CLINICAL PRACTICE GUIDELINE DOCUMENT

Editor's Choice — European Society for Vascular Surgery (ESVS) 2020 Clinical Practice Guidelines on the Management of Vascular Graft and Endograft Infections☆

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DEDICATION

After studying medicine at Hanover Medical School and philosophy and social psychology at the Leibniz University Hanover, Omke E. Teebken joined the Christian Albrechts University in Kiel at the end of the 1990s as a research fellow at the Clinic for Cardiovascular Surgery headed by Professor Dr Axel Haverich, whom Omke E. Teebken later followed back to Hanover.

In Hanover, besides working as a clinician, Omke E.Teebken was particularly active scientifically, contributing to the establishment of the then newly founded Leibniz Laboratories for Biotechnology and Artificial Organs (LEBAO). His work focused on regenerative medicine and tissue engineering, and subsequently he wrote his habilitation thesis in in this field. After basic training in cardiac surgery, he specialised clinically in vascular surgery and played a pioneering role in the development of this field. Before being appointed director of the Clinic for Vascular Surgery — Endovascular Surgery at the Peine Clinic in 2016, Omke E. Teebken headed the Vascular Surgery — Endovascular Surgery Division of the Department of Cardiothoracic, Thoracic, Transplantation and Vascular Surgery at Hanover Medical School. Professor Teebken was a highly appreciated, committed, and competent colleague and teacher.

On 8 April 2019, Professor Teebken passed away after a short and severe illness. He was member and author of the ESVS guideline writing committee, an esteemed colleague, and friend.

We will always honor his memory.

Prof. Dr. med. Omke Enno Teebken
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TABLE OF CONTENTS

LIST OF ABBREVIATIONS

ABF	Aortobronchial fistula
AEsF	Aorto-oesophageal fistula
AEnF	Aorto-enteric fistula
APF	Aortopulmonary fistula
AUF	Arterio-ureteral fistula
CDC	Centers for Disease Control and Prevention
CI	Confidence interval
CoNS	Coagulase negative staphylococci
CRP	C reactive protein
CT	Computed tomography
CTA	Computed tomography angiography
EANM	European Association of Nuclear Medicine
EAR	Extra-anatomic reconstruction
EG	Endograft
EGI	Endograft infection
ePTFE	Expanded polytetrafluoroethylene
ESC	European Society of Cardiology
ESVS	European Society for Vascular Surgery
EVAR	Endovascular aneurysm repair
EuREC	European Registry of Endovascular Aortic Complications
^{18}F-FDG-PET/CT	^{18}F-fluoro-D-deoxyglucose positron emission tomography/computed tomography
GC	Guidelines Committee
GMF	Gracilis muscle flap
GSV	Great saphenous vein
ISR	*In situ* reconstruction
LACA	Lateral approach to crural arteries
LCCA	Left common carotid artery
LSCA	Left subclavian artery
LRSB	Lateral retrosartorius bypass
MAGIC	Management of Aortic Graft Infection
MDR	Multidrug resistant
MRA	Magnetic resonance angiography
MRSA	Methicillin resistant *Staphylococcus aureus*
NPWT	Negative pressure wound therapy
OR	Odds ratio
OB	Obturator bypass
PCR	Polymerase chain reaction
PET	Polyethylene terephthalate
PGA	Perigeniculate artery
PTFE	Polytetrafluoroethylene
RAF	Rectus abdominis flap
RCT	Randomised controlled trial
RFF	Rectus femoris flap
RR	Relative risk
SAT	Supra-aortic trunk
SFA	Superficial femoral artery
SMF	Sartorius muscle flap

SPECT/CT	Single photon emission computed tomography
SSI	Surgical site infection
SUV$_{max}$	Maximum standardised uptake value
TEVAR	Thoracic endovascular aneurysm repair
US	Ultrasound

VAC	Vacuum assisted closure
VG	Vascular graft
VGI	Vascular graft infection
VGEI	Vascular graft or endograft infection
WBCS	White blood cell scintigraphy
WC	Writing Committee

1. INTRODUCTION AND GENERAL ASPECTS

1.1. Purpose of the guidelines

Guidelines driven by scientific societies on vascular graft/endograft infection (VGEI) have not been published. The European Society for Vascular Surgery (ESVS) has developed clinical practice guidelines for the care of patients with VGEI. The aim of this document is to assist physicians involved in the diagnosis and treatment of patients with VGEI in selecting the best management strategy in different scenarios. The potential users of this guideline include angiologists, vascular, cardiovascular and general surgeons, infectious disease physicians, and radiologists, and the target population comprises patients with VGEI in the supra-aortic trunks, thoracic and/or abdominal aorta, and peripheral arteries.

Guidelines have the purpose of promoting a standard of care according to specialists in the field, in this case represented by members of the ESVS. However, under no circumstances should these guidelines be seen as the legal standard of care in all patients. As the word guidelines states in itself, the document is a guiding principle, but the care given to a single patient is always dependent on the individual (symptom variability, comorbidities, age, etc.) and treatment setting (techniques available, local expertise).

1.2. Methods

1.2.1. The writing committee.
The members of this guidelines Writing Committee (WC) were selected by the ESVS, the European Association of Nuclear Medicine (EANM), and the Groupe de Recherche sur les Infections de Prothèses, to represent physicians involved in the management of patients with VGEI. They include vascular surgeons, radiologists, and infectious disease specialists. WC members have provided disclosure statements of all relationships that might be perceived as real or potential sources of conflicts of interest, which are kept on file at the ESVS headquarters. No ESVS reviewers or individual WC members received any financial support from third parties in direct or indirect relation to this guideline, and all WC members and reviewers signed declarations of interest.

1.2.2. Evidence collection
1.2.2.1. Search strategy.
The purpose, list of topics, and tasks and methods regarding the construction of the guidelines were agreed and distributed among the WC members in an initial meeting held in Strasbourg on 30 June 2017.
1.2.2.2. Literature search and selection.
All WC members performed a systematic literature search strategy for each of their assigned sections, carried out in PubMed, Scopus, Cardiosource Clinical Trials Database, and the Cochrane Library databases, first from January 1997 to November 2017, with a later update to February 2019 for relevant papers published in English. Reference checking and a hand search added other relevant literature. Abstracts were excluded. Single case reports or case series were included if they were of paramount importance to these guidelines to enlighten the manuscript.

Selection of the literature was performed based on information provided in the title and abstract of the retrieved studies. Only peer reviewed published literature and studies presenting pre-defined outcomes were considered. The selection process followed the pyramid of evidence, with aggregated evidence at the top of the pyramid (systematic reviews, meta-analysis), followed by randomised controlled trials (RCTs), then observational studies, leaving expert opinion at the bottom. The level of evidence per section in the guidelines is dependent on the level of evidence available on the specific subject.

1.2.2.3. Evidence and recommendation grading criteria.
To define the current guidelines, members of the WC reviewed and summarised the selected literature. Conclusions were drawn based on the availability and quality of the scientific evidence, and recommendations for the evaluation and treatment of patients with VGEI were formulated based on the analysis of the evidence and through consensus when evidence was scarce.

The European Society of Cardiology (ESC) grading system was used for evidence and recommendation rating. The letter A, B, or C reflects the level of current evidence (Table 1), and weighing the level of evidence and expert opinion, each recommendation is graded as class I, IIa, IIb, or III (Table 2). For those recommendations tables of evidence were built and are available as supplementary material.

1.2.2.4. The patient's perspective.
The goals behind patient participation in healthcare decision making can be categorised as democratisation and increased quality of decisions. Patient engagement improves the validity of clinical guidelines and is encouraged by international and national groups. In order to better understand patient feedback,

Table 1. Level of evidence	
Level of evidence A	Data derived from multiple randomized clinical trials or meta analyses.
Level of evidence B	Data derived from a single randomized clinical trials or large non-randomized studies.
Level of evidence C	Consensus of opinion of the experts and/or small studies, retrospective studies, registries.

Table 2. Classes of recommendations

Classes of recommendations	Definition
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, effective.
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure.
Class IIa	Weight of evidence/opinion is in favour of usefulness/efficacy.
Class IIb	Usefulness/efficacy is less well established by evidence/opinion.
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.

European patients were interviewed: representatives of patient associations in the field of aortic dissection and infectious diseases; and patients treated for abdominal VGEI (patients operated on by surgeons of the WC). The main questions that arose from discussions were: (1) Did you feel your physician provided enough information about the risk of infection at the time of the initial procedure? (2) What did you think about the management once the diagnosis of VGEI was made? and (3) Did you think that your physician provided enough information on the risks related to VGEI? Patients were interviewed with a focus on these three open questions.

1.2.3. The revision process. The guidelines document, merged and harmonised by the co-chairmen of the WC, underwent internal review. Once approved by every WC member, it moved on to external revision by the ESVS Guidelines Committee (GC) members and chosen external experts in the field. Each draft was revised by the WC and the final document, approved by all WC and GC members and external reviewers, was submitted to *the European Journal of Vascular and Endovascular Surgery* on 20 July 2019.

1.2.4. The update plan. As technology and disease knowledge in this field changes rapidly, current recommendations can become outdated. It is an aim of the ESVS to revise the guidelines when important new insights in the evaluation and management of VGEI become available or every five years at the latest.

2. GENERAL CONSIDERATIONS

2.1. Definition of incisional surgical site infection

Studies dealing with VGEI are mostly case series rather than randomised studies. Diagnosis of VGEI is usually related to clinical findings, imaging studies, and microbiological examinations.[1] Criteria for incisional surgical site infections (SSI), which can be both superficial and deep, have been described by the Centers for Disease Control and Prevention (CDC) and can be applied to the description of VGEI (Table 3).[2]

2.2. Classifications

While the CDC definitions[2] differentiate between superficial and deep incisional SSIs without placing emphasis on vascular grafts (VGs), the Szilagyi classification and the Samson classification specifically also consider VG involvement, while the extent of graft involvement can be described using the Bunt classification (Table 4).[3–5] Furthermore, aortic VGEI can also be divided into early (< 4 months) or late (> 4 months) onset, which, in many cases, is also extrapolated to other VGEI.[6] However, the clinical relevance of differentiation between early and late infections remains a matter of debate.

2.3. Definition of vascular graft/endograft infection

To overcome the numerous shortcomings of current classifications, the Management of Aortic Graft Infection (MAGIC) group has developed a list of major and minor criteria with respect to clinical, surgical, radiological, and laboratory findings (Table 5).[1] Once VGEI is suspected, an exhaustive evaluation of the clinical status, signs of infection, and comorbidities of the patient according to the MAGIC criteria is recommended.

According to the MAGIC criteria, VGEI is suspected in the presence of one major or two minor criteria of the three different categories, and VGEI is diagnosed when there is at least a single major criterion and any other criterion from another category. For example, a fever $\geq 38°C$ is considered non-specific for VGEI and therefore it is required that no other clinical cause is apparent. Sepsis and systemic inflammatory response syndrome may be caused by something other than VGEI and is defined as combinations of different findings. Anorexia, lethargy, and malaise may accompany aortic graft and endograft (EG) infection, but are also considered insufficiently specific.[1] Intra-operative fluids around a graft can represent pus, but despite a yellowish or cloudy appearance may be present for non-infective reasons and microbiological culture will be negative. Therefore, pus cells must be proven by direct microscopy to be considered a major criterion. Furthermore, a direct communication between non-sterile sites and a prosthesis indicates graft infection: aorto-enteric fistula (AEnF), aorto-bronchial fistula (ABF), deployment of a stent graft in an already infected field (e.g., infected aneurysm), and exposed grafts in deep open wounds.

2.4. Epidemiology

2.4.1. Incidence. VGEI are usually multifactorial and result from the complex involvement of patient, surgical, and environmental factors, making the real incidence difficult to assess. Reported incidences of VGEI by type and anatomical location will be developed in specific sections.

2.4.2. Risk factors. Multiple risk factors contribute to VGEI and are listed in Table 6.

2.5. Pathogenesis

The pathogenesis of VGEI is multifactorial. Presumably, early VGEI are mostly caused by a breach in sterility during

Table 3. Centers for Disease Control and Prevention criteria for superficial and deep surgical site infections (SSI)[2]

Criteria	Superficial SSI	Deep SSI
*Diagnostic criteria**		
1	Infection occurs within 30 days after the operative procedure	Infection occurs within 30 days after the operative procedure if no implant is left in place, or within one year if implant is in place and the infection appears to be related to the operative procedure
and 2	Infection involves only skin and subcutaneous tissue of the incision	Infection involves deep soft tissues (e.g., fascia and muscle layers) of the incision
and 3	Patient has at least one of the following:	Patient has at least one of the following:
	• Purulent drainage from the superficial incision	• Purulent drainage from the deep incision but not from the organ/space component of the surgical site
	• Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision	• A deep incision spontaneously dehisces or is deliberately opened by a surgeon and is culture positive or not cultured when the patient has at least one of the following signs or symptoms: fever (>38°C), or localised pain or tenderness. A culture negative finding does not meet this criterion
	• At least one of the following signs or symptoms of infection: pain or tenderness, localised swelling, redness or heat, and superficial incision is deliberately opened by surgeon and is culture positive or not cultured. A culture negative finding does not meet this criterion	• An abscess or other evidence of infection involving the deep incision is found on direct examination, during re-operation, or by histopathological or radiological examination
	• Diagnosis of superficial incisional SSI by a surgeon or attending physician	• Diagnosis of a deep incisional SSI by a surgeon or attending physician
Types		
Incisional primary	A superficial incisional SSI that is identified in the primary incision in a patient who has had an operation with one or more incisions	A deep incisional SSI that is identified in a primary incision in a patient who has had an operation with one or more incisions
Incisional secondary	A superficial incisional SSI that is identified in the secondary incision in a patient who has had an operation with >1 incision (e.g., donor site [leg] incision to harvest autologous veins for *in situ* reconstruction of an abdominal vascular graft infection)	A deep incisional SSI that is identified in the secondary incision in a patient who has had an operation with >1 incision (e.g., donor site [leg] incision to harvest autologous veins for *in situ* reconstruction of an abdominal vascular graft infection)
Reporting instructions		
	Do not report a skin suture abscess with minimal inflammation and discharge confined to the points of suture penetration, as an infection	Classify infection that involves both superficial and deep incision sites as deep incisional SSI
	Do not report a localised stab wound infection as SSI; instead, report as skin or soft tissue infection, depending on its depth	
	If the incisional site infection involves or extends into the fascial and muscle layers, report as a deep incisional SSI	

* For diagnosis of SSI, diagnostic criteria 1, 2, and 3 must all be true.

implantation or the presence of bacteria in the aneurysmal thrombus, while late VGEI are mostly caused by haematogenous seeding from a bacteraemia (mostly arising from the urinary or respiratory tract), or from bacterial translocation or iatrogenic contamination during catheterisation.[6–8] The pathogenesis of AEnF, aorto-oesophageal (AEsF), and ABF remains unclear. Ischaemia of the visceral wall due to occlusion of the feeding arteries, and mechanical erosion by the aneurysm or of a suture line pseudo-aneurysm, especially when still under pressure due to presence of an endoleak, have all been suggested. Fistula can occur as a result of direct trauma related to surgical injury, poor tunnelling, erosion by direct contact, or by the penetration of an oversised EG. Previous adjacent or remote infection in any site is considered to be a causative or contributing factor.[9,10]

The quality of material incorporation related to tissue ingrowth and healing also plays a role, explaining that VEGI might even be more frequent than VG infection (VGI), as there is no tissue ingrowth in the wall of the EG fabric that is surrounded only by thrombotic material, contrary to VG.[11]

2.6. Clinical presentation

As mentioned in the MAGIC criteria, the clinical presentation of patients with VGEI varies between mild symptoms (redness of the skin, non-purulent effusion from a wound) to severe and evident symtoms such as sepsis or anastomotic rupture with hypovolaemic shock.[1] Fever of unclear origin and an unexplained leukocytosis with concomitant increase of C reactive protein (CRP) and fever may be the only clinical or laboratory sign of VGEI. In other cases the clinical manifestations may include abscess, mass, septic embolisation, septic shock, bleeding, melaena, haematemesis, haematuria, ileus, or abdominal distension. When VGEI is suspected, a complete clinical and biochemical evaluation

Table 4. Classifications for wound and vascular graft infections with respect to wound infection (Szilagyi, Samson) and to the extent of graft involvement (Bunt)[3–5]

Szilagyi classification:
Grade I: cellulitis involving the wound
Grade II: infection involving subcutaneous tissue
Grade III: infection involving the vascular prosthesis
Samson classification:
Group 1: no deeper than dermis
Group 2: subcutaneous tissue, no direct contact with the graft
Group 3: body of graft but not anastomosis
Group 4: exposed anastomosis, no bleeding, no bacteraemia
Group 5: anastomosis involved, bleeding, bacteraemia
Extent of graft involvement (Bunt classification modified)
Peripheral graft infection:
P0 graft infection: infection of a cavitary graft (e.g., aortic arch; abdominal and thoracic aortic interposition; aorto-iliac, aortofemoral, iliofemoral graft infections)
P1 graft infection: infection of a graft whose entire anatomical course is non-cavitary (e.g., carotid–subclavian, axillo-axillary, axillofemoral, femorofemoral, femorodistal, dialysis access bridge graft infections)
P2 graft infection: infection of the extracavitary portion of a graft whose origin is cavitary (e.g., infected groin segment of an aortofemoral or thoracofemoral graft, cervical infection of an aortocarotid graft)
P3 graft infection: infection involving a prosthetic patch angioplasty (e.g., carotid and femoral endarterectomies with prosthetic patch closure)
Graft-enteric erosion
Graft-enteric fistula
Aortic stump sepsis after excision of an infected aortic graft

of the patient is required in order to provide a sufficient analytical overview.[12]

Post-implantation syndrome, characterised by transitory fever associated with elevated leukocytes and CRP may be observed following endograft implantation, but might also be distinguishing from an actual infection.[13]

2.7. Microbiology and sampling techniques

2.7.1. Microbiology. Micro-organism identification is a key issue in order to provide the patient with the best treatment. Using the different available sampling techniques, micro-organisms can be isolated in about 75% — 98% of cases.[14–16] Responsible pathogens are Gram positive

Table 5. The MAGIC classification[1]

Criterion	Clinical/surgical	Radiology	Laboratory
Major			
	Pus (confirmed by microscopy) around graft or in aneurysm sac at surgery	Perigraft fluid on CT scan ≥ 3 months after insertion	Organisms recovered from an explanted graft
	Open wound with exposed graft or communicating sinus	Perigraft gas on CT scan ≥ 7 weeks after insertion	Organisms recovered from an intra-operative specimen
	Fistula development, e.g., aorto-enteric or aortobronchial	Increase in perigraft gas volume demonstrated on serial imaging	Organisms recovered from a percutaneous, radiologically guided aspirate of perigraft fluid
	Graft insertion in an infected site, e.g., fistula, mycotic aneurysm, or infected pseudo-aneurysm		
Minor			
	Localised clinical features of graft infection, e.g., erythema, warmth, swelling, purulent discharge, pain	Other, e.g., suspicious perigraft gas/fluid soft tissue inflammation; aneurysm expansion; pseudo-aneurysm formation: focal bowel wall thickening; discitis/osteomyelitis; suspicious metabolic activity on FDG-PET/CT; radiolabelled leukocyte uptake	Blood culture(s) positive and no apparent source except graft infection
	Fever ≥38°C with graft infection as most likely cause		Abnormally elevated inflammatory markers with graft infection as most likely cause, e.g., erythrocyte sedimentation rate, C reactive protein, white cell count

CT = computed tomography; FDG-PET/CT = 18F-fluoro-D-deoxyglucose positron emission tomography/computed tomography

Table 6. Risk factors for vascular graft/endograft infection[6,7]

Pre-operative risk factors
Prolonged pre-operative hospitalisation
Infection in a remote or adjacent site
Recent percutaneous arterial access at the implant site
Emergency/urgent procedure
Re-intervention
Lower limb infection (ulcer, gangrene, cellulitis)
Groin incision
Intra-operative risk factors
Breach in aseptic technique
Prolonged operation time
Concomitant gastrointestinal or genitourinary procedure
Post-operative risk factors
Post-operative wound complications (infection, skin necrosis, lymphocoele, seroma, haematoma)
Graft thrombosis
Patient related risk factors/altered host defences
Malignancy
Lymphoproliferative disorder
Immune disorders
Corticosteroid administration
Chemotherapy
Malnutrition
Diabetes mellitus/peri-operative hyperglycaemia
Chronic renal insufficiency/end stage renal disease
Liver disease/cirrhosis
Immunosuppression by non-suspended anti-tumour necrosis factor alpha

bacteria in up to 58% of VGEI (including enterococci, *Staphylococcus aureus*, and coagulase negative staphylococci); Gram negative bacteria account for about 34% of VGEIs and and anaerobes 8%.[14–17]

In a recent meta-analysis, the risk of re-infection has been studied according to different infecting micro-organisms.[17] *Staphylococcus aureus*, Enterobacteriaceae, *Pseudomonas aeruginosa*, and beta haemolytic streptococci were classified as virulent, while bacteria belonging to the skin colonising flora such as *Staphylococcus epidermidis*, corynebacterial, and *Cutibacterium acnes* were classified as non-virulent agents. The results of this meta-analysis established that virulent organisms were significantly associated with an increased risk of re-infection.

Antimicrobial resistance of the causative bacteria is another factor that may reduce the chance of healing, but this relationship has not been clearly established in the setting of VGEIs.[14]

The susceptibility of bacteria to the few antibiotics that exhibit a sustained activity in the environment of a biofilm (e.g., rifampicin combinations for staphylococcal implant infections) is another element that may lead to re-infection in patients treated for VGEIs.[18–20]

2.7.2. Sampling techniques. Microbiological samples may support establishing the diagnosis of a VGEI. Ideally samples should be harvested before the start of antimicrobial therapy. However the accuracy and relevance of microbiological tests depend on whether specimens were collected without contamination, and in an adequate quantity.[21] Moreover, samples should be forwarded quickly to the microbiology laboratory. If they cannot be forwarded immediately they should be stored at $+4^\circ$C.

2.7.2.1. Directly obtained specimens. Meaningful results will be achieved with specimens obtained directly from the suspected infection site. These may include surgically explanted prosthetic materials, intra-operatively obtained tissue and graft biopsies from the infected area, or at least three samples from perigraft fluid collection.[22] Paediatric anaerobic tubes, which require very small amounts of material, can be used.

Aspirated specimens obtained under ultrasound (US) or computed tomography (CT) guidance provide material for an accurate microbiological diagnosis. The presence of graft incorporation into tissue reliably excluded the presence of bacteria in cultures in 97% of investigated grafts, whereas the finding of graft disincorporation accurately predicted a positive culture in 89% of all positive VGEI cases.[23] In general, tissue specimens or a portion of the graft material are superior to swab specimens of infected sites, even when collected using a sterile technique intra-operatively. At least three direct specimens should be collected in sterile containers.[22] Swabs should be avoided because they do not allow differentiation of colonising micro-organisms from true pathogens and may lead to overprescription of broad spectrum antibiotics. Swabs have an inherent difficulty transferring bacteria or fungi from the swab fibres onto culture media, and because the inoculum from the swab often is not uniformly distributed across several different agar plates.[24] If swabs are used, the type of swab should be selected on basis of its ability to collect micro-organisms. Polyethylene terephthalate (PET) swabs should be used.

A new innovative specimen collection technology (microDTTect) could help in the future, as it allows for contamination free sampling, and also it can dislodge bacteria embedded in a biofilm from prosthetic surfaces.[25]

2.7.2.2. Indirectly obtained specimens. Indirect specimens might also be meaningful, especially when direct specimens are not collected in cases when redo surgery is not performed. Such specments include blood cultures, specimens obtained from a superficial wound, a draining sinus, or otherwise close anatomical structures.

Despite being an indirect microbiological sampling method, blood cultures may yield supportive information, as pre-operative blood cultures have been found to be positive in about 35% of cases and both pre- and peri-operative samples are positive for the same micro-organism in about 22% – 30% of cases.[14,15]

However, other indirectly obtained specimens using swabs, biopsy samples or aspirates obtained from a superficial wound, a draining sinus, or otherwise close anatomical structures always contain skin flora or colonisation, and might not accurately reflect the causative micro-organism of a VGEI.[26] Bacteriological

investigation of negative pressure wound therapy (NPWT) foams should not be performed to support the diagnosis of a VGEI because of the low sensitivity and specificity.[27]

Therefore, results from indirectly obtained specimens should be considered with caution.

2.7.3. Microbiological sample processing.
Specimens may be investigated using different techniques such as direct streaking specimens on agar plates, placing specimens into broth culture, homogenisation of tissue or graft specimens with serial dilution techniques, sonication of a harvested graft, or vortex mixing tissue samples in order to enhance the recovery of biofilm forming micro-organisms.[28]

Enhanced sample processing techniques such as vortex mixing specimens or sonication improve the detection rate of microorganisms attached to graft material.[28–30] One study found that ultrasonic bath treatment released consistently more bacteria than direct ultrasonic disruption or vortex agitation.[31] Importantly, the high energy levels of direct ultrasonic disruption can decrease the number of viable Gram negative bacteria, and vortex agitation consistently produced the lowest bacterial numbers among the three methods tested. An ultrasonic bath treatment of one to five minutes duration of infected VG at a frequency of 25 – 40 KHz may be the optimal preparation method for causative bacteria detection.[31] Adding broad range polymerase chain reaction (PCR) detection to sonicated fluid cultures may even increase the detection rate of bacteria attached to graft material.[32]

2.8. Imaging modalities

2.8.1. Introduction.
Various imaging techniques are used in the diagnostic work up when VGEI is suspected. Conventional imaging techniques such as US, CT, CT with angiography (CTA), and magnetic resonance with angiography (MRA) are used most frequently. Other available imaging tools are nuclear medicine techniques, such as 18F-fluoro-D-deoxyglucose positron emission tomography (18F-FDG-PET) with or without diagnostic contrast enhanced CT (18F-FDG-PET/CT), and white blood cell scintigraphy (WBCS), that can be combined with single photon emission computed tomography (SPECT/CT) for better localisation of the infection.[33]

2.8.2. Conventional techniques

2.8.2.1. Ultrasound.
US is the most common, non-invasive, low cost imaging modality to identify findings associated with VGEI.

US characteristics of VGEI are the presence of pseudo-aneurysm, sustained presence of gas (if still present after > 7 weeks), and purely anechoic fluid collections (if still present >3 months after surgery).[33,34] It can differentiate between haematoma or abscess formation, which makes it a good primary imaging screening modality, especially for superficial peripheral VG. However, the absence of peri-prosthetic collections on US does not allow ruling out of a VGEI. US also allows investigating for graft thrombosis, which can be the first sign of VGEI, and it can guide puncture for bacteriological purposes.[14,33,35]

However, US has a high interoperator variability and the predictive value is limited in the case of a centrally located graft due to overlying bowel gas or obesity.[14,34] Therefore, the sensitivity of US for the diagnosis of VGEI is considered as low, and additional investigations are often needed to obtain more detailed information on VG status.

2.8.2.2. Computed tomography angiography.
CTA has been considered the reference imaging standard in diagnosing VGEI for a long time, as it is able to visualise the characteristic features of VGEI.[33] The use of intravenous contrast, with images acquired in the arterial phase, may show certain signs such as ectopic gas, fluid, soft tissue enhancement, pseudo-aneurysm, focal bowel thickening, and discontinuation of the aneurysmal wall, all of which can all be used as criteria to increase the likelihood of a VGEI.[36] Furthermore, in the arterial phase it may be possible to detect contrast passage from the aorta to the digestive tract in cases of AEnF.[33]

Although better than US, CTA sensitivity and specificity remains moderate and variable.[34] In a systematic review and meta-analysis of patients with suspected VGEI, the pooled sensitivity of CTA in diagnosing VGEI was 0.67 (95% confidence interval [CI] 0.57 – 0.75) and the pooled specificity was 0.63 (95% CI 0.48 – 0.76). This meta-analysis showed that an isolated CTA does not provide enough evidence to establish the diagnosis of VGEI (Fig. 1).[37]

Standalone CTA can confirm the diagnosis of VGEI, but a second imaging modality such as 18F-FDG-PET/CT or WBCS combined with SPECT/CT may be useful to map the extent of the infection.

2.8.2.3. Magnetic resonance angiography.
MRA has not been evaluated as extensively as CTA for the diagnosis of VGEI, but several studies have suggested that MRA offers better anatomical and functional information than CTA, including tissue characterisation.[14,35] Simultaneous or sequential acquisition of 18F-FDG-PET with MRA provides additional quantitative molecular functional information concerning the inflammatory lesion, and accurate localisation, as well as anatomical changes with motion correction. After six post-operative weeks, the presence of collections with a hypo-intense signal in T1 and a hyperintense signal in T2 strongly suggests a VGEI.[14,35]

In a series of patients with suspected aortic VGEI, the sensitivity of MRA was 0.68 (95% CI 0.50 – 0.86), and the specificity 0.97 (95% CI 0.91 – 1.00).[38] However, owing to low availability and long acquisition times resulting in motion artefacts, MRA is currently not used as a first line diagnostic modality if VGEI is suspected.

2.8.3. Nuclear imaging techniques.
Nuclear medicine imaging techniques, such as 18F-FDG-PET combined with (low

A

CTA

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Fukuchi (2005)[281]	7	3	4	19	0.64 [0.31, 0.89]	0.86 [0.65, 0.97]
Bruggink (2010)[34]	9	5	6	5	0.60 [0.32, 0.84]	0.50 [0.19, 0.81]
Khaja (2013)[282]	14	2	2	2	0.88 [0.62, 0.98]	0.50 [0.07, 0.93]
Erba (2014)[283]	23	3	24	5	0.49 [0.34, 0.64]	0.63 [0.24, 0.91]
Saleem (2015)[214]	21	16	0	0	1.00 [0.84, 1.00]	0.00 [0.00, 0.21]

B

FDG-PET

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Fukuchi (2005)[281]	10	8	1	14	0.91 [0.59, 1.00]	0.64 [0.41, 0.83]
Spacek (2009)[284]	54	10	1	31	0.98 [0.90, 1.00]	0.76 [0.60, 0.88]
Bruggink (2010)[34]	14	3	1	7	0.93 [0.68, 1.00]	0.70 [0.35, 0.93]
Saleem (2015)[214]	18	6	3	10	0.86 [0.64, 0.97]	0.63 [0.35, 0.85]

C

FDG-PET/CT

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Keidar (2007)[39]	14	2	1	22	0.93 [0.68, 1.00]	0.92 [0.73, 0.99]
Bruggink (2010)[34]	12	4	3	6	0.80 [0.52, 0.96]	0.60 [0.26, 0.88]
Karaca (2014)[285]	12	2	0	3	1.00 [0.74, 1.00]	0.60 [0.15, 0.95]
Chang (2015)[86]	5	5	0	19	1.00 [0.48, 1.00]	0.79 [0.58, 0.93]
Sah (2015)[43]	27	1	0	6	1.00 [0.87, 1.00]	0.86 [0.42, 1.00]

D

WBC scintigraphy

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Liberatore (1998)[286]	115	3	0	44	1.00 [0.97, 1.00]	0.94 [0.82, 0.99]
Tronco (2008)	5	1	0	13	1.00 [0.48, 1.00]	0.93 [0.66, 1.00]
Khaja (2013)[282]	12	0	4	4	0.75 [0.48, 0.93]	1.00 [0.40, 1.00]
Shahidi (2013)[38]	16	4	6	27	0.73 [0.50, 0.89]	0.87 [0.70, 0.96]
Erba (2014)[283]	36	4	11	4	0.77 [0.62, 0.88]	0.50 [0.16, 0.84]

E

WBC SPECT/CT

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Lou (2010)[287]	6	1	0	4	1.00 [0.54, 1.00]	0.80 [0.28, 0.99]
Khaja (2013)[282]	15	2	1	2	0.94 [0.70, 1.00]	0.50 [0.07, 0.93]
Erba (2014)[283]	47	0	0	8	1.00 [0.92, 1.00]	1.00 [0.63, 1.00]

Figure 1. Sensitivities and specificities for each imaging modality.[37] CTA = computed tomography angiography; TP = true positive; FP = false positive; FN = false negative; TN = true negative; CI = confidence interval; FDG-PET = ^{18}F-fluoro-D-deoxyglucose positron emission tomography/computed tomography; FDG-PET/CT = ^{18}F-fluoro-D-deoxyglucose positron emission tomography/computed tomography; WBC = white blood cell; SPECT/CT = single photon emission computed tomography/computed tomography.

dose or contrast enhanced) CT and WBCS combined with SPECT/CT, incorporate anatomical and metabolic information at the same time and are able to differentiate between VGEI, soft tissue infection, and, in some cases, inflammation by pattern recognition, heterogeneity, and intensity of uptake with FDG-PET,[39] and by increase in size or intensity with time with WBCS.[40]

2.8.3.1. Positron emission tomography. 18F-FDG-PET imaging is based on the uptake of radioactive labelled glucose in cells/tissue with enhanced glucose metabolism, such as inflammatory cells and micro-organisms such as bacteria or fungi. This diagnostic method may differentiate between periprosthetic collection and involvement of the graft material but should be combined with low dose CT for anatomical correlation. Nowadays, 18F-FDG-PET is mainly performed in hybrid mode with FDG-PET/CT, which has an established role in the assessment of suspected VGEI, providing accurate anatomical localisation of the site of infection.

The EANM and Society of Nuclear Medicine and Molecular Imaging published procedural guidelines on how to perform a 18F-FDG-PET scan for infectious purposes.[41] As the administered dose of 18F-FDG and time interval between the scan acquisition may cause heterogeneity between studies, the EANM launched a strategy to harmonise 18F-FDG-PET/CT studies (EANM Research Limited, EARL).[42] There are different ways to analyse and interpret 18F-FDG-PET/CT studies. The main interpretation criteria are the calculated maximum standardised uptake value (SUVmax), the tissue to background ratio, the pattern of uptake (focal/diffuse), or the visual grading scale.[41,43] It is suggested that SUVmax > 8 in the perigraft area is the cut off value for distinguishing infected grafts from non-infected grafts, but this is based on a small number of patients. It is also considered that linear, diffuse, and homogeneous uptake with projection of the vessel is highly suggestive of infection. Although in the past diabetes and use of antibiotics were supposed to degrade image quality, two recent studies demonstrated that diagnostic accuracy was not affected.[44,45]

In a meta-analysis, the sensitivity of single 18F-FDG PET without combined low dose or contrast enhanced CT in diagnosing VGEI in patients with a suspected VGEI was 0.94 (95% CI 0.88 − 0.98), with a specificity of 0.70 (95% CI 0.59 − 0.79).[37] 18F-FDG-PET combined with CT (adding low dose or contrast enhanced CT) showed even better results, with a sensitivity of 0.95 (95% CI 0.87 − 0.99) and a specificity of 0.80 (95% CI 0.69− 0.89) (Fig. 1).[37]

2.8.3.2. White blood cell scintigraphy.
WBCS detects infected sites by visualizing the increase of accumulation of radiolabelled white blood cells over time. Recently, procedural guidelines for the labelling of the white clood cells, and for the correct acquisition and interpretation criteria for WBCS were published.[40] The diagnosis of VGEI infection is based on the presence of pathological accumulation of labelled white blood cells at the site of infection. At least two sets of images are required (2 − 4 and 20 − 24 hours after injection) and an increase in intensity or size with time is considered positive for an infection. When positive, SPECT/CT images are mandatory for exact localisation of the infection (soft tissue only, graft, or extension).[40]

WBCS is a very specific method, but it has some limitations. The procedure is time consuming and labour intensive, as the imaging needs to be performed at least at two different time points (preferably 2 − 4 and 20 − 24 hours after injection) and in a laboratory specifically equipped to perform leukocyte labelling. Furthermore, the diagnostic accuracy of WBCS depends on the region of the body in which the images are performed. The accuracy is somewhat lower in the central parts of the body than in peripheral parts (so in case of aortic VGEI) as the tracer is eliminated via the intestinal tract and physiologically taken up in the bone marrow, leading to a difficult interpretation of the aorta. Using antigranulocyte antibody scintigraphy as an alternative does not demand laboratory labelling, but does require dual time point imaging and is hampered by physiological uptake in bone marrow and excretion in the intestinal tract.[40] Moreover, the diagnostic accuracy of antigranulocyte antibody scintigraphy is, in general, somewhat lower than WBCS.[40]

The estimated sensitivity of WBCS (without SPECT/CT) in diagnosing VGEI in the most recent meta-analysis was 0.90 (95% CI 0.85 − 0.94) with a specificity of 0.88 (95% CI 0.81 − 0.94).[37] When WBCS was combined with SPECT/CT, the sensitivity increased to 0.99 (95% CI 0.92 − 1.00), with a specificity of 0.82 (95% CI 0.57 − 0.96) (Fig. 1).[37] It is, however, not recommended as the first imaging modality in diagnosing VGEI because of the aforementioned limitations and limited availability (Fig. 2).
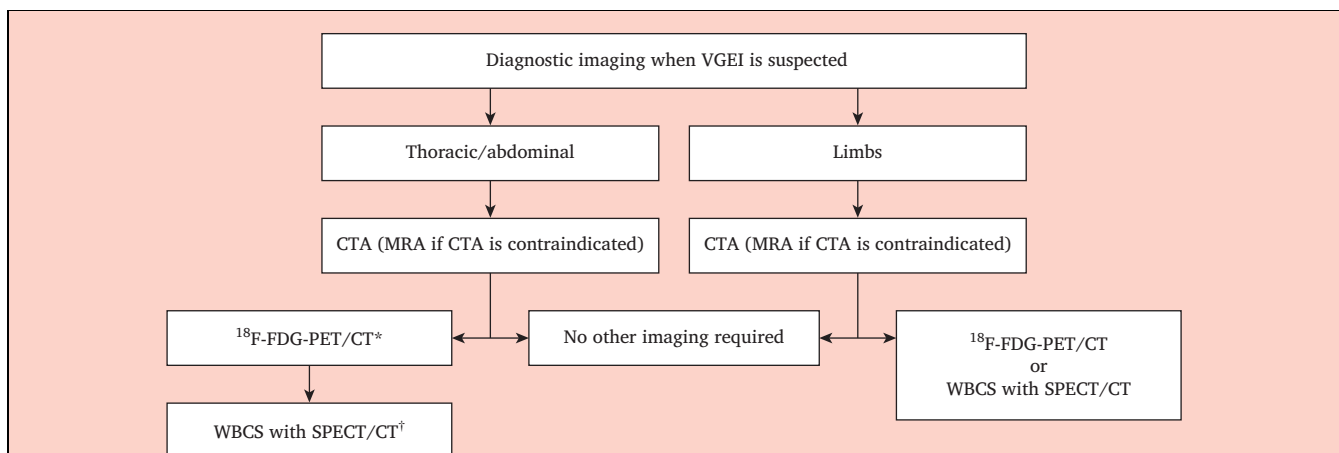
Figure 2. Imaging workflow if vascular graft/endograft infection (VGEI) is suspected, divided into thoracic/abdominal and limb grafts. CTA = computed tomography angiography; MRA = magnetic resonance angiography; 18F-FDG-PET/CT = 18F-fluoro-D-deoxyglucose positron emission tomography/computed tomography; WBCS = white blood cell scintigraphy; SPECT/CT = single photon emission computed tomography/computed tomography. *18F-FDG PET/CT can add more information, particularly in inconclusive CT. In some high grade infection cases a second imaging modality as 18F-FDG PET/CT and/or WBCS combined with SPECT/CT may be useful to map the extent of the infection. †WBCS can be applied if available otherwise, 18F-FDG PET/CT can be used.

Recommendation 1

Once vascular graft/endograft infection is suspected, exhaustive evaluation of clinical status, signs of infection and patient comorbidities according to the MAGIC criteria is recommended.

Class	Level	References
I	C	Lyons *et al.* (2016),[1] Back (2014),[6] Teebken *et al.* (2012)[12]

Recommendation 2

When a vascular graft/endograft infection is suspected, it is recommended that every effort is made to obtain microbiological proof of infection.

Class	Level	References
I	C	Baron *et al.* (2013)[21]

Recommendation 3

To obtain microbiological proof of vascular graft/endograft infection, the yield of at least three deep rather than superficial samples should be considered.

Class	Level	References
IIa	C	Baron *et al.* (2013),[21] Padberg *et al.* (1995)[23]

Recommendation 4

Microbiological investigation of negative pressure wound therapy foams should not be performed in order to support the diagnosis of vascular graft/endograft infection.

Class	Level	References
III	C	Scherrer *et al.* (2016)[27]

Recommendation 5

Sonification of intra-operatively harvested graft material may be considered in order to improve the micro-organism detection rate.

Class	Level	References
IIb	C	Wengrowitz *et al.* (1991)[30]

Recommendation 6

For patients suspected of vascular graft/endograft infection, the use of ultrasound as the sole diagnostic modality is not recommended.

Class	Level	References
III	C	Bruggink *et al.* (2011)[34]

Recommendation 7

For suspected vascular graft/endograft infection, CTA is recommended as the first line diagnostic modality.

Class	Level	References
I	B	Reinders Folmer *et al.* (2018)[37]

Recommendation 8

For patients suspected of vascular graft/endograft infection, if CTA is contra-indicated, the use of MRA may be considered.

Class	Level	References
IIb	C	Shahidi *et al.* (2007)[38]

Recommendation 9

For patients with a clinical suspicion of vascular graft/endograft infection and with non-convincing findings on CTA, the use of 18F-FDG-PET combined with low dose CT is recommended as an additional imaging modality to improve diagnositc accuracy.

Class	Level	References
I	B	Reinders Folmer *et al.* (2018)[37]

Recommendation 10

In patients with a clinical suspicion of peripheral vascular graft/endograft infection, single photon emission computed tomography, if available, is recommended as an additional imaging modality to improve diagnostic accuracy.

Class	Level	References
I	B	Reinders Folmer *et al.* (2018)[37]

3. STRATEGIES TO PREVENT GRAFT OR ENDOGRAFT INFECTION

3.1. Raw materials

Currently, vascular devices are mainly made of two different polymers, PET or expanded polytetrafluoro-ethylene (ePTFE) for soft materials, and different alloys, nitinol being the most used, for stents. The choice of these polymers and alloys is mainly related to their chemical and mechanical stability, rather than their properties for inhibiting micro-organism colonisation. They can be implanted as raw materials or associated with different adjuncts such as matrices of impregnation or surface treatment. There is no strong evidence on differences of susceptibility to infection of synthetic vascular raw biomaterials. Differences of susceptibility to infection between PET and ePTFE have been evaluated *in vitro* and *in vivo* with conflicting results. *In vivo* studies did not find differences in infectability between PET and ePTFE materials,[46–48] or less adherence of bacteria for ePTFE than for PET.[49] Although all bacteria are able to adhere to an inert support, some bacteria, such as coagulase negative staphylococci (e.g., *S. epidermidis*) or streptococcus viridans (e.g., *Streptococcus mitis oralis*), show a high propensity for adhesion to foreign materials.[50] No significant difference has been found *in vivo* between PET and glutaraldehyde treated bovine pericardium, used as aortic patches, to resist bacterial infection.[51] Geometric configuration, early plasma protein adhesion, and healing sequence can influence the risk of bacterial adherence with subsequent infection.[52] Bacterial strains demonstrated a greater affinity to

velour knitted PET than to ePTFE grafts.[51] No difference of susceptibility to infection between different alloys currently used for stents or stent grafts has been demonstrated.[48]

3.2. Logistics and peri-operative care

3.2.1. *Staphylococcus* nasal carriage. The high prevalence of nasal carriage of *S. aureus* in the general population and its role in potentially severe VGEIs raises the question of the beneficial effect of its decolonisation in patients undergoing vascular surgery.[53] In a prospective study, *S. aureus* nasal carriage using a PCR technique was not different in patients with or without post-operative SSIs.[54] In another prospective study, patients undergoing aorto-iliac surgery were screened for *S. aureus* nasal carriage and, if positive, were treated with mupirocin nasal ointment and chlorhexidine body washes, and compared with a historical control group of patients who tested positive but received no treatment. The incidence of *S. aureus* SSI was significantly lower in patients who were screened positive and who were treated for methicillin resistant *S. aureus* (MRSA) nasal carriage compared with patients negative for nasal MRSA carriage (0% *vs.* 13.6%).[55] In addition, both 30 day mortality reintervention rates were significantly lower in the treated group (1.3% *vs.* 13.6%).[55] Of note, *S. aureus* eradication in this setting was associated with a decrease in *S.aureus* related SSIs but not in the SSIs due to other bacteria, which may be explained by the competing behaviour of bacteria causing SSI.[55]

3.2.2. Peri-operative care

3.2.2.1. *Shower regimen and hair removal.* In a meta-analysis, there was no evidence of any benefit from a pre-operative bathing or shower regimen with antiseptic agents over unmedicated bathing.[56]

A number of measures have been found to reduce the risk of SSI of 51% in patients undergoing open and endovascular elective surgery or elective lower limb amputation: peri-operative normothermia; hair removal the day before surgery; and discipline in aseptic care in the operating room.[57]

3.2.2.2. *Antimicrobial prophylaxis.* In a meta-analysis, antimicrobial prophylaxis with broad spectrum systemic antibiotics significantly reduced the risk of wound infection and early graft infection in arterial reconstructions (relative risk [RR] 0.25, 95% CI 0.17 — 0.38; and RR 0.31, 95% CI 0.11 — 0.85, respectively).[56] In all patients undergoing open or endovascular abdominal aortic aneurysm repair, therefore peri-operative systemic antimicrobial prophylaxis is recommended.[58] However, antimicrobial prophylaxis for more than 24 hours does not seem to bring any additional benefit.[56]

Antimicrobial prophylaxis for vascular surgery should cover the bacteria most likely to be responsible for SSIs and achieve adequate tissue levels at the time of incision and throughout the procedure to prevent any bacterial colonisation of injured skin/soft tissue and implant. First or second generation cephalosporins are the most widely used agents owing to their profile of tolerance and antibacterial spectrum that cover methicillin susceptible staphylococci (i.e., *S. aureus* and coagulase negative staphyloccoci), streptococci, and some Gram negative bacilli. A meta-analysis of 22 RCTs concluded that prophylactic systemic antibiotics for patients undergoing peripheral arterial reconstruction reduced the risk of SSI (RR 0.25, 95% CI 0.17 — 0.38) and early VGI (RR 0.31, 95% CI 0.11 — 0.85).[56] In the same study, no difference in the protective effect on SSI rate was noted between first or second generation cephalosporins, penicillins with lactamase inhibitors, aminoglycosides, or vancomycin.[56]

The coverage of MRSA and/or coagulase negative staphyloccoci may be considered according to the local prevalence of these strains, even though no significant difference concerning SSI rates using cefazolin plus vancomycin or daptomycin *vs.* cefazolin alone has been found.[59,60]

Antibiotic prophylaxis has the best efficacy when administered before the incision (ideally within 30 min) with re-injection for longer interventions according to the half life of the compound (i.e., two hours for cefuroxime and four hours for cefazolin).[56]

3.2.2.3. *Gloves.* A prospective RCT did not establish the benefit of intra-operative glove change before handling prosthetic grafts.[61]

3.2.2.4. *Wound closure.* In a retrospective study including all SSIs after lower extremity revascularisation procedures between 2012 and 2016, meticulous wound closure with a monofilament absorbable suture has been shown to be superior to staples in decreasing SSI rates.[62]

3.3. Antibiotic prophylaxis during dental extraction

Beyond the peri-operative risk of SSI, the implant can be infected at any time after the intervention, especially in the presence of bacteraemia (i.e., secondary haematogenous related to SSI). Analogous to prosthetic cardiac valves, antibiotic prophylaxis after VG for secondary infection may follow the recommendations of the ESC and American College of Cardiology/American Heart Association.[63] In their most recent guidelines, antimicrobial prophylaxis was recommended for patients with a prosthetic cardiac valve for high risk procedures such as dental procedures involving the manipulation of the gingival or peri-apical region of teeth or perforation of the oral mucosa, including scaling and root canal procedures.[63] Antibiotic prophylaxis has therefore been proposed recently for those patients with an aortic prosthesis, whether placed by open surgical repair or endovascular aneurysm repair (EVAR), before any dental procedure involving the manipulation of the gingival or peri-apical region of teeth or perforation of the oral mucosa, including scaling and root canal procedures.[58]

Recommendation 11		
In every case where a vascular graft/endograft is implanted, antimicrobial prophylaxis to cover the first 24 hours, by intravenous administration of a first/second generation cephalosporin or vancomycin in the event of penicillin allergy, is recommended.		
Class	**Level**	**References**
I	A	Stewart et al. (2007)[56]

Recommendation 12		
Before implantation of any vascular graft/endograft, elimination of any potential source of sepsis, especially of dental origin, should be considered.		
Class	**Level**	**References**
IIa	C	Habib et al. (2015)[63]

Recommendation 13		
Antimicrobial prophylaxis to prevent vascular graft/endograft infection should be considered before any dental procedure involving the manipulation of the gingival or peri-apical region of teeth or perforation of the oral mucosa, including scaling and root canal procedures for patients previously operated on with a vascular/endovascular graft.		
Class	**Level**	**References**
IIa	C	Habib et al. (2015)[63]

4. GENERAL THERAPEUTIC STRATEGIES

In view of the rarity of VGEI, the complexity of diagnosis and treatment, and the difficulty of the interventions and severity of complications, centralisation of the patients suffering from VGEI is clearly indicated. Accordingly, patients should be transferred to specialised high volume centres with multidisciplinary experience in VGEI, including angiologists, vascular, cardiovascular and general surgeons, microbiologists and radiologists.

4.1. Antimicrobial therapy

4.1.1. Choice of antimicrobial therapy. Antimicrobial therapy is an integral part of VGEI treatment. In the acute phase intensive antimicrobial therapy with broad spectrum antibiotics or antibiotics directed against the most likely infecting organisms is indicated to control infection and sepsis. In the choice of antimicrobial therapy the fact that the graft material may be covered with a biofilm and also the local epidemiology of resistance patterns have to be considered. In some specific situations, the addition of antifungal agents should be considered, especially in visceral fistula cases. Once the responsible infecting organisms are known the spectrum should be narrowed if possible.

4.1.2. Duration of treatment. There is no consensus on the optimal length of antimicrobial therapy for VGEI. If

prosthetic material can be removed and a thorough debridement of all infected tissue can be performed, a minimum of two weeks of intravenous therapy, if possible, followed by an oral regimen for another two to four weeks is indicated. If the infected material is replaced by a new VG, four to six weeks of intensive antimicrobial therapy is usually proposed to prevent recurrent infection. Many authors favour a total treatment time of three to six months in this situation, and some even advocate one year of treatment. In those patients in whom general conditions preclude any surgery, lifelong treatment should be considered.[64,65] This can be an option in patients at higher risk of surgery, especially in low grade infections with less virulent infecting organisms, susceptible to suitable antibiotics, and without other complications. In some cases, the infection cannot be totally eradicated but kept under control by year long or even lifelong therapy.[66]

4.1.3. Antimicrobial therapy management. Because of the complexity of interpreting microbiological tests results and the permanent evolution of antimicrobial therapies and micro-organism resistance, antimicrobial therapy management must be done by an infectious diseases specialist within a multidisciplinary team, including vascular surgeons, radiologists, microbiologists, anaesthetists, and gastrointestinal and pulmonary specialists for cases with a concomitant fistula.[65,67]

4.1.4. Isolation. Patients with multidrug resistant (MDR) bacteria (such as MRSA, but not methicillin resistant coagulase negative staphylococci, extended spectrum beta lactamase producing Enterobacteriaceae, or glycopeptide resistant enterococi) should be isolated and should remain isolated during the hospital stay as carriage is prolonged, especially in patients receiving antibiotics. The wearing of gloves is required for the manipulation of any biological material, but this is not specific to MDR bacteria (gown for any contact with the patient, mask if pulmonary colonisation or infection). Both colonised and infected patients with these bacteria should be managed in a single patient room to reduce the risk of spread to other patients.[68]

4.2. Surgical principles

Historically, total removal of the infected VG or EG, debridement and rinsing with antiseptic solution of the infected area, and extra-anatomic reconstruction (EAR) outside the infected field was considered as the gold standard to avoid recurrent infection. This procedure should be performed in two stages when possible. However, this approach is not always feasible, often not easy, and increases the risk of complications like stump blowout. Therefore, most authors now prefer an *in situ* reconstruction (ISR) with infection resistant material combined with removal of the infected graft material, aggressive debridement of the arterial bed and targeted antimicrobial therapy.[67,69] In most situations the

results with ISR are at least equivalent to extra-anatomic repair.

In thoracic and abdominal procedures especially, it is recommended that any VG and anastomosis should be covered with viable tissue such as omentum, muscle, or pericardial patch.[70] Direct contact with viscera or organs should also be avoided. If no viable tissue is available, a bovine pericardial patch can be used. Anastomoses or suture lines can be reinforced with fascia or pledgets.

Recommendation 14		
Antimicrobial therapy is recommended in every patient with an infected graft/endograft.		
Class	Level	References
I	B	Darouiche (2004),[65] Revest et al. (2015)[67]

Recommendation 15		
For the diagnosis and treatment of vascular graft/endograft infection it is recommended that the patient be transferred to specialised high volume centre with multidisciplinary experience in this pathology.		
Class	Level	References
I	C	Consensus of expert opinion

5. SUPRA-AORTIC TRUNKS

5.1. Specific aspects

5.1.1. Incidence. The exact incidence of VGEI in the supra-aortic trunks (SAT) is unknown, but probably extremely low. SAT VGEI includes prosthetic patch, bypass, and stent graft infections.[71] However, infection rates might be underestimated, owing to lack of recognition and under reporting, as in other locations.

Over the last three decades, a total of 140 cases of SAT patch/bypass infections have been reported, mostly involving carotid patches. An overall incidence of 0.25% – 0.5% was reported in a systematic review of carotid endarterectomies involving PET patches.[72] Because of its infrequent occurrence, it is difficult to identify the aetiology of SAT EG or endograft infection (EGI), but haematoma could be a risk factor in promoting the development of early infection.[73,74]

The incidence of SAT EGI is low, with an estimated incidence of less than one EGI in 10 000 cases.[75] A systematic review of the literature identified only 12 patients with SAT stent graft infections in the last three decades: eight patients presented with infected carotid stent graft, three with infected subclavian stent graft, and one with an infected tandem brachiocephalic and subclavian artery stent graft.[71]

5.1.2. Clinical presentation. Half of all reported infections occurred within the first four post-operative months and involved carotid patches. The most common clinical presentation for early infection is abscess, neck mass, and haemorrhage. When infection presents later, the main symptom is a draining sinus. Patients with SAT stent graft infection typically present with fever, malaise, and pain. SAT stent graft infection is mostly encountered after stent graft implantation for carotid blowout syndrome.[76,77] However, it is often a diagnosis of exclusion after other sources of bacteraemia are excluded or inconclusive.[72,74,78] In the setting of early post-operative infection, *S. aureus* is the most commonly encountered micro-organism, while *S. epidermidis* is the predominant pathogen in patients who present with late infections.[71,79]

5.1.3. Specific diagnostic modalities. US allows the evaluation of the patency of the revascularisation, the presence of a collection, and its characterisation. It has been highlighted that carotid patch corrugation on US might be an early warning sign of VGI.[80] CTA in combination with cerebral CT may show pseudo-aneurysm formation, abscess, thrombosis or perivascular stranding, and brain abscesses.[81]

5.2. Treatment options for supra-aortic trunk vascular graft/endograft infection

5.2.1. Conservative treatment. Conservative treatment of SAT VGEI is not recommended in patients fit for intervention because of the risk of suture line rupture for patch/bypass and vascular wall necrosis for stent graft, potentially leading to uncontrollable major bleeding in the chest and tracheal compression in the neck. However, successful conservative treatment of the infected stent graft using parenteral antimicrobial therapy alone has been described.[75]

5.2.2. Endovascular treatment. Endovascular treatment is also an option for SAT VGEI. It is mostly used in life threatening presentations in order to control a major bleeding related to VGEI.[82] For acute bleeding related to blowout syndrome, open surgery in an irradiated area may be challenging, and emergency operative ligation may be associated with high rates of major morbidity, meaning that an endovascular approach is usually preferred. A systematic review and meta-analysis including 559 patients demonstrated that both coil embolisation and reconstruction with stent grafts may be safe treatment options for carotid blowout syndrome: peri-operative mortality was 3% for patients treated by carotid embolisation and 12% for patients treated with covered stent grafts, while the peri-operative stroke rate was 1% in both groups.[83]

The stent graft first strategy can also be considered as a bridge to definitive therapy in unstable patients. This

Open surgery	Management		Outcomes
Described cases *n* = 140 Described treatment *n* = 138	Removal of infected material + arterial reconstruction *n* = 86	Veins (*n* = 71) (1 muscle flap)	None (*n* = 41), nerve injury (*n* = 15), stroke (*n* = 8), re-infection (*n* = 4), bleeding (*n* = 3), cardiac failure (*n* = 2), missing data (*n* = 2)
		SFA (*n* = 7) (4 muscle flaps)	None (*n* = 5), nerve injury (*n* = 1), re-infection (*n* = 1)
		Prosthetic grafts (*n* =4)	None (*n* = 3), stroke (*n* = 1)
		Arterial homografts (*n* = 3)	None (*n* = 3)
		Primary closure + flap (*n* = 1)	None (*n* = 1)
	Phlegmon excision (*n* = 2)		None (*n* = 2)
	EndoVAC (*n* = 10)		None (*n* = 10)
	Ligation (*n* = 7)		None (*n* = 6), nerve injury (*n* = 1)
	Covered stent (*n* = 7) (Including 1 muscle flap)		None (*n* = 21), cardiac failure (*n* = 1)
	Conservative treatment (*n* = 26)		None (*n* = 3), bleeding (*n* = 1)

Figure 3. Management and outcomes of supra-aortic trunk patch/bypass infection.[71] SFA = superficial femoral artery; VAC = vacuum assisted closure.

strategy allows a controlled and semi-elective graft revision, including excision and reconstruction. Patients should receive intravenous broad spectrum or microbiology based antimicrobial therapy, followed by long term treatment.

The endo vacuum assisted closure (VAC) technique is a hybrid approach that has been used in 10 cases of infected SAT reconstructions. The EndoVAC technique is a three step procedure: relining of the infected reconstruction with a stent graft; removal of the infected VG without clamping; and use of NPWT to permit granulation.[84,85] This technique provided good results but was performed in few patients selected on an individual basis for the treatment as they had severe co-morbidity and adverse anatomy, providing limited evidence.

5.2.3. Reconstruction
5.2.3.1. Graft material. A surgical approach with total explantation of infected foreign material is recommended in the elective setting only. Replacement of the explanted bypass/patch or arterial segment is usually mandatory to avoid cerebral ischaemia or infarction. However, primary ligation of the vessel may be considered in emergency life threatening situations, if the infected reconstruction is already thrombosed without neurological symptoms or with an already completed cerebral infarct, to avoid revascularisation syndrome and cerebral haemorrhage.[86]

Autologous material is usually considered as the first line option for reconstruction. Because of the typically short length of these reconstructions, autologous saphenous vein reconstruction (bypass or patch) can be performed in the majority of cases.[79,87,88]

5.2.3.2. Partial or total explantation. A direct surgical approach with total explantation of the foreign material is mostly performed in non-emergency conditions in an attempt to avoid emergency procedures for life threatening haemorrhage in the neck or chest. Usually, bypasses and arterial segments with infected patches or stent grafts are short, except bypasses starting from the ascending aorta.[89] Consequently total explantation is usually performed. Obtaining proximal arterial control is mandatory, even using an occlusion balloon or at an unscarred site through a sternotomy or a thoracotomy, in order safely to enter the cervical phlegmon and minimise peri-operative complications.[90]

The approach that involves proximal arterial control is even more relevant in cases with active bleeding due to infection associated arterial wall breakdown.[90–92] Indeed, primary arterial control through the same cervical incision may be difficult, with the risk of significant blood loss and increased inadvertent peripheral nerve injuries.

Partial explantation with local wound debridement is less often performed.

5.2.3.3. Adjunctive therapy. A muscle flap may be considered as a possible adjunctive option for SAT VGEI infection. Sternocleidomastoid or pectoralis major muscle flaps have been reported in 11 cases following SAT patch/bypass infections. Of these 11, seven flaps were following complete removal of the infected material and arterial reconstruction, while four flaps were combined with local wound debridement.[93–96] None of the 11 patients died from a related cause. Of the seven patients who underwent complete removal of the infected material and arterial reconstruction, one stroke and two transient nerve injuries were reported. Of the four patients who underwent local wound debridement, one pseudo-aneurysm occurred requiring the placement of a covered stent.

5.3. Follow up and prognosis

Over the last three decades, 140 cases of SAT patch/bypass infections have been reported and 138 treatment modalities described: total removal of infected material and arterial reconstruction in 86 cases; phlegmon excision in two cases; the EndoVAC technique in 10; ligation in seven; endovascular treatment using covered stent grafts in seven; and conservative treatment in 26 cases.[71] Peri-operative complications occurred in 27.9% of cases. Six patients died from related causes with a median follow up of 36 months (Fig. 3).[71]

Of the 12 cases of SAT stent graft infections reported over the last three decades, 11 treatment modalities were described: stent graft removal and arterial reconstruction in six cases; stent graft removal without arterial reconstruction in two cases; carotid embolisation in two cases; and conservative treatment in one case. Peri-operative complications were described in 54.6% of cases. Median follow up was 4.5 months, and five patients died from related causes (Fig. 4).[71]

Recommendation 16

When patch corrugation is found on ultrasound follow up after carotid endarterectomy further investigations may be considered to exclude a vascular graft infection.

Class	Level	References
IIb	C	Lazaris et al. (2005)[80]

Recommendation 17

For patients with supra-aortic trunk vascular graft/endograft infection, total removal of infected material followed by reconstruction with autologous material is recommended.

Class	Level	References
I	C	Lejay et al. (2018),[71] Son et al. (2014),[79] Grazziotin et al. (2002),[87] Kaviani et al. (2006)[88]

Recommendation 18

The EndoVAC technique may be considered as a treatment option in selected patients with supra-aortic trunk vascular graft/endograft infection when neither total removal of infected material nor when usual conservative VAC therapy are considered feasible or safe.

Class	Level	References
IIb	C	Kragsterman et al. (2011),[84] Thorbjornsen et al. (2016)[85]

Recommendation 19

In the emergency setting with active bleeding in patients with supra-aortic trunk vascular graft/endograft infection, a combined endovascular and surgical approach may be considered.

Class	Level	References
IIb	C	Younis et al. (2006)[82]

Recommendation 20

Conservative treatment, including antimicrobial therapy without reconstruction, for supra-aortic trunk vascular graft/endograft infection may be considered for patients unfit for surgery.

Class	Level	References
IIb	C	Myles et al. (2000)[75]

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6. THORACIC/THORACO-ABDOMINAL AORTA

6.1. Specific aspects

6.1.1. Incidence. The frequency of thoracic aortic VGEI is reported to be up to 6%, with mortality rates, depending on the clinical presentation, of up to 75%.[15,64] Together with the increasing number of procedures performed on the thoracic aorta, including thoracic EVAR (TEVAR), the incidence of thoracic VGEI is also increasing. Additionally, thoracic VGEI is often associated with AEsF, ABF, or aorto-pulmonary fistula (APF), which makes treatment more complicated, with the need (besides the aortic reconstruction) for adjunctive surgical procedures to repair the oesophageal or bronchial lesion.[97—99]

A systematic review identified 43 studies reporting on 233 patients with 49 VGI and 184 EGI. Only four were multicentre studies, which included 107 patients, all with EGI. The remaining 39 single centre studies included 49 patients with VGI and 77 with EGI.[100] In two large registries on complications after TEVAR, the incidence of AEsF was estimated to be 1.7% and 1.5%, respectively.[97,99] In a systematic review, the association with AEF was significantly more frequent (60% vs. 31%) and the time interval from index procedure to infection was significantly shorter (17 ± 21 months vs. 32 ± 61 months) with EG compared with VG, respectively.[100]

6.1.2. Clinical presentation. The clinical symptoms of thoracic aortic VGEI can range from unexplained fever, as observed in post-implantation syndrome, to sepsis, massive bleeding, and shock. Owing to the depth of the thoracic aorta, visible local signs of infection are mostly absent. Septic emboli can cause secondary loci of infection or even abscesses. For AEsF or ABF, haematemesis or haemoptysis may be the first symptom. This bleeding may be massive, especially for AEsF but is often preceded by self limiting "herald bleeding".

6.1.3. Specific diagnostic modalities. Definite diagnosis mostly demands a CTA showing perigraft fluid, air in the aneurysm sac or surrounding it, or abscess formation in the surrounding tissues.[1,8] 18F-FDG-PET/CT should be performed when low grade infection is suspected but not confirmed by CT.[43] When an AEsF is present, the prosthetic material may be seen protruding in the oesophagus on oesophagoscopy. In case of an ABF the defect in the bronchus can only be seen when it is centrally located, e.g., in the left main bronchus. Diagnostic examinations should

Figure 4. Management and outcomes of supra-aortic trunk SAT stent graft infection.[71]
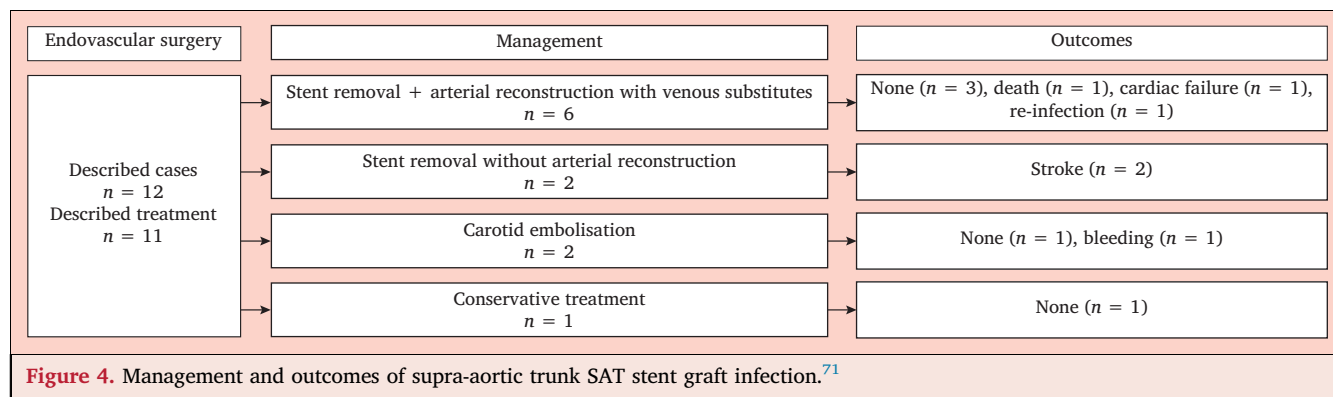
be performed without delay when infection of a thoracic aortic VGEI is suspected.[66]

6.2. Thoracic vascular graft/endograft infection without fistula

6.2.1. Conservative treatment. Although surgical principles dictate control of sepsis, removal of all infected prosthetic material, and reconstruction in a clean field, this is not always achievable because it places a huge burden on an already sick patient (Fig. 5). Therefore,

staged repair or conservative treatment are the only options.[101]

6.2.1.1. Percutaneous drainage. In the presence of perigraft fluid collections or abscesses, percutaneous drainage under ultrasound or CT guidance can be performed in combination with antimicrobial therapy. A 10 — 14 F pigtail catheter or a 12 — 20 F drain is inserted percutaneously and left in place until the collection is totally or sufficiently drained.

6.2.1.2. Irrigation. Irrigation with saline or an antiseptic solution can be used in order to dilute the bacterial burden


Figure 5. Proposed algorithm for the management of thoracic aortic graft/endograft infection. * In a single or staged prodecure. † Materials that can be used are cryoprerved allografts, treated PET grafts or biological xenografts. VGEI = vascular graft/endograft infection; PET = polyethylene terephthalate.
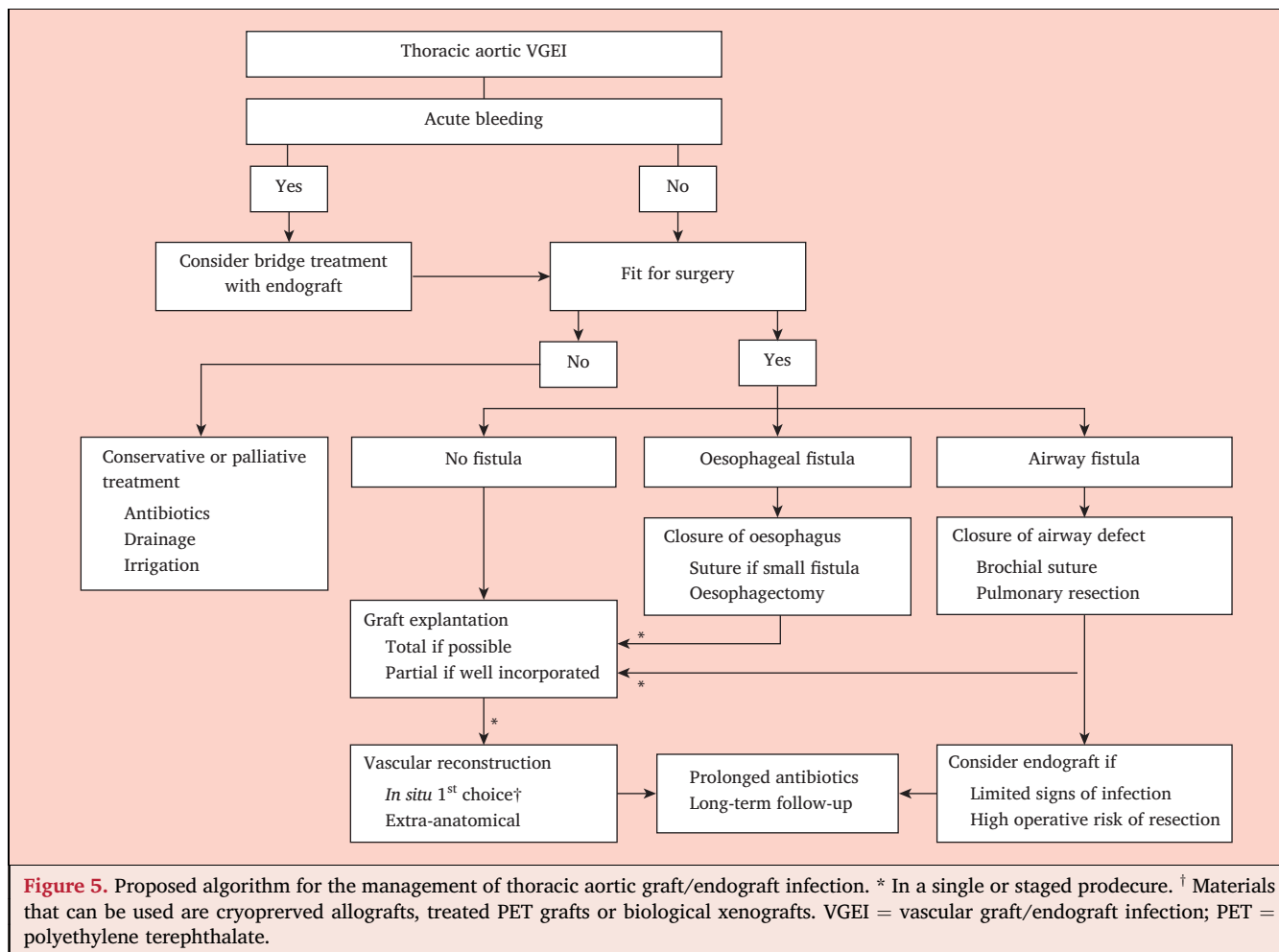
in prosthetic and peri-prosthetic tissues. It can be performed through percutaneous drains or after a surgical procedure with aortic reconstruction.[66]

In a systematic review of single centre series, conservative treatment (antimicrobial therapy with or without percutaneously inserted drainage of fluid collections or flushing) was performed in 2% of patients with VGI (n = 1/49) and 17% with EGI (n = 13/77). The mortality rate was 100% at 30 days in VGI group; and 38% at 30 days, 75% at one year, and 100% at five years in the EGI group.[100]

Five studies provided data on one year mortality in both conservatively (n = 12) and surgically (n = 42) treated patients.[102–106] One year mortality was 75% and 50%, respectively, without any significant difference.[100]

6.2.2. *In situ* reconstruction. Removal of the infected graft material, aggressive debridement of the arterial bed, and arterial reconstruction with suturing in healthy non-infected tissue using infection resistant material constitute the basis of this treatment modality

6.2.2.1. *Specific techniques.* The operative technique largely depends on the VG or EG location. If the prosthesis extends into the aortic arch, a median sternotomy or a clamshell incision is indicated, and the intervention needs to be performed under total cardiopulmonary bypass, circulatory arrest, and selective cerebral perfusion. If the proximal extent of the VG or EG is distal to the left subclavian artery (LSCA), the procedure can be performed through a left thoracotomy with single lung ventilation and left heart bypass. Dissection of the proximal neck, usually between the left common carotid artery (LCCA) and the LSCA, can be difficult owing to the inflammation caused by the infection or the previous intervention. Care should be taken not to damage adjacent structures, like the lung, vagus nerve, or oesophagus. If extensive reconstructions need to be performed, measures like cerebrospinal fluid drainage may be considered in order to reduce the risk of spinal cord ischaemia. Intraoperative evaluation using motor evoked or somatosensory evoked potentials may also be used. If the VG or EG extends to the level of the LSCA and clamping between the LCCA and the LSCA is not possible, hypothermic circulatory arrest is needed to allow complete prosthetic material excision.[107] When exposure of the thoracoabdominal aorta is required, a thoracophrenolaparotomy is the preferred approach. As in primary procedures, the splanchnic arteries can be perfused with normothermic blood and the renal arteries with cold crystalloids during cross clamping.

6.2.2.2. *Graft materials.* Cryopreserved aortic allografts have been proposed for the replacement of infected thoracic VGEI.[102,108] As a biological material, allografts have demonstrated a higher resistance to infection than synthetic VGs, but are exposed to the subsequent risk of degeneration, rupture, and bleeding when the infection is caused by necrotising organisms, such as *P. aeruginosa* or *Candida spp.*[66,109–111] Long term durability results,

especially with regard to the development of calcification and aneurysms when used in the thoracic aorta, are still lacking.[64]

Treated PET VGs, such as rifampicin soaked and silver coated (with or without triclosan) VGs, have been proposed in order to decrease the risk of early infection.[64,66,112] In a series including mainly explanted infected abdominal endografts, treated PET VGs have been shown to provide better results in terms of prevention of re-infection and five year overall survival than standard PET VGs (53% *vs.* 12%) These results can probably be extrapolated to the TEVAR setting. Bovine pericardium has been reported as a technical option for ISR of thoracic aortic VGEI, tailoring a custom made tube by sewing pericardial sheets.[113–115] While promoted in case series, this technique still needs further studies and longer follow up.

6.2.2.3. *Adjunctive therapy.* The recommended VG coverage to avoid its direct contact with surrounding organs like lung or oesophagus using the surrounding tissues is often not possible. Therefore, it is advised to cover the VG with other viable tissue. Intercostal flap coverage can be used, as well as pericardial or omental flaps. An intercostal flap has limited volume and is best prepared at the moment of thoracotomy to avoid damage caused by the retractor. When a pericardial flap is used, the pericardial defect may need to be repaired with synthetic material. Omentum can be prepared by laparoscopic access and routed through the diaphragm via the aortic hiatus, to cover the VG and fill a dead space after mediastinal debridement.[66] More extensive muscular flaps, such as latissimus dorsi or serratus muscle have been proposed.[101] If there is no viable tissue available, use of a bovine pericardial patch is suggested.

6.2.3. Extra-anatomic reconstruction. To avoid reconstruction in a contaminated field and recurrent infection, EAR outside the infected field and secondary aortic ligation with removal of the infected VG or EG can be performed in one or two stages.

6.2.3.1. *Technique.* To restore distal perfusion after aortic ligation, axillo-bifemoral or bilateral axillofemoral bypasses can be performed, but retrograde blood flow to the visceral organs under all these circumstances may be insufficient.[101] The most commonly used EAR is the so called ventral aorta, consisting of a retrosternally placed VG that originates from the ascending aorta, the distal anastomosis being on the supracoeliac abdominal aorta or more distally, on the infrarenal aorta or iliac arteries.[101,116] If possible, this reconstruction is performed in two steps, the first step being the bypass through sternotomy and upper laparotomy, and the second step being the removal of the thoracic VG or EG through thoracotomy.[66] The main limitations of this technique are acute bleeding, or involvement of the aortic arch because of the necessity to intervene first on the infected VG or EG.[100] Depending on the situation, alternative routes can be used, such as a posterior pericardial bypass.

6.2.3.2. Stump management.
The aortic stump is oversewn and covered with an omental or muscular flap in order to reinforce the stump and diminish the risk of blow out.[101] Typically, the aorta is transected just distal to the LSCA origin or at the level of the diaphragm. Alternatively, staplers can be used to divide the aorta, but these stapled stumps should also be covered with viable tissue, such as pedicled intercostal muscle flap.[101]

6.2.4. Partial or total graft explantation.
Partial removal of graft material is usually not an option as the whole of the VG or EG needs to be considered to be infected,[109] the only exception being when only part of the VG or EG is documented as being infected through clinical and imaging findings.[109]

In a meta-analysis, the one year mortality was 37% ($n = 15/41$) for graft material explantation *vs.* 85% ($n = 17/20$) for total or partial graft material preservation.[100] In a systematic review and meta-analysis including 96 patients with infected thoracic endografts (41 EG explantation, 55 EG preservation), in hospital mortality was 37% *vs.* 42%, while late mortality was 46% *vs.* 82% for EG explantation and EG preservation, respectively.[117]

Recommendation 21

For persistent fever or inflammatory symptoms after implantation of a thoracic graft/endograft, further diagnostic investigations are recommended in the search for infection.

Class	Level	References
I	C	Lyons *et al.* (2016)[1]

Recommendation 22

For fit patients with proven thoracic/thoraco-abdominal vascular graft/endograft infection, total graft explantation is recommended.

Class	Level	References
I	B	Kahlberg *et al.* (2019),[100] Moulakakis *et al.* (2013)[117]

Recommendation 23

For patients with *in situ* reconstructions of thoracic/thoraco-abdominal vascular graft/endograft infection, coverage of the newly inserted graft with autologous, and ideally vascularised, tissue is recommended.

Class	Level	References
I	C	Spiliotopoulos *et al.* (2018),[66] Roselli *et al.* (2014)[101]

Recommendation 24

For patients with thoracic vascular graft/endograft infection that are at major risk of surgery, conservative treatment may be considered.

Class	Level	References
IIb	B	Czerny *et al.* (2014),[97] Kahlberg *et al.* (2019),[100] Chiesa *et al.* (2010)[111]

Recommendation 25

For patients with suspected thoracic graft/endograft infection, in the absence of fistulisation to the oesophagus or airway, or generalised sepsis, prolonged antimicrobial therapy combined with drainage of peri-graft fluid and/or irrigation, may be considered.

Class	Level	References
IIb	C	Kahlberg *et al.* (2019)[100]

Recommendation 26

For patients with thoracic/thoraco-abdominal vascular graft/endograft infection, partial explantation may be considered if infection is limited.

Class	Level	References
IIb	C	Kahlberg *et al.* (2019)[100]

Recommendation 27

For the reconstruction of thoracic/thoraco-abdominal vascular graft/endograft infection, cryopreserved allografts may be considered the first choice graft material.

Class	Level	References
IIb	C	Smeds *et al.* (2016)[110]

Recommendation 28

After extra-anatomic reconstruction for thoracic/thoraco-abdominal vascular graft/endograft infection, reinforcement of the aortic stump with autologous, and ideally vascularised, tissues should be considered.

Class	Level	References
IIa	C	Roselli *et al.* (2014)[101]

6.3. Thoracic vascular graft or endograft infection with oesophageal fistula

Thoracic aortic VGEIs associated with AEsF or ABF require a more complex and difficult treatment than those without the presence of a fistula. Even when an aortic VG is resected and the oesophagus repaired, the mortality is twice that of a VGEI without fistula.[117] An AEsF seems to occur more frequently and earlier in the thoracic aorta than in the abdominal aorta and after EG than VG implantation.[110]

Any strategy in this patient population needs to be highly individualised given the risks associated with major thoracic aortic surgery often in a hostile operative field.

6.3.1. Conservative treatment.
Conservative treatment of an AEsF is almost invariably fatal.[97,99,103,110] Only one conservatively managed case of secondary AEsF after EG implantation without sign of recurrent haemorrhage or chronic mediastinitis at 14 months has been described.[118] Conservative treatment should be considered palliative in patients unfit for major surgery.

6.3.2. Endovascular treatment as bridging therapy.
In patients presenting active and life threatening bleeding from

an AEsF, emergency EG insertion may be proposed as the primary strategy to control bleeding and restore haemodynamic stability.[119] This approach has been proposed as a "stopgap" strategy. However, it has considerable limitations, mainly related to the inability to eliminate the primary infected material and therefore should be considered mainly as a bridge to definitive treatment.[64]

In a national survey that analysed 25 cases of AEsF and ABF treated by TEVAR, the 30 day mortality rate was 28%. After a follow up of 23 months, overall mortality was 44% and the re-intervention rate due to TEVAR failure was 28%, with an associated re-intervention mortality rate of 60%. Patients treated by TEVAR associated with oesophageal or bronchial repair had a lower mortality than patients treated with TEVAR alone (30% *vs.* 55%).[99] The European Registry of Endovascular Aortic Repair Complications (EuREC) also showed a statistically significant survival benefit for a radical surgical approach compared with any other treatment strategy in cases of AEsF after TEVAR.[97] Other reports confirmed the idea that patients with VGEI, treated by initial TEVAR and considered unfit for subsequent definitive open surgical repair, had the worst prognosis, with mortality approaching 100% in most series.[64]

To conclude, patients with bleeding due to an AEsF can be treated successfully in the emergency setting by TEVAR, but once they have recovered and conditions are stable, a definitive surgical procedure must be performed to ensure durable results.[100,106]

6.3.3. Treatment of the oesophagus

6.3.3.1. Limited treatment of fistula. In the presence of an AEsF, both the aortic and oesophageal lesions need to be addressed. To avoid persistent or recurrent infection and/or mediastinitis and fistula recurrence, closure of the oesophageal lesion is needed. This can be performed together with the vascular reconstruction or as an isolated procedure.

Administration of methylene blue through a naso-oesophageal tube can help to localise a small oesophageal lesion. If the lesion is limited, primary oesophageal repair can be accomplished by a double layer of absorbable interrupted stitches to the oesophageal wall. In this case, the oesophageal repair is usually reinforced and covered by a pericardial flap or a previously prepared pedicled intercostal muscle flap.[64] Primary repair of the oesophageal defect always entails the risk of anastomotic leakage and mediastinitis with the subsequent need to remove the oesophagus. Therefore, most authors agree that limited fistula treatment has a limited place in treatment of AEsF.[112]

6.3.3.2. Radical fistula treatment. In most cases, the oesophageal lesion cannot be repaired primarily because it is too large, the oesophageal wall is of insufficient quality or ischaemic, or the infection is too extensive. In these situations, more radical treatment with partial or total resection of the oesophagus is indicated. When a staged procedure is used, in a first step (e.g., when a thoracic EG is inserted to control the bleeding) a cervicostomy with closure of the

cervical oesophagus and a nutritional gastro- or jejunostomy is performed. This is followed by removal of the oesophagus and reconstruction with gastric or colonic pull up in a second or third stage.[100]

6.3.3.3. Oesophageal prosthesis. The goal of the oesophageal endoprosthesis implantation is to close the fistula from the oesophageal side and prevent further contamination of the mediastinum. It can be performed as a standalone procedure or be combined with aortic stenting in the case of bleeding.[120] In a EuREC report, survival at one year was only 17% with oesophageal stenting alone *vs.* 43% when oesophagectomy was performed.[97] Therefore, this technique should only be considered in patients unfit for further surgery. Migration of the stent with need for repositioning can occur.[121]

6.3.3.4. Definitive treatment: one or two stage. When massive bleeding occurs, insertion of an EG to control the bleeding can be a life saving procedure. Multiple combinations of treatment options have been used to deal with AEsF, including arterial ISR, extra-anatomic bypass with concomitant primary oesophageal repair, or oesophagectomy with cervical oesophagostomy and secondary restoration of gastrointestinal tract continuity.[109,112]

Resection of the oesophagus and restoration of gastrointestinal continuity can be performed in a one stage or two stage procedure. The same applies to the vascular reconstruction and resection of the aortic graft.[105] The choice of strategy to be followed will often depend on the urgency of the situation, the condition of the patient and the possibility of controlling the infection.

Encouraging results were reported in a series of eight patients operated on for AEsF or ABF by means of a staged approach: emergency endovascular exclusion of the aortic rupture, followed by staged open surgical repair of the oesophageal or bronchial lesion and removal of the infected aortic graft with associated intercostal muscle flap interposition. At a mean follow up of 34 months, one death was observed (87.5% overall survival), and no conversion or aortic bleeding was recorded.[122]

In every case, patients should receive intensive medical and nutritional support to get them into a better general condition before being referred for open surgery.[123]

6.4. Thoracic vascular graft or endograft infection with airway fistula

In the EuREC registry, the incidence of ABF and APF is 0.56%.[98]

6.4.1. Endovascular treatment. Acceptable results have been described after treatment of ABF or APF by TEVAR, with the risk of delayed recurrent fistula or persistent infection, despite EG coverage with a muscle or pleural flap.[124,125] In the EuREC report, 15 patients presented with ABF, predominantly on the left side ($n = 14/15$) and 11 with APF of 4 680 TEVAR procedures. In half of the patients, external compression, mostly due to presence of an endoleak, was considered to be the responsible mechanism.

A systematic review including 134 patients showed that emergency TEVAR for ABF can be performed with 93.2% technical success in the short term, and 5.9% 30 day mortality.[126] Aortic related mortality was 14.3% at 17.4 months, and ABF recurrence was 11.1%.[126] Despite these results, the risk of ABF recurrence and infection of the newly inserted EG remains, as the defect in the respiratory system is not treated.[119]

In a systematic review with meta-analysis of patients that underwent TEVAR for AEsF or ABF, 114 patients were included (71 ABF and 43 AEsF). Patients with AEsF presented more frequently with hypovolaemic shock (33% vs. 13%) and systemic infection (36% vs. 9%) than patients with ABF. In hospital mortality was 3% for ABF and 19% for AEsF. Additional procedures after TEVAR and within the first 30 days were performed in 3% of ABF and 37% of patients with AEsF. Therefore, ABF and AEsF are not comparable and should be considered separately.[127]

6.4.2. Open surgical treatment. Open ABF operative mortality ranged from 15% to 41%.[123,128] Open surgical treatment remains the first choice for patients fit for surgery as the EuREC registry showed that a radical surgical approach resulted in significantly better survival (63% at two years) compared with any other treatment strategy.[98]

To decrease the risk of ABF recurrence and re-infection, the bronchial defect needs to be repaired surgically. The defect can be closed primarily or with an intercostal muscle or pericardial flap, but in most cases a bronchial resection and anastomosis or a lung resection (mostly wedge resection) is necessary. After bronchus repair, the EG should be covered with a muscle or pleural flap.[99,123] Sometimes APF can be treated successfully in a more conservative manner by implantation of an EG to control the bleeding, followed by conservative treatment with antimicrobial agents. For persistent infection or recurrent of EG infection, pulmonary resection is indicated.[123]

It is not clear whether the EG always needs to be removed to definitely treat an ABF.[129] In patients fit for surgery, EG removal and *in situ* vascular repair should be considered.[125,126,130]

6.5. Follow up and prognosis

Overall mid term mortality ranges from 14.3% to 75% but largely depends on presentation, the condition of the patient, and the treatment that has been given.[64,66,99,105,126] The presence of an AEsF or ABF is a worsening prognostic factor.

Follow up, including inflammatory parameter monitoring and repeated imaging with CTA and/or 18F-FDG-PET/CT, remains indicated for a longer time to detect delayed recurrent infection and the risk of degeneration of cryopreserved allograft when used.

Recurrence or persistent infection and subsequent multiorgan failure due to septic shock are the main reasons for the demise of these patients.[66] Secondary interventions

due to infective or bleeding complications can be needed in up to 50% of patients.[105]

Recommendation 29		
For patients with aorto-oesophageal fistula complicating thoracic/thoraco-abdominal vascular graft/endograft infection, explantation of the infected material, repair of the oesophagus, and coverage with viable tissue is recommended as definitive treatment.		
Class	Level	References
I	B	Kahlberg *et al.* (2019),[100] Moulakakis *et al.* (2013)[117]

Recommendation 30		
In the emergency setting with active bleeding complicating thoracic/thoraco-abdominal vascular graft/endograft infection with an aorto-oesophageal fistula, initial treatment with an aortic endograft, as a bridge to definitive treatment, should be considered.		
Class	Level	References
IIa	B	Chiesa *et al.* (2010),[99] Chiesa *et al.* (2010),[111] Canaud *et al.* (2014),[125] Canaud *et al.* (2014)[130]

Recommendation 31		
Conservative treatment of patients with an aorto-oesophageal fistula complicating thoracic/thoraco-abdominal vascular graft/endograft infection is not recommended, except in a palliative setting.		
Class	Level	References
III	B	Czerny *et al.* (2014),[97] Chiesa *et al.* (2010),[99] Smeds *et al.* (2016),[110] Chiesa *et al.* (2010)[111]

Recommendation 32		
Treatment of aorto-oesophageal fistula complicating thoracic/thoraco-abdominal vascular graft/endograft infection with an oesophageal endoprosthesis alone is not recommended.		
Class	Level	References
III	C	Czerny *et al.* (2014)[97]

Recommendation 33		
In patients with aortobronchial or aortopulmonary fistula complicating thoracic/thoraco-abdominal vascular graft/endograft infection, closure of the airway defect and explantation of the infected material with *in situ* reconstruction should be considered as definitive treatment.		
Class	Level	References
IIa	C	Czerny *et al.* (2015),[98] Chiesa *et al.* (2010),[99] Chiesa *et al.* (2010)[111]

Recommendation 34
In the emergency setting of active bleeding complicating thoracic/thoraco-abdominal vascular graft/endograft infection with an aortobronchial or aortopulmonary fistula, treatment with an aortic endograft should be considered.

Class	Level	References
IIa	C	Canaud et al. (2013),[123] Canaud et al. (2013)[126]

Recommendation 35
For patients with an aortobronchial or aortopulmonary fistula complicating thoracic/thoraco-abdominal vascular graft/endograft infection, preservation of the endograft may be considered after closure of the airway defect and coverage with viable tissue.

Class	Level	References
IIa	C	Canaud et al. (2013),[123] Canaud et al. (2013)[126]

Recommendation 36
For all patients treated for thoracic/thoraco-abdominal vascular graft/endograft infection, lifelong follow up is recommended because of the risk of recurrent infections or fistulae.

Class	Level	References
I	C	Kahlberg et al. (2017),[64] Spiliotopoulos et al. (2018),[66] Kahlberg et al. (2019),[100] Luehr et al. (2014)[105]

7. ABDOMINAL AORTA

7.1. Specific aspects

7.1.1. Incidence. In a large population of 13 902 patients, the two year rate of abdominal aortic VGI was 0.19% after open surgery *vs.* 0.16% after EVAR, without a significant difference, and 0.2% in both elective and non-elective patients.[53] On the basis of 514 patients, the 30 day incidence was 1.6% (95% CI 0.4% — 2.8%), one year incidence 3.6% (95% CI 1.7% — 5.5%), and two year incidence 4.5% (95% CI 2.4% — 6.6%).[131] EGI is a rare complication, the incidence being < 1%.[132] AEnF was present in 1% — 2% of the reported cases. In an American multicentre registry including 180 infected stent grafts, 43 EGI (23.9%) with an AEnF were reported.[110] In a multicentre study, an AEnF occurred in 32 patients (0.08%).[133] EVAR after anastomotic postsurgical pseudoaneurysm was significantly associated with developing an AEnF.

7.1.2. Clinical presentation. Most patients (70%) with abdominal VGEI have pain, fever, and leucocytosis; 33% of patients suffer weight loss, fatigue, or generalised weakness.[132] In a systematic review of the literature conducted in 2016, 216 studies representing 823 patients with AEnF were compiled.[134] Bleeding was the most common symptom (71.7%), followed by sepsis (39.7%) and haemorrhagic shock (33.1%).

7.1.3. Specific diagnostic modalities. In cases of suspected AEnF or haemorrhage, gastroduodenoscopy and/or colonoscopy are indicated. Polymicrobial Gram negative and enteric species, *Candida* spp., or fungi are highly suggestive of an AEnF.

7.2. Treatment options

7.2.1. Conservative treatment. Conservative treatment, including percutaneous drainage irrigation can be either a preparation for open surgery at a later stage or a definitive palliative strategy in patients unfit for open surgery, owing to comorbidities or their current state of infection (e.g., sepsis). For all conservative strategies, long term or lifelong antimicrobial therapy is important. AEnF or suture line rupture precludes a conservative approach.[135] Conservative management of abdominal aortic VGI is hardly ever possible. Persisting septic complications and rupture are the major drawbacks, with a 30 day mortality rate of 100% in small series.[136]

7.2.1.1. Percutaneous drainage. Fluid collections around the aortic VGEI may be amenable to percutaneous drainage. This minimally invasive intervention potentially reduces the local bacterial and infectious burden, and provides directly obtained specimens for microbiological sampling. Image guided insertion of a pigtail catheter into the perigraft space is performed under local anaesthesia. The catheter is connected to a gravity drainage bag, and repeated saline irrigation prevents occlusion. Catheter removal can be considered once drainage output is low and after imaging confirmation of resolution of the fluid collection. A further benefit of drainage is the possibility of performing sinography to rule out enteric fistula. A 30 day mortality rate of 40% in infrarenal VGEI treated with percutaneous drainage alone has been reported.[137] Its role in the eradication of aortic VGEI remains controversial.

7.2.1.2. Irrigation. Irrigation of the infected perigraft space can be started once a drainage catheter has been inserted. Mainly earlier reports (published before 1997) on the use of povidone iodine solution, antibiotic solution, gentian violet, and saline solution are available. A few case reports with limited follow up data and no comparison of the irrigation solutions address this technique.[138,139]

7.2.2. *In situ* reconstruction. ISR includes complete removal of the infected material with reconstruction in the infected field, and can be done with autologous veins, cryopreserved allografts, rifampicin bonded or silver coated synthetic grafts, and xenogenous grafts.

7.2.2.1. Specific techniques. For treatment, surgeons need to decide for each patient individually. Whatever the type of reconstruction chosen, removal of the infected material is necessary at a certain point. The first step of this intervention consists of securing the aortic clamping zone.[140] For

Table 7. Autologous vein reconstruction of abdominal aortic vascular graft infection: a literature overview

Author	Publication date	Study type	n	Follow up — mo	Early mortality — %	Late mortality — %	Amputation rate — %	Graft occlusion — %	Re-infection — %	Graft rupture — %
Cardozo et al.[267]	2002	Retrospective	12	22	15.3	15.3	16.7	NA	0	NA
Daenens[268]	2003	Prospective database	49	41	8	NA	2	4	0	NA
Beck[269]	2008	Retrospective	240	59	NA	NA	NA	6	NA	NA
Ali et al.[145]	2009	Prospective database	165	32	10	33	7.4	0	5	5
Aavik[270]	2008	Retrospective	11	59	0	0	18.2	9.1	0	NA
Ehsan and Gibbons[223]	2009	Retrospective	46	48	4.3	30	7	9	4	NA
Batt et al.[137]	2012	Retrospective	6	41	NA	NA	0	0	16	NA
Dorweiler et al.[142]	2014	Retrospective	67	60	9	55	6	3	0	NA
Charlton-Ouw et al.[177]	2015	Retrospective	11	42	0	9	27	NA	14	NA
Heinola et al.[143]	2016	Retrospective	55	32	9	40	7	3.6	4	5

NA = not available.

bleeding with haemodynamic instability, some authors have proposed the use of an intra-aortic balloon inserted via a femoral approach.[141]

7.2.2.2. Graft materials.

7.2.2.2.1. Reconstruction with autologous vein. Autologous veins have the lowest infection rates (0% — 6%) and low graft thrombosis rates (Table 7). Disadvantages include harvesting difficulties in the emergency setting and a longer operation time so that it may not be tolerated by elderly patients with comorbidities. Previous deep vein thrombosis is a contraindication.[142,143] Harvesting veins is associated with venous morbidity with chronic venous insufficiency reported in up to 15%. Deep vein thrombosis occurred in up to 22%.[142,143] Venous grafts might be effective for highly virulent pathogens, but their use in multiresistant strains remains unclear.[17,142] There are reports of poor outcomes in the presence of Gram negative micro-organisms, MRSA, or *Candida* spp., especially when concomitant sepsis or AEnF exists.[144,145]

7.2.2.2.2. Cryopreserved allografts. A number of studies have employed cryopreserved allografts for ISR of abdominal aortic VEGI.[146—153] Cryopreserved allografts have shown low re-infection rates (0% — 7%). Their limited availability, and rate of graft related complications resulting from allograft degradation (up to 21%), including aneurysm, dilatation, and graft rupture, are the main drawbacks.[146—153] Allograft related re-intervention rates were found in up to 55% of patients after five years (Table 8).[147] However, a better outcome with no dilatation or aneurysm during a mean follow up of five years was observed in another series of 71 patients.[154] Survival rates varied between 40% and 54% after five years.[147,154]

Graft specific factors such as allograft age, pre-treatment, and cryopreservation were of no prognostic value in multivariable analysis for ISR with cryopreserved allografts.[150] There is no evidence that post-operative immunosuppressive therapy would prevent rejection in patients with cryopreserved allografts.

7.2.2.2.3. Rifampicin bonded grafts. Rifampicin bonded grafts can be used in less virulent and low grade infections, especially if these infections are caused by *S. aureus* or coagulase negative staphylococci. The re-infection rate is 11.5% (range 0% — 18%), but the amputation rate is low (Table 9).[17,144,155—158] Experimentally, there is weak activity against non-fermenter Gram negative bacilli, such as *Pseudomonas* species.[159,160] In retrospective studies, they are less effective in infections caused by MRSA, Gram negative strains, and fungi.[156,157] The antimicrobial efficiency of rifampicin bonded grafts is concentration and time dependent. Owing to dilution, the protective effect is reduced after approximately one week.[157,159] The rifampicin bonded concentration currently used in the treatment solution is heterogeneous, making the prediction of outcomes impossible. Concentrations of 1 — 60 mg/mL, with a maximum dose of 600 mg (soaked for 15 — 30 min) have been used in clinical and *in vitro* studies.[140,144,155,157,159] Development of rifampicin resistance might occur.[157] Their major advantage is their off the shelf availability in the emergency setting.

7.2.2.2.4. Silver coated grafts. Silver coated grafts are available in two different options, silver acetate, which dissolves within two to four weeks, and elemental silver, which remains for about one year.[161,162] The advantage of silver is its wide antimicrobial activity and paucity of resistance development in experimental studies. But in clinical trials for ISR, the re-infection rate of silver grafts is 11% (range 0% — 16%) within 2.5 years. Low amputation rates and high patency rates are reported (Table 10).[17,163] There is no comparison between different silver coatings. Only one study (n = 10 patients) has reported the effect of elemental silver graft for ISR in the aorto-iliac position, and describing one re-infection.[164]

Table 8. *In situ* reconstruction with cryopreserved allografts for abdominal aortic vascular graft infection: an overview

Author	Publication date	Study type	n	Follow up — mo	Early mortality — %	Late mortality — %	Graft related complications					
							Total — %	Re-infection — %	Aneurysm dilatation — %	Rupture — %	Thrombotic occlusion — %	Amputation — %
Nevelsteen et al.[271]	1998	Retrospective	30	25	27	10	NA	7	13	3	23	6.7
Lesèche et al.[185]	2001	Retrospective	28	35.4	17.8	17.8	17	0	11	0	9	0
Verhelst et al.[227]	2000	Retrospective	90	36	17	16	21.1	1	7.8	8.8	10	1
Vogt et al.[272]	2002	Retrospective	49	27	6	20	16.3	0	0	8.1	2	0
Noel et al.[273]	2002	Registry database	56	5.3	13	25	25	0	2	9	9	5
Kieffer et al.[102]	2003	Retrospective	68	34	30	28	20.3	7	NA	0	29.7	0
Bisdas et al.[151]	2010	Retrospective	57	36	9	36	5.3	0	1.8	1.8	1.8	NA
McCready et al.[228]	2011	Retrospective	26	46.6	31	62	19	0	0	0	0	NA
Garot et al.[152]	2014	Retrospective	18	12	48	48	NA	0	NA	NA	23	NA
Touma et al.[150]	2014	Retrospective	54	12	28	39	19	3.7	1.9	11.1	11.1	1.9
Harlander-Locke et al.[148]	2014	Retrospective	220	30	9	49	24	3.6	2.7	5.5	4.1	NA
Minga Lowampa et al.[229]	2016	Retrospective	96	49	8	85	29	0	9.4	7	11.7	NA
Heo et al.[149]	2017	Retrospective	25	19	8	28	12	0	4	4	4	NA
Batt et al.[198]	2011	Retrospective	21	41	45.5	72.7	NA	14.2	NA	NA	18.2	0
Lejay et al.[147]	2018	Retrospective	25	47	24	40	55	NA	20	24	NA	NA
Ben Ahmed et al.[154]	2018	Retrospective	71	45	2.8	2.8	18.3	4	0	2	7	1.4

NA = not available.

Table 9. *In situ* reconstruction with rifampicin bonded grafts for abdominal aortic vascular graft infection: an overview

Author	Publication date	Study type	n	Rifampicin dose — mg/ml	Follow up — mo	Early mortality — %	Late mortality — %	Amputation rate — %	Graft occlusion — %	Re-infection — %
Torsello et al.[158]	1997	Retrospective	11	60	33	18	18	0	NA	9
Hayes et al.[156]	1999	Retrospective	11	45–60	12	18.2	36.4	0	18.2	0
Young et al.[155]	1999	Retrospective	25	1	36	8	24	0	14	4
Bandyk et al.[157]	2001	Retrospective	19	45–60	17	9.1	NA	0	0	8
Oderich et al.[144]	2006	Retrospective	52	2.4	41	8	16	0	8.8	11.5
Batt et al.[198]	2011	Retrospective	8	NA	41	31.8	40	NA	0	0
Schaefers et al.[274]	2018	Retrospective	10	NA	27	0	0	NA	NA	0

NA = not available.

Table 10. *In situ* reconstruction with silver coated grafts for abdominal aortic vascular graft infection: an overview

Author	Publication date	Study type	Graft type	n	Follow up — mo	Early mortality — %	Late mortality — %	Amputation rate — %	Graft occlusion — %	Reinfection — %
Batt et al.[275]	2003	Prospective	Silver acetate	24	17	16.6	16.6	0	0	0
Mirzaie et al.[163]	2007	Prospective	Silver acetate	11	30	0	0	0	0	0
Batt et al.[276]	2008	Prospective	Silver acetate	24	32.5	20.8	25	4	8.3	12.5
Pupka et al.[277]	2011	Prospective	Silver acetate	27	22.8	11	NA	0	0	16
Bisdas et al.[151]	2010	Retrospective	Silver acetate	11	24	18	27	0	0	9
Zegelman et al.[164]	2013	Retrospective	Metallic silver	10	18	NA	NA	NA	NA	10

NA = not available.

7.2.2.2.5. Xenogenous grafts. Bovine pericardium is an off the shelf solution and can be tailored to form a tube or bifurcated graft. There are a few case series involving thoracic, thoraco-abdominal, and abdominal reconstructions. Re-infection rates were described in up to 16% in small series; other studies reported 100% freedom from re-infection or re-intervention in follow ups of up to four years involving thoracic, thoraco-abdominal, and abdominal reconstructions.[113–115,165–168] The largest series included 35 patients and four years of follow up, and reported only seven patients who got bovine pericardium for abdominal aortic VGIs. Four of the seven died within 30 days owing to ongoing sepsis and multi-organ failure. Three were free of re-infection, without signs of degeneration. Further studies with longer follow ups are mandatory. Their role in case of sepsis remains unclear.

There is little experience with the use of ovine, mesh reinforced biosynthetic prosthesis in ISR for abdominal aortic VGIs (n = 13, follow up 11 – 26 months), with a trend to low infection rates, but only case series have been reported.[169,170] Consequently, no recommendation can be made.

7.2.2.3. Adjunctive therapies. During ISR, the proximal anastomosis can be re-inforced with fascia.[143,171] After inserting the new VG, it should be covered by biological material, especially pediculed omentum flap, muscle, fascia, or retroperitoneal tissue.[171] An omental wrap around the graft lowers the rate of infection, even when rifampicin or silver coated VGs are used.[144,155] There are insufficient data on the efficacy of bio-absorbable and non-absorbable local antibiotic beads or sponges, and reported data have described their use in an extracavity position only.[172,173] If the groin is involved, the distal anastomosis can be covered by a muscle flap.[142,143]

The use of NPWT in abdominal aortic VGI has only been described in five patients with aorto-iliac VGI treated via a retroperitoneal approach with radical debridement and placement of the foam directly on the graft. There was no 30 day mortality and no one year re-infection. The technique requires repeated dressing changes every three to five days, but long term data are not available.[174]

7.2.3. Extra-anatomic reconstruction. Axillobifemoral or axillobipopliteal bypasses through a non-infected field were performed to restore lower limb perfusion following excision of an infected graft. The disadvantages of axillobifemoral reconstructions are low patency rates (64% – 75% at five years) and high amputation rates (up to 11% at five years). Stump rupture with life threatening haemorrhage can occur in up to 27% of reported cases (Table 11).[137] Finally, re-infection rates after extra-anatomic bypasses were 0% – 15%[144,175,176] and up to 27% in small series.[137,177] A 30 day mortality rate of 18% (range 11% – 45%) and a late mortality of 32% (range 22% – 44% within 47 months) have been described (Table 11).[137,138,175,177,178] Compromised colonic and pelvic blood supply due to insufficient internal iliac and inferior mesenteric artery perfusion might also lead to complications.

Axillofemoral and axillopopliteal bypass patency rates were lower in patients with concomitant occlusive disease (primary patency of 71% vs. 73%, secondary patency rates of 79% vs. 92% after five years).[175,179] The outcome for axillopopliteal bypasses in abdominal aortic VGEI is poor, with a reported primary patency of 0% at 7 months, and a secondary patency 53% at 17 months and 27% at 50 months in a series of five axillopopliteal bypasses performed for abdominal aortic VGEI.[175]

7.2.3.1. Two stage procedure. EARs can be performed directly after bypass removal for reconstruction of the lower extremities or as staged procedure. A two stage procedure one to five days before removal of the infected graft should be considered in haemodynamically stable patients because of the reduction of operative metabolic (acidosis) and haemodynamic stress.[175,179,180] Reductions in the mortality and amputation rates were found when staged procedures were performed.[181] Competitive flow has never been reported to be the causative factor of graft occlusions, but no specific studies have been performed.

7.2.3.2. Stump management. Risk of stump rupture is related to the mechanical consequences of too short a stump and to the properties of the persistent infected

Table 11. Extra-anatomic reconstructions for abdominal aortic vascular graft infection: literature overview

Author	Publication date	Study type	n	Follow up – mo	Early mortality – %	Late mortality – %	Amputation rate – %	Graft occlusion – %	Re-infection – %	Stump rupture – %
Seeger et al.[175]	2000	Prospective	36	32	11	44	11	31	3	3
Ohta et al.[138]	2001	Retrospective	9	47	11	22	NA	33	0	0
Bandyk et al.[157]	2001	Retrospective	19	17	9.1	NA	11	34.4	2.8	NA
Hart et al.[184]	2005	Retrospective	15	15	40	45	6	NA	13	7
Armstrong et al.[216]	2005	Retrospective	25	51	21	NA	6.9	NA	13.8	4
Oderich et al.[144]	2006	Retrospective	43	41	11.6	NA	9	37.2	11.6	9
Brown et al.[176]	2009	Retrospective	18	14	22	44	6	11	0	0
Batt et al.[198]	2011	Retrospective	11	41	45	NA	27	27	27	27
Charlton-ouw et al.[177]	2015	Retrospective	5	72	20	40	0	NA	20	20
Yamanaka et al.[183]	2014	Retrospective	7	27	NA	NA	6.9	NA	13.8	NA

NA = not available.

tissue after incomplete debridement. If the stump is too short, transposition or renal and visceral artery bypass is likely to be necessary and should be performed first to minimise the visceral ischaemic time. Techniques to prevent stump rupture include double suture layers, reinforcement with venous or felt pledgets, prevertebral fascia, or a layer of posterior rectus fascia peritoneum. Most authors recommend covering the stump with omentum or an omental wrap.[144,178,182] Another technique is a pedicled latissimus dorsi flap.[183]

7.2.4. Systematic reviews and meta-analyses on *in situ* reconstruction, extra-anatomic reconstruction, and graft materials. Two meta-analyses (37 studies, 1 417 patients;[180] 36 studies, 1 464 patients[17]) showed significantly better outcomes for ISR than EAR with overall lower complication, re-infection, and mortality rates.

There was no significant difference in the re-infection rate between the different grafts (veins 2%; cryopreserved allografts 9%, rifampicin bonded or silver coated prosthesis 11%), but they all demonstrated significantly better results than standard ePTFE and PET grafts for ISR. Veins and silver coated prostheses had significantly lower graft occlusion rates (2% and 7%, respectively) than cryopreserved allografts (13%) or rifampicin bonded prostheses (11%). The amputation rate was significantly lower in cryopreserved allografts (3%), and rifampicin (3%) or silver coated grafts (4%) than veins (9%). Standard ePTFE and PETgrafts gave significantly worse results for graft occlusion and amputation. Meta-regression analysis preferred veins for ISR neo-aorto-iliac reconstruction, especially in younger patients, except in the presence of AEnF, while autologous veins provided the best outcomes with virulent infecting organisms.

For abdominal aortic VGEI without AEnF, the data suggest that ISR with autologous veins, cryopreserved allografts, silver coated grafts, or rifampicin bonded grafts should be considered. Long term follow up is missing for bovine pericardium. Even though there is no ideal graft material, most authors prefer a biological material conduit for ISR. Veins might be preferable in young patients with a longer life expectancy. Rifampicin soaked grafts or silver grafts have been shown effective, especially in low virulence infections. For large perigraft abscesses and MRSA infections, EAR and removal of the infected graft may be considered.[17,146,151,176,177,180]

7.2.5. Partial or total excision of the material. Total excision of the infected graft is mandatory to control VGI.[148] There are some inconclusive reports of partial graft resection in high risk patients, when complete graft excision was not possible.[184] In retrospective studies, successful treatments were reported, when infection was limited to the groin and supra-inguinal VGs were incorporated. Retrospective studies reported no re-infection after 2.5 years in 11 patients after interposition of unilateral silver coated grafts,[163] no re-infection after partial interposition with

cryopreserved allografts in 10 patients after three years,[185] and a 9.5% re-infection rate after partial resection with autologous vein interposition in 20 patients after 72 months.[186] Limited resection of only infected material and reconstruction by obturator canal bypass ($n = 18$) or part of a bifurcated graft ($n = 34$) was also proposed with re-infection rates of 6% and 0%, respectively, and no amputation at 36 months.[187] Other series reported a 47% ($n = 7/15$ patients) infection rate of the contralateral limb after 39 months, when partial resection of an aortobifemoral VGI and EAR was performed.[188] Overall mortality was 40% and similar between patients with or without recurrent graft infections.

As an individual decision, partial resection, and ISR or an extra-anatomic route might be an option. Successful treatment was based on low virulence organisms, especially monomicrobial determination of multidrug sensitive streptococci species and *S. aureus*. If polymicrobial, fungal or Gram negative organisms were present, total excision of the infected graft was recommended.[163,186–188]

7.2.6. Endograft infection. Eradication of infection by removal of the infected EG and ISR with biological material has the lowest re-infection rate ($< 10\%$) and is recommended for patients who are fit for surgery.[132] Reported 30 day mortality rates have been 8% − 39% and amputation rates 0% − 2%.[189–191] In the case of EAR, stump management may be more difficult because clearly the neck can be damaged by the extraction owing to the presence of suprarenal hooks promoting ligation very proximal to the renal artery ostia. If the patient is unfit for surgery, conservative treatment with lifelong antibiotics or drainage of infection without removal of the EG must be considered. The mortality of patients managed by conservative treatment with antimicrobial therapy alone is high, reported as 63.3% at 30 days.[132]

7.3. Graft enteric fistula

Fistulisation can occur early after bowel injury during the surgical dissection or poor tunnelling, or as a consequence of a suture line pseudo-aneurysm or as a consequence of prior graft infection.[192]

7.3.1. Specific treatment modalities. Depending on the location of the AEnF, securing the aortic clamping zone can be obtained more proximally using a supracoeliac approach.[140,192] Whenever possible, it is advised to avoid entering the enteric communication during the dissection and to leave a "patch" of the old graft attached to the intestine to avoid enteric content spillage.[140]

7.3.2. Stent grafts as a temporary or a definitive solution. In the last decade, several studies have reported the use of stent grafts to treat AEnF. In a retrospective comparative, multicentre study no post-operative mortality in patients treated with EG *vs.* VG, a mortality of 35% in patients treated with VG was reported.[193] Late sepsis occurs

significantly more often after endovascular surgery than after open surgery (42% *vs.* 19% at two years). In particular, the presence of pre-operative sepsis was found to be a factor indicative of an unfavourable outcome.[193] As a consequence, some authors have proposed the temporary use of EG as a "bridge technique" followed by EG extraction and *in situ* repair, or even as a definitive treatment in select cases of bleeding without clear sepsis.[194—198]

7.3.3. *In situ* reconstruction or extra-anatomic reconstruction.
A systematic review based on 1 467 patients concluded that axillobifemoral bypass and abdominal aortic VG removal in a staged procedure was the best therapeutic option, with a mortality of 31% *vs.* 51% in a non-staged procedure and 47% in the case of ISR.[199] No difference in re-infection rate between EAR and ISR was found.[198] In both cases, PET VGs were used. These findings contrast with other findings that report ISR as an independent factor for survival in a multivariable analysis.[200]

7.3.4. Total or partial graft excision.
In a series of 54 patients treated for a secondary AEnF, partial graft excision led to a 4% re-infection rate with a mean follow up of 51 months.[140] In several studies, no difference in outcome was seen between patients with partial resection and complete resection.[146,198] The only factor influencing the re-infection rate (47%) was the length of follow up, led to the conclusion that lifetime follow up was mandatory.[198] Mortality after re-infection was 100%.

7.3.5. Bowel repair
7.3.5.1. Direct suture vs. complex bowel reconstruction.
The type of the bowel repair depends on the size and the location of the defect.[140,192,201] A tension free duodenorrhaphy with direct suture of the duodenum can be performed if the bowel defect is small, avoiding an extensive approach, but a complex duodenal reconstruction with resection anastomosis and re-routing decreases the risk of recurrent infection.[200]

7.3.5.2. Omental interposition.
Pedicle omental flap can be transferred in order to cover the reconstruction and separate the new vascular reconstruction from the bowel. It is recommended when feasible as it has been shown to be the strongest predicitive factor for survival.[140,192,200,201]

7.4. Visceral artery revascularisation infection
Although the nature of the bypass suggests that visceral bypasses are frequently involved with VGEI, a systematic literature search yielded only two case reports that specifically deal with the treatment of an infected mesenteric bypass.[202,203]

Owing to the optimisation of endovascular possibilities, the treatment of mesenteric occlusive disease has evolved towards an endovascular first approach whereas in the recent past this was not the case. In some cases of infected mesenteric bypasses, an initial endovascular recanalisation of the native vessel followed by removal of the infected graft material and closure of the anastomotic site with an autologous vein patch might be possible.[202]

7.5. Ureteral fistula
Arterio-ureteral fistulae (AUFs) occur in < 1% of patients after aorto-iliac surgery and are almost entirely related to VGIs. A pseudo- or true common or external iliac artery aneurysm with fever, pain, or swelling due to abscess can be found in 38% of cases.[204] Recurrent urinary tract infections were concomitant findings in 40% and urinary outflow obstruction with hydronephrosis in 70%.[205—207]

For acute haemorrhage, stent graft insertion as a bridge might be proposed.[206] For definitive healing, open surgery with VG or EG explantation and AUF resection is mandatory. VG or EG removal and EAR with femorofemoral crossover bypass and ligation of the iliac artery should be considered.[205—208] In ISR with cryopreserved allografts, silver coated or rifampicin bonded VG were reported, but there are no available data concerning morbidity or mortality rates. Primary ureteric repair, ligation (with or without nephrectomy), re-implantation to a site away from the vessels, or diversion of the urine with a nephrostomy tube may be necessary. Nephrectomy has to be considered if irreversible damage to the renal collecting system has occurred.[205—208] An indwelling ureteral stent should be removed owing to the risk of recurrent fistulisation or re-infection.[207]

7.6. Follow up and prognosis
Abdominal aortic VGEI are some of the most challenging and highly morbid conditions to treat, with an early mortality rate ranging from 16% to 22%, an amputation rate ranging from 3% to 18%, and a re-infection rate of 6% — 20%.[17,137] However, late mortality is not assessable owing to insufficient data, a wide range of follow up, and the use of different types of conduit. Because it is a rare condition, studies include all kind of abdomino-iliac or aortofemoral graft reconstruction. Therefore, there is very limited evidence, with no comparative studies at all and with most published series burdened by seletion bias, indicating that individual surgical decisions for treatment are needed (Fig. 6).

Management and treatment choice must be performed within a multidisciplinary team, including vascular surgeons, visceral surgeons, infection disease specialists, radiologists, and anaesthetists.

Mortality rates relate to: (i) the patient's condition — an American Society of Anaesthesiology score 4 and sepsis are independant risk factors for mortality,[145,154] as well as individual local presentation of infection affect the outcome; (ii) the emergency or elective setting — there is higher 30 day mortality in emergency settings;[146] (iii) the presence of an AEnF — studies that included AEnF resulted in worse outcome;[198] (iv) virulence of the micro-organisms — operative mortality rate was higher in abdominal aortic VEGI caused by virulent micro-organisms.[17,144,145,156,157,180] MRSA and multiresistant strains are associated with poor outcome.[17,137,144,156,157,180]

Post-operative duration of antibiotics and the variety of bacterial specimens are quite different. The number of studies and wide range of included patients ($n = 5—220$) might not adequately represent diverse patterns of clinical manifestation, specific patient subgroups or the experience
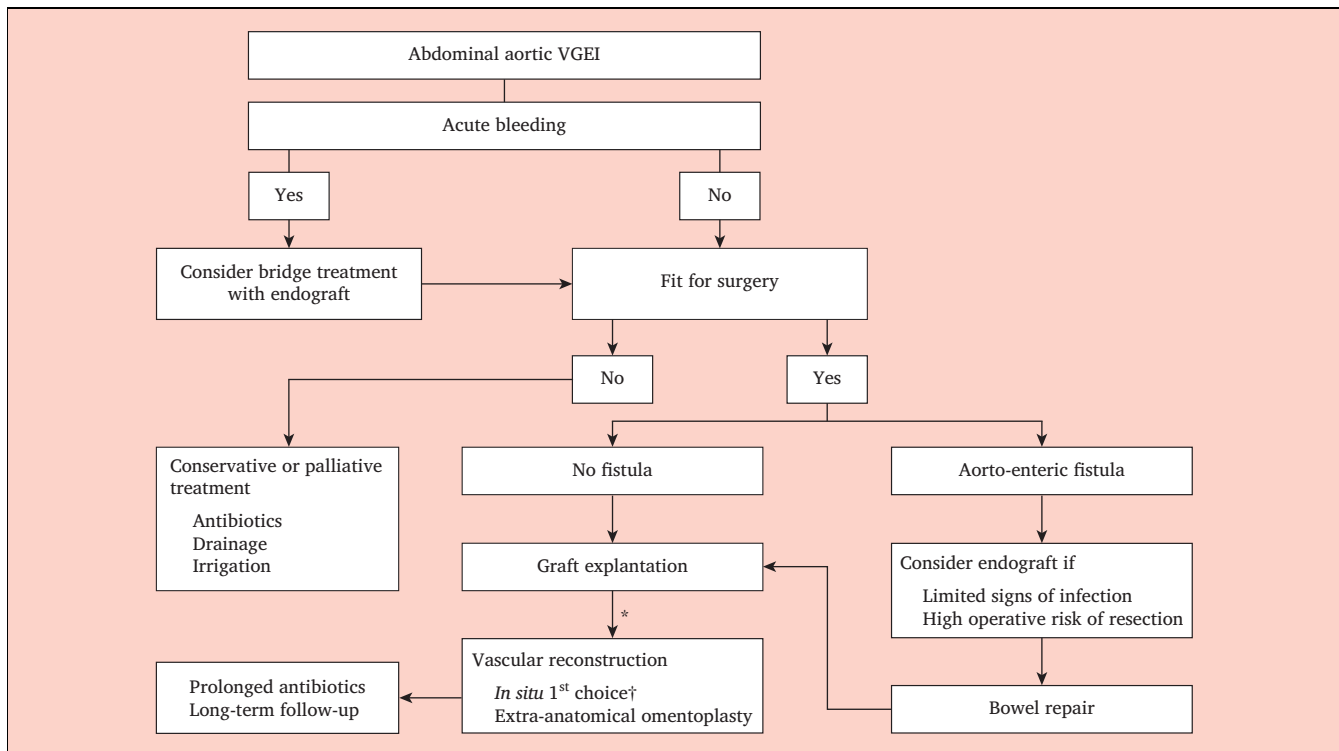
Figure 6. Proposed algorithm for the management of aortic vascular graft/endograft infection. * In a single or staged prodecure. † Materials that can be used are deep femoral veins, cryoprerved allografts, silver grafts or rifampicin impregnated grafts. VGEI = vascular graft/endograft infection.

of each centre because of the low numbers of patients treated annually.

Post-operative follow up is important. Close surveillance, including laboratory test and US is mandatory every three to six months for a minimum of one year, and later lifelong every 6 – 12 months is reasonable. If clinical signs, laboratory tests or US are suspicious, CT is recommended. Special attention should be paid if ISR with allografts was performed.

Recommendation 37

Percutaneous drainage of peri-graft fluid with or without irrigation may be considered for microbiological identification and to reduce the bacteriological burden, but not as ultimate treatment in abdominal aortic graft/endograft infection.

Class	Level	References
IIb	C	Calligaro et al. (2003),[135] Batt et al. (2012),[137] Ohta et al. (2001),[138] Igari et al. (2014)[139]

Recommendation 38

For fit patients with an abdominal aortic vascular graft/endograft infection, complete excision of all graft material and infected tissue is recommended for definitive treatment.

Class	Level	References
I	B	Batt et al. (2018),[17] O'Connor et al. (2006)[180]

Recommendation 39

For patients with an abdominal aortic vascular graft/endograft infection, *in situ* reconstruction with autologous vein should be considered as the preferred method.

Class	Level	References
IIa	C	Batt et al. (2018),[17] Spiliotopoulos et al. (2018),[66] Dorigo et al. (2003),[69] Dorweiler et al. (2014),[142] Heinola et al. (2016),[143] Ali et al. (2009),[145] Harlander-Locke et al. (2014),[148] O'Connor et al. (2006),[180] Rodrigues dos Santos et al. (2014)[200]

Recommendation 40

For patients with abdominal aortic vascular graft/endograft infection, cryopreserved allografts, silver coated grafts, rifampicin bonded polyester grafts, or bovine pericardium should be considered as alternative solutions.

Class	Level	References
IIa	C	Batt et al. (2018),[17] Spiliotopoulos et al. (2018),[66] Dorigo et al. (2003),[69] Dorweiler et al. (2014),[142] Heinola et al. (2016),[143] Ali et al. (2009),[145] Harlander-Locke et al. (2014),[148] O'Connor et al. (2006),[180] Rodrigues dos Santos et al. (2014)[200]

Recommendation 41

Partial excision of infected an aortic vascular graft/endograft may be considered when infection is documented as limited and the remaining material is well incorporated.

Class	Level	References
IIb	C	Mirzaie *et al.* (2007),[163] Simmons *et al.* (2017),[186] Phang *et al.* (2019)[187]

Recommendation 42

For patients with abdominal aortic vascular graft/endograft infection and a large abscess or multiresistant micro-organisms, extra-anatomic reconstruction may be considered.

Class	Level	References
IIb	B	Oderich *et al.* (2006)[144]

Recommendation 43

Lifelong imaging follow up is recommended after *in situ* reconstruction with cryopreserved allografts for abdominal aortic vascular graft/endograft infection, in order to detect allograft degeneration.

Class	Level	References
I	C	Lejay *et al.* (2017),[147] Harlander-Locke *et al.* (2014),[148] Touma *et al.* (2014),[150] Kieffer *et al.* (2004),[153]

Recommendation 44

Securing a supracoeliac clamp zone or using an aortic occlusion balloon may be considered as the first step before entering the aorto-enteric fistula area.

Class	Level	References
IIb	C	Miyamoto *et al.* (2016),[141] Schoell *et al.* (2015)[192]

Recommendation 45

In the emergency setting of active bleeding complicating abdominal aortic graft/endograft infection with or without aorto-enteric fistula, initial treatment with an endograft should be considered, but only as a temporary measure.

Class	Level	References
IIa	C	Chick *et al.* (2017),[197] Batt *et al.* (2011)[198]

Recommendation 46

In surgical repair of aortic abdominal graft/endograft infection with aorto-enteric fistula, omentoplasty or transfer of autologous vascularised tissue to cover the vascular reconstruction is recommended.

Class	Level	References
I	B	Rodrigues dos Santos *et al.* (2014)[200]

Recommendation 47

In the emergency setting of active bleeding complicating abdominal aortic graft/endograft infection with an arterio-ureteral fistula, initial treatment with an endograft may be considered, but only as a temporary measure.

Class	Level	References
IIb	C	van den Bergh *et al.* (2009),[204] Das *et al.* (2016),[205] Malgor *et al.* (2012),[206] Madoff *et al.* (2004),[207] Pillai *et al.* (2015)[208]

Recommendation 48

For patients with an arterio-ureteral fistula and vascular graft/endograft infection, complete explantation of the graft combined with urological treatment with or without *in situ* arterial reconstruction should be considered.

Class	Level	References
IIa	C	van den Bergh *et al.* (2009),[204] Das *et al.* (2016),[205] Malgor *et al.* (2012),[206] Madoff *et al.* (2004),[207] Pillai *et al.* (2015)[208]

8. PERIPHERAL ARTERIES

8.1. Specific aspects

8.1.1. Incidence and risk factors. VGI has been reported in up to 2.5% in femorofemoral prosthetic bypasses,[209] and in up to 2.8% in femoropopliteal prosthetic bypasses.[210] Higher incidences have been reported in series where 90% of the indication had been critical limb threatening ischaemia. In a retrospective review of 141 grafts involving the femoral artery, VGI occurred in 25 (18%) and occurred most frequently after major amputation (41% *vs.* 6%) or early re-intervention after initial grafting (70% *vs.* 16%). Risk of VGI after amputation was highest when amputation was performed less than four weeks after the bypass (70% *vs.* 32% when amputation was done more than four weeks after the bypass).[211]

The differentiation of deep wound infection and VGI is unclear in many reports. SSI is the strongest risk factor for VGI. Risk factors for SSI must be known, but it should always keep in mind that these are not strictly correlated with VGI (Table 12).

Peripheral arteries are a major site for stent implantation. One systematic review identified 48 bare metal stent infections outside the coronary arteries.[212] Emergency prior procedures with adjunctive thrombolysis represented almost 20% of the infected non-cardiac cases and were considered as risk factors for infection.

8.1.2. Clinical presentation. The most common site of VGEI after aorto-iliac or infra-inguinal reconstructions is the groin. Clinical presentation can include fever, pain, mass, or redness of the skin. This can be explained not only by the specific risk of contamination of the groin or by the

poor healing of the wound, but also because this approach is frequently performed in patients with infected distal wounds. Wound complications following vascular procedures involving the groin have been reported in up to 20% of patients.[213]

8.2. Treatment options

8.2.1. Conservative treatment.
Conservative treatment of lower limb VGI without removal of the infected VG is rarely an option because it is associated with a high mortality (up to 45% at five years),[214] persistent infection, anastomotic disruption, or active bleeding. However, if the infected VG is not removed, the risk of persistent infection, anastomotic disruption, or active bleeding must be anticipated.

8.2.1.1. Negative pressure wound therapy.
NPWT is an option to manage VGI in order to promote wound healing after debridement of the infected tissue.[215] Even though −125 mmHg has been shown to allow faster granulation and faster healing, lower pressure is recommended if the NPWT is in contact with the graft in order to avoid the bleeding when higher pressure is employed.[215] NPWT may lead to serious bleeding complications in up to 10% of patients and eradication of infection is uncertain.[216–220]

In a retrospective series of 68 patients with 72 deep inguinal infections below the inguinal ligament and with a blood culture negative for septicaemia, septic anastomotic involvement, or bleeding, 85% ($n = 61/72$) of the grafts were in place nine months after initiation of treatment.[215] Another retrospective study of 161 patients treated by NPWT showed that major bleeding occurred in 7.1% and re-infection after complete wound healing in 6.4%. The cumulative estimated proportion of completely healed wounds was 70% after 80 days. Independent risk factors for failure of wound treatment were synthetic graft infection (OR 6.1, 95% CI 2.6 − 14.2) and bleeding or pseudoaneurysm as presenting symptom (OR 2.9, 95% CI 1.0 − 8.2).[218]

The EndoVAC technique can be an option for bleeding or high risk of bleeding.[84,85] The principle in this hybrid technique is to use endograft to control or prevent bleeding and NWPT for secondary wound healing, but so far the number of patients treated is too low to give any recommendations on the use of this technique.

8.2.1.2. Irrigation.
The aim of irrigation is to reduce bacterial colonisation of the infected VG. Irrigation is mostly established in the groin, through drainage provided by a tube placed in the infected cavity after wound debridement. Irrigation may be considered only in patients in whom removal of the infected prosthetic material is not possible.[213]

8.2.2. In situ reconstruction

8.2.2.1. Specific technical aspects.
If the original indication was claudication, immediate revascularisation may not be necessary. In these cases, arteriotomies performed for VG removal must be closed with biological substitutes, such as saphenous vein or bovine pericardial patch. A secondary bypass may be performed later after total eradication of the infection. Conversely if the original indication was chronic limb threatening ischaemia (Rutherford 4 − 6) and if the leg presents with severe ischaemia after VG removal, immediate redo bypass is mandatory to avoid major amputation. Revascularisation is also needed when the arterial disease has progressed significantly or infected graft removal is not possible without sacrificing other crucial vessels.

8.2.2.2. Graft materials.

8.2.2.2.1. Reconstruction with autologous material.
The great saphenous vein (GSV) and other superficial venous materials is preferred when available, as such material is easily harvested.[221,222]

Autologous femoral vein has also been reported to be useful in treating peripheral graft infections with a low rate of recurrent infection, if the diameter of the femoral vein fits the inflow and outflow arteries.[223]

ISR, preferably with autologous vein, has gained popularity as it is less invasive than extra-anatomic

Table 12. Patient related independent risk factors for surgical site infections of the lower limbs

Risk factor	Adjusted risk estimation OR (95% CI)	Reference
Female sex	1.44 (1.28−1.63)	Greenblatt et al. (2011)[278]
Obesity	2.08 (1.78−2.43)	Greenblatt et al. (2011)[278]
Body mass index >25 kg/m^2	1.78 (1.23−2.57)	Davis et al. (2017)[279]
Chronic obstructive pulmonary disease	4.29 (2.74−6.72)	Davis et al. (2017)[279]
Dialysis	2.10 (1.07−4.09)	Leekha et al. (2016)[280]
	1.51 (1.08−1.44)	Greenblatt et al. (2011)[278]
Critical limb ischaemia	4.35 (3.45−5.47)	Davis et al. (2017)[279]
Re-intervention	2.91 (1.61−5.27)	Leekha et al. (2016)[280]
Blood glucose >11 mmol/L	2.68 (1.38−5.22)	Leekha et al. (2016)[280]
Hyponatraemia <134 g/dL	11.0 (1.9−63)	Brothers et al. (2009)[211]
Post-operative immobilisation	1.20 (1.02−1.42)	Greenblatt et al. (2011)[278]
Major amputation	12 (4.1−34)	Brothers et al. (2009)[211]
Prior revascularisation	2.68 (1.38−5.22)	Leekha et al. (2016)[280]

OR = odds ratio; CI = confidence interval.

bypass.[14,224,225] If autologous vein is not available, there remains controversy over which is the optimal ISR graft following excision of the infected graft.

8.2.2.2.2. Cryopreserved allografts. The re-infection rate of ISR with cryopreserved allograft is lower than with prosthetic grafts, and the amputation rate is lower than with the removal of the infected graft without reconstruction.[147,223,226–228] However, graft degeneration may lead to serious complications in the long term. In a retrospective analysis of 53 patients treated for VGI with cryopreserved arterial allograft, the graft related re-intervention rate at five years was 33% in patients who underwent peripheral reconstruction (two allograft ruptures, seven aneurysmal degenerations). Allograft related complications, such as allograft thrombosis, anastomotic pseudo-aneurysm, degenerative aneurysm, and allograft disruption leading to bleeding have been reported in 29% of patients.[229] Cryopreserved GSV has the advantage of a better size match in the infrapopliteal segments and crural vessels and longer segments may be available than arteries; however, patency is limited (around 35% at one year).[230] Primary and secondary patency rates of 56% and 73% have been reported at one year, respectively, and 17% and 38%, respectively, at five years.[231]

8.2.2.2.3. Prosthetic grafts. The advantages of prostheses are that they are readily available and operating time is shorter, but the re-infection rate is high vs. non-prosthetic solutions. Silver impregnated or rifampicin soaked PET grafts have been used for ISR following removal of the infected graft, but the level of evidence is low and most of the studies have been on the treatment of aortic infection.[232,233] In a study of 24 femoropopliteal bypasses replaced with silver impregnated PET grafts, the overall re-infection rate was 19%.[234] When rifampicin soaked grafts are used, the results of wound cultures (when available) should be considered, as data have shown that the micro-organism causing the infection was resistant or not susceptible to rifampicin in 31% of cases.[235]

A biosynthetic collagen prosthesis (Omniflow II; LeMaitre, Burlington, MA, USA) has also been tested in the presence of graft infection. In a series of nine consecutive patients who underwent replacement of an infected peripheral graft with a biosynthetic prosthesis, the early morbidity rate was 56% (n = 5), with no in hospital death. Four patients had post-operative wound healing problems treated conservatively and one patient had acute early graft occlusion leading to major amputation. Two patients had late graft occlusion six and 12 months after the procedure, respectively. FDG-PET was performed in six patients after a median follow up of 19 months, which excluded graft re-infection in all patients.[236]

8.2.2.2.4. Xenogenous grafts. Bovine pericardial patches can also be used to replace an infected patch or close the arterial defect. They are are supposed to be more resistant to infection than prosthesis.[237] In a series of 51 bovine pericardial patches used to close an arteriotomy where the original ePTFE patch had to be removed as a result of infection, one rupture was reported, but no other infection related complication occurred after a median follow up of 25 months.[237]

8.2.2.3. Adjunctive therapy. A growing body of research and experience suggests that muscle flap coverage in addition to graft removal with or without vascular reconstruction can be efficacious in managing these complex groin wounds and improve graft and limb salvage and survival. Several retrospective studies support the use of different types of muscle flaps in high risk vascular surgery patients, and report decreased groin wound morbidity.[238–240] However, more data are required to see clearly which type of muscle flap is ideal in which type of wound.

8.2.2.3.1. Sartorius muscle flap (SMF). SMF has been considered the standard surgical technique to treat infectious complications in the groin, in addition to a simultaneous vascular procedure.[238] There were concerns about sartorius muscle arterial blood supply in cases with superficial femoral artery occlusion, as sartorius arterial blood supply originates from multiple segmental branches of the superficial femoral and deep femoral artery. When the deep femoral artery is patent, the patency of superficial femoral artery does not affect the viability of the SMF.[241,242] However, SMF viability is compromised when the deep femoral artery is occluded. In such cases, revascularisation of the deep femoral artery is needed, either directly or indirectly (obturator bypass). In a series of 170 SMFs, of which 36 (21%) were done for infection, surgical re-intervention within 30 days was required in seven of 170 patients (4.1%) and any 30 day complications occurred in 47 patients (28%).[243] Another study compared efficacy analysis of the SMF vs. the rectus femoris flap (RFF) in the treatment of wounds following an infra-inguinal vascular procedure in 184 patients and a total of 201 flaps. There were no sentinel bleeding events through the course of graft salvage or peri-operative morbidity beyond local wound complications.[244]

8.2.2.3.2. Rectus femoris flap (RFF). A RFF is easy to harvest, has a good blood supply from the lateral circumflex femoral artery, has an excellent arc of rotation, and provides a large bulk to fill larger defects after aggressive debridement for infected and necrotic tissue. With a smaller donor site incision, the morbidity of the procedure can be decreased further.[239] There was no statistically significant difference in complications or graft salvage rates comparing the RFF with the SMF.[244]

8.2.2.3.3. Gracilis muscle flap (GMF). The retroflexed GMF has been reported to have a high rate of healing for complex infected groin wounds, even in the presence of a synthetic conduit. It is technically easy to perform and it can be accomplished safely, with a high rate of complete healing.[245] A prospective study reported a 24 month outcome of 22 patients who underwent GMF after complications resulted from arterial bypass surgery: 45% of

patients presented with graft infection, 50% with wound dehiscence, and 5% with graft disruption and bleeding. At one month, 54% of wounds were healed and 100% at three months with adjunctive NPWT and lifelong antibiotics.[246]

8.2.2.3.4. Rectus abdominis flap (RAF). When skin closure of the groin wound is desired and impossible following debridement and after infection control, RAF may provide excellent soft tissue coverage with different variations of skin paddle design. This flap has reliable anatomy, with an arc of motion that can address complex defects of the groin. Although flap transfer is highly successful, significant donor site problems, including abdominal hernia, abdominal laxity, and groin hernia have been reported.

8.2.2.3.5. Musculocutaneous anterolateral thigh flap. The musculocutaneous anterolateral thigh flap has been reported to have fewer donor site complications and can be used if the flap inflow is not compromised by previous operations.[247,248] In a retrospective study of 39 patients who underwent 30 musculocutaneous anterolateral thigh flaps and 10 RAF procedures, all flap recipient sites healed. Early post-operative complication rates (such as haematomas, surgical site infections, flap dehiscence, or skin graft necrosis) were similar in both groups. Musculocutaneous anterolateral thigh flap patients had a shorter time to healing and fewer post-operative complications than RAF patients.[247]

8.2.2.3.6. Antibiotic loaded beads. Antibiotic loaded beads can be implanted adjacent to the infected VG after wound debridement and antibacterial irrigation.[173,249,250] Different types of beads are available, including vancomycin, tobramycin, and gentamicin, or a combination thereof.[250] Conservative treatment can allow complete healing, but reinfection rates following irrigation with antibiotic beads of up to 20% have been reported.[173,250] In a retrospective series of 34 patients (36 limbs) with SSI treated with antibiotic loaded polymethylmethacrylate beads, the lower extremity bypass graft was preserved in 28 limbs and ISR was performed in eight. After a median follow up of 17 months, limb loss was 21.4% and the recurrent infection rate was 19.4%.[173]

8.2.3. Extra-anatomic reconstruction.
To avoid routing in an infected area or in multi-operated areas, extra-anatomic routing may be mandatory. Extra-anatomic routing is particularly suggested when MRSA, *Pseudomonas*, or multidrug resistant bacteria are present.

8.2.3.1. Obturator bypass (OB). OB is used to bypass infection in the groin. The inflow vessel is the intact external iliac artery and the outflow vessel is the distal superficial femoral artery (SFA) or less frequently, the distal deep femoral artery.[251] In a recent retrospective study, 15 patients underwent 18 OBs with a polytetrafluoroethylene (PTFE) prosthesis. Indications for surgery were chronic infection in 10 patients and acute bleeding in five. Midterm

outcomes included five late deaths and one myocardial infarction. Three patients underwent above knee amputation in the 58 months of follow up. Another procedure was required in 11 (61%) limbs. One OB (6%) became infected, requiring removal at 42 months. Primary, primary assisted, and secondary patency rates were 65%, 71%, and 88% at 24 months, respectively.[252] The autologous femoral vein provides an excellent size match and a suitable length for OB.[253]

8.2.3.2. Lateral retrosartorius bypass (LRSB). As an alternative route to OB, case reports have been published on LRSB when routing of the graft from the external iliac artery to the distal SFA is needed.[254] The infected area in the groin may affect the approach to the distal anastomosis on the deep femoral artery. A useful technique is to perform the dissection lateral to the sartorius muscle a few centimetres distal to the contaminated wound between healthy tissues.[255] In a recent series of 16 patients with VGI in the groin, 19 lateral femoral bypasses were performed. Three patients were operated on urgently for acute bleeding. The new bypass conduit was tunnelled laterally in the clean tissue, medial to the anterior superior iliac spine, and under the inguinal ligament through the psoas canal. The ipsilateral iliac artery was used as an inflow artery and deep femoral artery or SFA as the target distal vessel. Choice of conduit included six (32%) autogenous vein grafts, 10 (53%) allografts, two (11%) rifampicin soaked PET grafts, and one (5.3%) ePTFE graft. Two year primary assisted patency and limb salvage rates were 83% and 93.9%, respectively.[256]

8.2.3.3. Perigeniculate arteries (PGAs). PGAs may be useful if the standard exploration of the popliteal artery and the crural vessels is not possible because of infection. If the PGAs have an adequate diameter, they can be used for distal revascularisations.[257] A series of 47 bypasses with the distal anastomosis to the descending genicular artery ($n =$ 25) or medial sural artery ($n =$ 24) reported peri-operative mortality and amputation rates of 8.5% and 21.3%, respectively. Three year secondary patency, limb salvage, and overall survival rates were respectively, 83%, 74%, and 77%.[257]

8.2.3.4. Lateral approach to crural arteries (LACA). Finally, the LACA represents a simple solution to threatened limbs in otherwise difficult situations and LACA may be the ideal approach for vein grafts to the anterior tibial and distal peroneal arteries via a lateral tunnel far away from the infected tissues and with acceptable patency rates.[258] In a series of 21 patients who underwent 23 bypasses for limb salvage (10 due to infection), a LACA approach and subcutaneous graft tunnelling with autologous vein ($n =$ 21) and PTFE ($n =$ 2) was reported. The distal anastomosis was done to anterior tibial artery ($n =$ 16), peroneal artery ($n =$ 3), above knee popliteal artery ($n =$ 3), and dorsalis pedis ($n =$ 1). There were three early (13%) and four late (17%) bypass occlusions. The primary patency at one year was 61% and

the secondary patency 86%. One (4.3%) amputation was required.[258]

8.2.4. Total or partial graft explantation and need for revascularisation.
For total explantation without revascularisation, arteriotomies performed for VG removal must be closed with biological substitutes, such as saphenous vein or pericardial patch. A secondary bypass can be performed later after total eradication of the infection.

If the VG is well incorporated and does not have any obvious contamination, explantation of the whole graft may not be necessary.[259] The infected part of the VG is usually unattached to the surrounding tissue, allowing for easy removal. The removed part should be replaced with autogenous vein or biological graft material (see 8.2.2.2). Using this approach, elimination of the infection, preservation of limb perfusion, and long term survival are achieved in at most, 70% — 80% of patients presenting with VGI.[225,260,261]

Of the 27 patients who underwent 30 partial PTFE graft explantations, re-infection occurred in 15% during a 27 month follow up.[262] In another series of 86 extracavity VGIs (initial procedure: 24 aortobifemoral bypasses; 19 extra-anatomic bypasses; 31 infra-inguinal bypasses; and 12 combined outflow/inflow reconstructions) the graft was preserved in three patients, graft excision and extra-anatomic bypass was performed in four patients, and graft was removed and replaced with ISR in 79 patients (rifampicin bonded PTFE $n = 22$, autologous conduit $n = 57$). Recurrent SSIs occurred in 66% of patients treated initially with graft preservation and 14% of patients receiving rifampicin bonded ISR and in one patient who had an isolated segment of retained PET graft from a previous bypass procedure. One early and five late re-infections (mean interval of 23 months) were reported. Re-infection rates by life table estimate were 2.3% and 8% at one and five years, respectively.[216]

Although several studies have suggested equivalent rates of re-infection and sepsis after partial or complete resection of infected lower extremity bypasses, these patients should be under surveillance until the infection has been eradicated and the wounds are healed owing to the possibility of recurrent infection in the remaining graft.[157,262,263] Consequently, the combination of leaving minimal VG in the surgical field, aggressive irrigation, and antimicrobial therapy may alleviate the risk of further infection.[262] The treatment modality should be tailored to the patient's condition, to the availability of autologous vein, cryopreserved allograft or an alternative vascular conduit, and to the surgeon's experience.[260,264,265]

8.2.5. Timing of surgery.
Procedures to treat VGI should take place without delay. For any bleeding from an anastomosis, there is a possibility of massive bleeding and the patient should be operated on on an on call basis. Also, abscesses should be drained and devitalised tissue resected within a few hours. Infected graft material should be resected during the same procedure. If there is no critical limb threatening ischaemia after the graft resection, secondary revascularisation may be delayed until the wounds have healed.

8.3. Follow up and prognosis
Peripheral VGI is always a serious condition with high risk of amputation and re-infection if all prosthetic material cannot be removed. Surveillance is usually needed not only because of underlying severe lower limb arterial disease, but also to exclude re-infection, especially if resection of the infected material was incomplete. Few reports on surveillance and prognosis after lower limb VGI exist. In a report of 66 cases, 13% had major limb amputation within 30 days post-operatively regardless of location and type of bypass.[266] In another study of 37 cases of femoral VGI, the re-infection rate was 12% and the long term limb salvage rate was 86.5%.[221]

There are no scientific studies on the optimal surveillance protocol. Surveillance after reconstruction should be close until there are no signs of infection. Thereafter, routine follow up visits should be done annually, including laboratory markers of infection and CT. Patients unfit for surgery who are under conservative treatment and lifelong antibiotics should also be under continuous follow up. After the acute situation has settled down, check up every three months during the first year and every 6 — 12 months thereafter if no sign of recurrent infection is seen, can be performed.

Recommendation 49		
For patients with peripheral vascular graft/endograft infection, *in situ* reconstruction with autologous vein is recommended if removal of the infected graft is likely to lead to limb ischaemia.		
Class	Level	References
I	C	Siracuse et al. (2013)[221]

Recommendation 50		
For patients with peripheral vascular graft/endograft infection limited to only a part of the graft and in patients unfit for surgery, local irrigation and/or negative pressure wound therapy may be considered.		
Class	Level	References
IIb	C	Thermann and Wollert (2014),[213] Andersson et al. (2018),[218] Monsen et al. (2014)[219]

Recommendation 51		
For patients with a peripheral vascular graft/endograft infection and a large tissue defect, negative pressure wound therapy should be considered in order to promote wound healing following infected graft removal and debridement with or without vascular reconstruction.		
Class	Level	References
IIa	C	Verma et al. (2015),[215] Armstrong et al. (2007),[216] Cheng et al. (2014)[217] Andersson et al. (2018),[218] Monsen et al. (2014)[219]

Recommendation 52

For patients with peripheral vascular graft/endograft infection, *in situ* reconstruction with cryopreserved allografts should be considered as an alternative after infected graft removal if it is likely to lead to limb ischaemia.

Class	Level	References
IIa	C	Lejay *et al.* (2017),[147] Ehsan and Gibbons (2009),[223] Zetrenne *et al.* (2007),[225] Gabriel *et al.* (2004),[226] Verhelst *et al.* (2004)[227]

Recommendation 53

For patients with a peripheral vascular graft/endograft infection and a large tissue defect, muscle or musculocutaneous flaps should be considered to promote groin healing following graft removal and debridement with or without vascular reconstruction.

Class	Level	References
IIa	B	McMillan *et al.* (2012),[237] Brewer *et al.* (2015),[238] Mirzabeigi *et al.* (2017),[244] Dua *et al.* (2018)[246]

9. PATIENTS' PERSPECTIVES

Whatever the specialty in medicine, the treatment proposal to a patient should be based on a clear, honest and informed consent. The patient should be able to understand the balance between risks and benefits when a surgical procedure is proposed to him/her. As vascular procedures require the use of definitively implanted synthetic devices in the vast majority of the cases, and because infection is a more threatened complication following synthetic material implantation, the risk of VGEI should clearly be explained to the patient.

9.1. Specific aspects

The following specifics of vascular procedures must be considered from a patient perspective:

9.1.1. Pathological presentations. Vascular procedures are performed either in an emergency setting for life or limb threatening presentations, or for functional impairment or prevention of arterial disease related complications. While the risk of secondary infection is easily accepted in life or limb threatening presentations, this is not the case in the latter presentations.

9.1.2. Patient age and comorbidities. Vascular surgery procedures are aimed at a specific population consisting of older patients with a lot of cardiovascular, pulmonary, and renal comorbidities. This particular frailty makes not only secondary interventions more difficult to handle, but also all the management, including imaging procedures with iodine, or antimicrobial therapy.

9.1.3. The "easy" endovascular surgery. Currently, most procedures are performed endovascularly in the era of minimally invasive surgery and consequently open surgery

is performed less often. Even if endovascular surgery is a less invasive modality, it must be presented as a treatment option for a severe disease. Moreover, open surgery in the setting of VGEI is more difficult than the initial procedure. This should be explained to patients.

9.2. Patient feedback

9.2.1. Question 1: Did you feel your physician provided enough information about the risk of infection at the time of the initial procedure? Patients felt that not enough information was provided about the risk of infection: some patients answered that they had not been informed about the risk of infection, while others said that they had been informed but did not understand what a VGEI meant. Some patients had received antibiotic prophylaxis from general physicians, while others had not.

Patients also stated that the the initial procedure was performed in general hospitals, while re-intervention was performed at university hospitals. Later daily care was performed by general physicians lacking experience in advanced knowledge of graft infections, stents, and so on.

9.2.2. Question 2: What did you think about the management once the diagnosis of VGEI was made? It seems that the difficulty in obtaining a final diagnosis was generally well understood by patients. They understood the necessity of a complete microbiological and imaging work up. However, it has been pointed out that during the management, the patient moved from one department to another, i.e., vascular surgery, infectious disease, and intensive care unit, which impaired the relationship with a central physician. Patients thought that management was lacking in structure, and that a treatment algorithm was followed.

9.2.3. Question 3: Did you think your physician provided enough information on the risks related to the VGEI? Patients thought that they were not aware of the complexity and seriousness of the situation. The majority of patients did not anticipate the difficulties, the pain, or the tiredness induced by the procedures. They mentioned that they were not aware of the close follow up needed after re-intervention.

9.3. Ways of improvement

To summarise, the following recommendations from a patient's perspective could be suggested: (i) vascular surgeons must provide more precise information on the risk of VGEI whatever the procedure, even endovascular, and explain precisely what VGEI means in terms of invasiveness; (ii) there is a need to provide more education to general practitioners on antibiotic prophylaxis measures, as well as on when to suspect VGEI to make patients more confident once out of the hospital — precise information about the necessity for post-operative follow up screening is important; (iii) physicians should be more aware of the need for a referral physician to provide information on his/her status, the evolution of the disease, and the next management steps.

10. UNRESOLVED ISSUES

The literature review performed to propose these ESVS Guidelines on VGEI demonstrated a lack of robust evidence in many aspects of VGEI management. The literature did not provide enough RCTs, but mostly retrospective studies, literature reviews, and some consensus articles. As a consequence, most recommendations in these guidelines have a low level of evidence, mostly level C. Even the conclusions of papers, including large series, are debatable for the following reasons:

- Populations are difficult to compare because of the multiple clinical presentations, the different microbiology protocols for micro-organism sampling and studies, the different imaging modalities, and the different management options in terms of antimicrobial therapy duration, or choice of vascular substitutes.
- Large series proposing treatment modalities have mostly been published by tertiary centres and most often favour one technique over another. Consequently, there is a high risk of bias related to only publishing positive results while probably not reporting poorer experiences.
- Reviews and meta-analysis, potentially more valid than series, could help in establishing valuable comparisons between different management options. However, they still have to be interpreted with caution because of the previous comments on the potential heterogeneity of the series they pooled.

Consequently, we conclude on the necessity to set up large, multicentre registries that could include patients prospectively with predefined variables allowing for valuable comparisons and conclusions. Such registries could be the way to answer unresolved issues. Accordingly, the WC proposes the following areas for research:

- The evaluation of the risk of brain abscesses in the presentation of VGEI involving the SAT in order to evaluate the potential emergency for management: This major risk is often mentioned, but there are no reliable data available on its incidence (see 5.1).
- The evaluation of the efficacy of EndoVAC therapy: this is mandatory before extensive use, as to date results have only been reported by one team (see 5.2.2).
- The efficacy of conservative treatment of VGEI with drainage and irrigation: it is often presented as a conservative alternative to direct graft explantation, but there are no data reported, as in most series it is only considered as a second line option in unfit patients (see 5.2.1, 6.2.1, 7.2.1, and 8.2.1).
- Evidence on the best antiseptic solutions for irrigation: the literature and expert exchanges demonstrate there is no strong evidence on the different choices in clinical practice (see 4.1).
- Evidence on the efficacy of conservative treatment with implantation of an abdominal or thoracic EG as a bridge: this includes taking into consideration research on the two previous points (see 6.2.2).

- The future role of imaging technologies, including bio-optical imaging, which is a technology allowing evaluation of biomaterials. The wide range of morphological signs corresponding to the different VGEI anatomical locations and clinical presentations requires development of functional imaging. Bio-optical imaging is a technology allowing evaluation of biomaterial associated infections with highly sensitive cameras enabling detection of very few photons from bioluminescent or fluorescent sources inside the body without the need of surgical intervention (see 2.8).
- Antibiotic prophylaxis for invasive manipulation in VGEI carriers (see 3.2.2.2).
- Evidence on the optimal duration of antimicrobial therapies: it is necessary to define the optimal duration and type of antimicrobial treatment depending on the clinical presentation, the anatomical location, and the type of reconstruction, after VGEI according to clinical, biological, and imaging follow up (see 4.1).
- Evidence on the best substitutes for ISR are still needed: this includes clinical evidence of the best coating for an infection resistant VG and evaluation of the risk of microbiological mutation related to the use of a rifampicin soaked VG. The actual risk of re-infection depending on the materials must be evaluated. The growing interest in the effectiveness of xenograft materials for ISR must be evaluated and confirmed (see 6.2.2.2, 7.2.2.2, and 8.2.2.2).
- The best management of duodenal anastomosis, on site or supramesocolic, for AEnF treated by ISR (see 7.3).
- The best management of infected aortic EG constructed with suprarenal bare stents (see 7.2.6).
- The evaluation of secondary endovascular treatment of an occluded superficial femoral artery in infected femoro-popliteal bypass cases (see 8.2).
- The new products that are under evaluation: they include biological extracellular patches such as the one derived from porcine small intestinal mucosa, which provides a biological scaffold for cellular ingrowth and eventual tissue regeneration.
- Vascular tissue engineering is also at the forefront of its translation into practice, as tissue engineered VG have already been successfully implanted in children. There is a high demand in other areas of vascular surgery for ideal graft materials, such as for infection resistant conduits in septic conditions. However, tissue engineered VG are not ready for routine use as the quest for the ideal scaffold materials, cell sources, and *in vitro* culturing conditions continues. Further animal and clinical trials are required.

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