perform surveillance at 1 month, 6 months and 12 months and then annually (1).
**MDCT angiography**

MDCTA remains the most commonly used imaging modality for surveillance of TEVAR – EVAR. Sensitivity in depicting endoleaks is higher in comparison to DSA (92% versus 63%). Multiphasic MDCTA is recommended to increase sensitivity since endoleaks have variable flow rates and therefore they can be detected at variable time points after injection of contrast material (1). Images without contrast are used to differentiate between contrast in the aneurysm sac and calcifications, while delayed venous phase images are used to depict endoleaks that are not visible during arterial phase (such as some slow flow type II endoleaks). Multiphasic MDCTA can therefore also help to classify an endoleak, although classification – together with radiation dose – remains a problem.

In order to reduce patient radiation dose from MDCTA exams, several possibilities has been suggested. One way to reduce radiation dose is to replace the triple-phase MDCTA by a dual-phase MDCTA, by removing either the pre-contrast phase (except for the first examination at 1 month), or the arterial or venous phase. Removing the arterial phase was suggested in case of a stable or regressing aneurysm. Moreover since sensitivity for depicting endoleaks is higher with a delayed venous phase than with an arterial phase in some studies, it has been suggested to eliminate the arterial phase (1, 13). Others found that the decrease of sensitivity to detect endoleaks in the arterial phase was not significant, while specificity and PPV improved with the arterial phase. Therefore they proposed to eliminate the venous phase (13, 14).

Dual-energy dual-source CT has been examined, comparing standard triple-phase examinations to either virtual non-enhanced + arterial + delayed phase or virtual non-enhanced images + delayed phase in 2 studies (15, 16). Examinations with virtual non-enhanced images + delayed phase resulted in a 61% reduction of radiation dose compared with triple-phase imaging and a 41% reduction in radiation compared with dual phase imaging with a sensitivity, specificity, NPV and PPV of 100%, 97%, 100%, and 96% respectively (15, 16).

To improve the classification of endoleaks with CT, Sommer and colleagues examined time-resolved CTA in 54 patients and compared the findings to contrast-enhanced US (17). They found a sensitivity, specificity, PPV and NPV of 94%, 93%, 89% and 96% respectively, with a mean effective radiation dose of 14.6 mSv (17).

**Digital subtraction angiography**

Although time-resolved imaging techniques may be promising, DSA remains the most specific modality for endoleak classification. Stavropoulos reported only an 86% agreement between CTA and DSA for the classification of endoleaks and DSA did change the choice of treatment in 11% of cases (18). In nearly all endoleaks contrast was seen in the inferior mesenteric artery and/or the lumbar arteries, but these findings reflect either inflow/outflow in a type II endoleak or – and probably more importantly – outflow from a type I or type III endoleak (1).

Moreover in 2 studies on percutaneous interventions for suspected type II endoleaks (based on CTA findings), respectively 21% and 36% of the presumed type II endoleaks appeared to be occult type I or type III endoleaks (19, 20).

**Endoleak treatment**

**Type I and type III endoleaks**

Because of the high risk for rupture in type I and type III endoleaks (arterial systemic pressure on the aneurysm sac), immediate treatment is needed. This can be done by endovascular means such as angioplasty balloons and bare stent to improve the apposition of the stent-graft to the vessel wall (in type I endoleaks) or additional stent-graft to extend the covering of the aneurysm (type I) (Fig. 5 A,B) or to cover the hole in the fabric/the zone of the junctional disconnection (type III) (Fig. 6). In selective cases with a type I endoleak, embolization has been described (1).

Surgical conversion is another option.

**Type II endoleak**

A lack of consensus exists concerning the treatment of type II endoleaks. Some groups believe that a type II endoleak that persists beyond 6 months should be treated (unless shrinkage of the aneurysm sac is documented) because the endoleak prevents thrombosis of the aneurysm sac and therefore a potential risk of aneurysm expansions and rupture exists (1). Others advocate a more conservative approach with close follow-up of the endoleak as long as the aneurysm does not show an increase in size. This is based upon the fact that 40% of the type II endoleaks will seal spontaneously. For this reason they believe that a
occult type I/III endoleak in about one third of patients. If this could be extrapolated to other studies, this may affect directly the outcome, because stabilization of the aneurysm is not to be expected (or at least to a lesser degree) in type I/III endoleaks. In general one could conclude from the available studies that a second percutaneous intervention is often needed and that technical success varies between 28% and 100%. There was also a wide range of clinical success between 44% and 100% if it was defined as stable or decreasing aneurysm diameter. No aneurysm rupture or aneurysm-related death was seen. Complication rate varied between 0% and 9.6%.

Type IV endoleak

As this is rare with the current devices and only seen during placement while the patient is fully anticoagulated, there is no specific treatment for this type of endoleak. Type IV endoleaks are self-limited and resolve spontaneously when the coagulation status of the patient is normalized (1). An endoleak demonstrated during follow-up is per definition not a type IV endoleak.
Type V endoleak

Treatment options are limited in type V endoleaks. It is important however to confirm the diagnosis of endotension (and to exclude an underlying endoleak as cause of the aneurysm expansion). Endotension can be treated by relining (placing a new endoprosthesis within the old}

Fig. 8. — A. Contrast-enhanced CT-scan showing an endoleak anterior (arrow) and posterior (arrowhead) to the two legs of the endoprosthesis. (B,C). DSA shows that these are two independent type II endoleaks. The anteriorly located endoleak (asterisk) is opacified through retrograde flow in the inferior mesenteric artery (arrow) via the superior mesenteric artery. The inflow/outflow arteries of the posterior endoleak (white asterisk) are a common origin of the fourth lumbar artery on both sides and middle sacral artery and lumbar artery L3 at the left side (arrows). D. Percutaneous direct puncture of the posterior endoleak was done guided by a pre-planned trajectory on a cone-beam CT-scan performed with a flat-panel detector system. E. DSA through the percutaneous needle showing the endoleak (white asterisk) and the inflow/outflow vessels (arrows). F. DA after embolization of lumbar artery L4 on the left side with microcoils (arrows) and after embolization of the endoleak with glue (white asterisk). There is some spilling of glue in left lumbar artery L2 (arrowhead) and in the psoas muscle (white circle).

G. DA showing superselective transarterial catheterization of the anterior endoleak (white asterisk) with a microcatheter. The outflow vessel of this endoleak is a right inferior polar artery (arrowheads). H. DSA after embolization of the endoleak (arrows) and the proximal segment of the inferior mesenteric artery (arrowhead) with microcoils. No more filling of the anterior endoleak through the inferior mesenteric artery is seen.
one) or by conversion to surgery. The risk of rupture in patients treated conservatively is however very low (< 1%) in the first 4 years if patients were not treated with Vanguard stent-grafts and had aneurysm diameters < 70 mm (8).

Conclusions

Endoleak is a common complication of TEVAR and EVAR and therefore lifelong imaging surveillance is important. The ideal frequency and imaging technique have not yet been determined and the pros and cons of the different imaging modalities should be kept in mind. For the moment MDCTA is the most widely used technique for detection of endoleaks and classification is done by DSA. Type I and III endoleaks require immediate treatment because these endoleaks are prone to rupture. Available evidence support the conservative management of type II endoleaks, with treatment restricted to type II endoleaks with enlargement of the aneurysm sac >5 mm over a 6-month period or >10 mm in comparison with the diameter before TEVAR – EVAR. Before treating type II endoleaks one must exclude underlying occult type I/III endoleaks and the goal of the intervention should be to emolize the inflow and outflow and the communicating channels in between.

Treatment of type V endoleaks remains unclear and may be conservative, endovascular or surgical.

References