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Review

Reoperations for infective endocarditis in adults with congenital heart disease: Challenges, considerations, and contemporary strategies

Özge Çetinarslan[a,*], Sertaç Çiçek[b]

[a] Department of Cardiology, Istanbul Florence Nightingale Hospital, T.C. Demiroğlu Bilim University, Faculty of Medicine, Istanbul, Turkey
[b] Department of Cardiovascular Surgery, Istanbul University Faculty of Medicine, Istanbul, Turkey

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ABSTRACT

Infective endocarditis (IE) in adults with congenital heart disease (ACHD) is a rare but increasingly recognized complication that carries significant morbidity and mortality. The presence of prosthetic material, complex surgical histories and frequent multi-organ involvement render reoperations particularly complex and challenging. This review synthesizes current understanding and strategies for the management of IE in the ACHD population, with a focus on surgical reintervention. It incorporates contemporary guideline updates, registry data, and institutional insights to propose best practices in preoperative planning, operative technique, and long-term care. As the ACHD population grows and the burden of IE persists, individualized protocols and treatment at experienced centers remain crucial for achieving optimal outcomes.

Reoperaciones por endocarditis infecciosa en adultos con cardiopatías congénitas: desafíos, consideraciones y estrategias contemporáneas

Palabras clave:
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Endocarditis infecciosa
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RESUMEN

La endocarditis infecciosa (EI) en adultos con cardiopatía congénita (ACHD) es una condición rara pero cada vez más frecuente, con alta morbilidad y mortalidad. Los antecedentes quirúrgicos complejos, la presencia de material protésico y la afectación multiorgánica en esta población hacen que las reoperaciones sean particularmente desafiantes. Esta revisión destaca la comprensión y el manejo actuales de la EI en la población con ACHD, con énfasis en las estrategias de reintervención quirúrgica. Se basa en actualizaciones recientes de guías clínicas, datos de registros y experiencias institucionales para resaltar las mejores prácticas en la planificación preoperatoria, la técnica quirúrgica y el manejo a largo plazo. Dada la creciente población con ACHD y la importante morbilidad asociada a la EI, los protocolos personalizados y los centros con experiencia siguen siendo esenciales para lograr resultados óptimos.

Introduction

The steady expansion of the adult congenital heart disease (ACHD) population reflects the remarkable progress made in pediatric cardiology, cardiac surgery, and long-term surveillance strategies over the past several decades.[1] While most children born with congenital heart disease (CHD) previously faced limited survival prospects, today the overwhelming majority estimated at over 95% reach adulthood, transforming CHD into a lifelong condition rather than a fatal early diagnosis.[2] Nevertheless, only a small proportion of these individuals can be considered fully corrected following surgical intervention. The long-term outcome depends not only on whether the initial procedure was palliative or corrective, but also on the complexity of the underlying defect and the extent of surgical reconstruction. Many patients, particularly those with anatomically complex lesions continue to live with prosthetic valves, conduits, patch materials, non-physiologic flow patterns,

Abbreviations: ACHD, adult congenital heart disease; AHA, American Heart Association; CHD, congenital heart disease; CIED, cardiac implantable electronic device; CNIE, culture-negative infective endocarditis; CoNS CPB, coagulase-negative staphylococci cardiopulmonary bypass; CT, computed tomography; Duke-ISCVID, Duke-International Society for Cardiovascular Infectious Diseases; ESC, European Society of Cardiology; FDG-PET/CT, fluorodeoxyglucose positron emission tomography/computed tomography; GUCH, grown-up congenital heart disease; HACEK, Haemophilus, Aggregatibacter, Cardiobacterium, Eikenella, Kingella; IE, infective endocarditis; MRI, magnetic resonance imaging; PVE, prosthetic valve endocarditis; RV-PA, right ventricle-pulmonary artery; TEE, transesophageal echocardiography; TPV/TPVI, transcatheter pulmonary valve/transcatheter pulmonary valve implantation; TTE, transthoracic echocardiography; WBC, white blood cell.

* Corresponding author.
E-mail address: ozgecetinarslan@windowslive.com (Ö. Çetinarslan).

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and residual shunts or obstructions. In addition, long-standing circulatory abnormalities may result in extracardiac complications, including hepatic, renal, or pulmonary involvement.

These long-standing structural and hemodynamic abnormalities create a vulnerable cardiac substrate that predisposes patients to infective endocarditis (IE), which, although uncommon, remains a clinically formidable condition with significant diagnostic and therapeutic implications.[3] Although the incidence remains low in absolute terms, ACHD patients exhibit a dramatically elevated lifetime risk of IE up to 27–55 times that of the general population owing to prosthetic interfaces, non-physiologic flow patterns, and altered endothelial surfaces.[4] Infections may occur on native, prosthetic, or surgically manipulated tissue, often in anatomically inaccessible regions, complicating both diagnosis and intervention.

Material and methods

This review was conducted as a narrative, non-systematic synthesis of the current literature on IE in ACHD patients, emphasizing surgical strategies and reoperation principles. A focused search was performed in PubMed, MEDLINE, and Google Scholar between January 2000 and June 2025, using keywords such as "infective endocarditis," "congenital heart disease," "adult congenital," "reoperation," "prosthetic infection," and "Duke criteria."

We prioritized English-language publications, giving preference to original research, large cohorts, expert guidelines, and recent systematic reviews. While not exhaustive, this approach reflects standard practices for narrative clinical reviews.[5]

Given the heterogeneity of the literature and lack of randomized trials in this population, this review aims to provide a clinically grounded summary rather than a meta-analysis. Strength of evidence is contextualized based on study design and consistency across sources.

Infective endocarditis in ACHD patients

While ACHD includes both unrepaired and repaired lesions, in contemporary healthcare settings, particularly in resource-advanced regions most ACHD-IE cases arise in previously repaired adults. Yet these "repaired" hearts often harbor turbulence-inducing anatomies, residual shunts, valve prostheses, or synthetic conduits, which not only perpetuate hemodynamic stress but also serve as niduses for microbial colonization.[5] The incidence of IE in ACHD is significantly elevated compared to the general population, with estimates ranging from 1.3 to 4.1 cases per 1000 patient-years. This translates to an approximately 15- to 140-fold increase in relative risk, primarily influenced by underlying anatomy and the presence of prosthetic material.[6]

Importantly, this risk is not uniform across the ACHD population. Patients with cyanotic lesions, prior palliative shunts, or prosthetic conduits are particularly vulnerable, whereas those with simple, well-repaired defects and no residual shunts exhibit considerably lower susceptibility.[4] The inclusion of synthetic valves, patches, or conduits especially in the context of residual turbulence further increases the risk of bacterial colonization. Moreover, the risk persists long after surgical repair and may increase over time, particularly in those with hemodynamic abnormalities or extracardiac organ involvement. These patterns highlight the need for individualized, anatomy-based risk stratification models in ACHD patients.[3,7]

In a large population-based cohort of nearly 30,000 ACHD patients followed over 379,000 patient-years, the highest incidence was observed in patients with left-sided lesions primarily aortic and mitral malformations at a rate of 1.61 cases per 1000 patient-years.[6] Cyanotic lesions and ventricular septal defects followed, with incidence rates of 1.17 and 0.65 per 1000 patient-years, respectively. In contrast, atrial septal defects, right-sided lesions, and patent ductus arteriosus were associated with lower incidence rates, ranging from 0.24 to 0.28 per 1000 patient-years. The study further identified that endocardial cushion defects, recent cardiac surgery, and medical interventions were independently associated with a significantly elevated risk of IE. These lesion-specific insights are critical for stratifying risk, guiding prophylactic strategies, and prioritizing follow-up intensity in the ACHD population.[6]

Not all adults with ACHD carry the same risk of developing IE. The incidence of IE in this population varies significantly depending on the underlying cardiac lesion and a range of contributing factors including whether the defect has been repaired or remains unrepaired, the presence of residual valvular regurgitation or stenosis, and the implantation of prosthetic materials such as valves, conduits, or patches.[7] These structural and hemodynamic alterations, particularly when combined, create a vulnerable substrate that predisposes certain subgroups patients to a markedly higher risk of endocardial infection.

Infective endocarditis in ACHD patients demonstrates unique patterns in anatomic involvement when compared to non-CHD populations. While left-sided IE remains more frequent overall, ACHD patients exhibit a disproportionately higher prevalence of right-sided or mixed valvular involvement due to prior surgical interventions and anatomical substrates such as conduits, shunts, or prosthetic materials. Aortic and pulmonary valves are more commonly affected in ACHD patients, whereas non-CHD patients more often present with mitral or tricuspid valve involvement.[8]

Surgical implications differ accordingly. Right-sided IE, often associated with lower-pressure circuits and fewer embolic complications, may permit more conservative management or valve repair. Conversely, left-sided IE particularly involving the aortic or mitral valves is associated with a higher risk of embolization, systemic complications, and mortality, necessitating prompt surgical intervention. These distinctions should guide operative urgency, approach, and prosthesis selection in ACHD reoperations.

Of particular concern in recent years is the subgroup undergoing transcatheter pulmonary valve implantation. Patients undergoing transcatheter pulmonary valve implantation notably with Melody™ or Sapien™ valves comprise a distinct high-risk subgroup.[9] Melody valve recipients have demonstrated an annualized IE incidence of approximately 2.4% per patient-year, while comprehensive reviews estimate TPVI-associated IE rates ranging from 13 to 91 per 1000 person-years for Melody using aggregated registry data.[10] Meta-analytic comparisons further show higher incidence following Melody implantation (~8.5%) vs. Sapien valves (~2.1%), underscoring device-specific risks.[11] Moreover, data show that while TPV recipients may have lower early IE risk compared to surgical pulmonary valve replacements, long-term risk increases and eventually exceeds that of surgical cohorts.[12] Expert consensus situates TPVI-IE risk between 13 and 91 per 1000 person-years for Melody and 8–17 for Sapien implants, with stratifiers including prior IE, residual gradient and immunosuppression.[10]

In contrast, patients with simple, well-repaired defects and no residual prosthetic material remain at much lower risk. These observations emphasize the imperative for lesion- and device-specific risk stratification when evaluating long-term IE surveillance strategies in ACHD patients.

Microbiological and clinical differences between ACHD and non-congenital IE

ACHD exhibit distinctive microbiological patterns and clinical characteristics in IE compared to individuals with structurally

normal or acquired heart disease. The underlying cardiac substrate, prior surgical interventions, and long-term exposure to prosthetic materials contribute to a unique infectious profile in this population.

Several studies report *Streptococcus* species as the predominant causative agents of IE in ACHD patients, highlighting the potential role of oral or mucosal entry points in these cases.[13–15]

Conversely, other contemporary investigations have found *Staphylococcus* species, particularly *Staphylococcus aureus* to be the most frequently isolated pathogens.[8,16]

Large registry data, such as those from the EURO-ENDO study, further support this variability: while *S. aureus* remains the leading single pathogen in both ACHD and non-ACHD cohorts, ACHD patients tend to have a relatively higher proportion of infections due to *Streptococcus* species and fastidious organisms.[17] A recent population-wide registry analysis by Havers-Borgersen et al. found *Streptococcus* species to be the leading cause of IE in CHD patients across all age groups, responsible for nearly one-third of cases. The study also noted a higher incidence of *coagulase-negative staphylococci* (*CoNS*) and *HACEK* organisms in the CHD population, whereas *Enterococcus* species were comparatively less common than in non-CHD patients.[18]

Culture-negative IE (CNIE) appears to be more frequent in the ACHD population, with reported rates ranging from 18% to 35. This is likely related to prior antibiotic exposure, the presence of prosthetic materials, and chronic use of indwelling lines.[19,20]

Fungal IE, particularly due to *Candida* species, is reported more frequently in ACHD patients. These cases are often linked to long-term central venous access, immunosuppression, or prosthetic conduits, and they tend to follow a more aggressive course with poorer outcomes.

Although specific fungal endocarditis incidence in ACHD populations is underreported, case series from congenital patients clearly demonstrate elevated mortality often exceeding 30–50% driven by rapid progression, embolic complications, and diagnostic delays.[21] Notably, prosthetic *Candida albicans* endocarditis in ACHD surgical cohorts frequently necessitates combined surgical debridement and long-term antifungal therapy to achieve stability and prevent recurrence. Additionally, rare fastidious organisms such as *Bartonella* and *Coxiella burnetiid* appear more commonly in ACHD patients, often contributing to subacute or culture-negative presentations of IE.

Clinically, ACHD patients tend to present with IE at a much younger age (~38–40 years) than the general IE population (~70 years) and often experience systemic embolism, right-sided vegetations, and multivalvular involvement.[22] Despite the frequent presence of complex congenital anatomy, extracardiac comorbidities, and diagnostic challenges, in-hospital mortality among ACHD patients with IE remains lower (~5–6%) than in non-ACHD populations (~10–17%). Data from the European Infective Endocarditis Registry (EURO-ENDO) on a subset of patients with endocarditis and CHD reported an in-hospital mortality of 9% vs. 18% and 1-year mortality of 13% vs. about 25% in those with compared to those without CHD.[8] This favorable outcome may partly reflect the younger age and greater physiological reserve typically seen in this group. In addition, ACHD patients are often managed in specialized centers with congenital expertise, where routine surveillance and heightened clinical awareness facilitate earlier diagnosis and timely intervention. A further contributing factor may be the relatively higher prevalence of right-sided endocarditis in this population, which is generally associated with more favorable outcomes compared to left-sided involvement.

Clinical presentation and diagnosis

The clinical manifestations of IE in ACHD are often subtle, variable, and influenced by the underlying anatomy and the presence of prosthetic materials. Fever remains the most common presenting symptom, but it may be absent in immunosuppressed individuals or those on chronic suppressive antibiotics. Constitutional symptoms such as malaise, fatigue, weight loss, night sweats, and arthralgia are common. Cardiac findings including new or changing murmurs and signs of heart failure must be interpreted with caution in the context of complex anatomy and baseline hemodynamic abnormalities.[23]

While the EURO-ENDO registry encompasses a broad spectrum of adult patients with IE rather than exclusively those with congenital heart disease, it provides valuable general insights relevant to ACHD populations. In this large multinational cohort of 3113 adults hospitalized for IE, fever (77.7%), new cardiac murmur (64.5%), and congestive heart failure (27.2%) were the most commonly reported clinical features. Embolic events were observed in 25.3% of patients upon admission, and conduction disturbances including atrioventricular block were noted in 11.5%.[17]

Right-sided IE may present with cough, dyspnea, pleuritic pain, and septic pulmonary emboli, particularly in patients with right ventricle-to-pulmonary artery conduits, central shunts, or transvenous devices. Left-sided IE, more common in older ACHD patients and those with aortic or mitral prostheses, often presents with systemic embolization, neurologic events, splenic infarcts, or glomerulonephritis.

Diagnosis is based on a combination of clinical, microbiologic, and imaging findings. While the modified Duke criteria have long served as the diagnostic cornerstone, they are less sensitive in ACHD patients due to altered anatomy, prosthetic interference, and complex flow dynamics. The updated 2023 Duke-ISCVID criteria now incorporate advanced molecular diagnostics and hybrid imaging techniques, offering improved sensitivity and specificity in this challenging group.[24] Blood cultures remain a diagnostic mainstay and should be obtained prior to initiation of antibiotics, ideally in at least three separate sets over a 24-h period. Culture-negative IE due to fastidious organisms, prior antibiotics, or fungal infection is more common in ACHD and requires molecular or histopathologic confirmation when possible.

Transthoracic echocardiography (TTE) remains the first-line imaging modality in suspected IE, with the detection of vegetation, abscesses, or new valvular regurgitation constituting major diagnostic criteria. However, TTE is inherently limited by its variable sensitivity – reported between approximately 40% and 80% – especially in patients with prosthetic valves or complex anatomy.[25,26] Transesophageal echocardiography (TEE), by contrast, offers superior sensitivity (often exceeding 90%) for native valve cases and remains the preferred modality when TTE is nondiagnostic or pretest probability is high. Yet even TEE has important limitations in ACHD: visualization of anterior right-sided structures such as the right ventricle, RVOT conduits, and branch pulmonary arteries may be compromised, particularly in patients with prosthetic material or stents. In these scenarios, a negative TEE cannot be assumed to exclude IE reliably.[25]

Specifically, acoustic shadowing or prosthetic artifact often obscures identification of vegetations in the RV-PA region or pulmonary valve. Masking by prior surgical alterations can significantly reduce TEE sensitivity in ACHD. Therefore, while TEE generally outperforms TTE overall, interpreting a TEE as definitively negative in ACHD requires caution. In cases of persistent clinical suspicion despite negative imaging, repeat or alternative imaging modalities should be considered.[26]

In summary, both TTE and TEE remains indispensable in diagnostic evaluation but must be applied with awareness of its reduced diagnostic yield in ACHD. When standard imaging is inconclusive, multimodality imaging such as cardiac CT, PET/CT and MRI can provide critical supplemental information. [18F]-FDG PET/CT and cardiac CT angiography offer added value by identifying metabolically active infection, prosthetic infection, or paravalvular extension not visualized on echocardiography.[26]

Cardiac computed tomography (CT) plays a valuable role not only in the diagnosis of IE but also in detecting associated intracardiac complications. Cardiac CT demonstrates superior accuracy to TEE in identifying perivalvular and periprosthetic abnormalities, such as abscesses or pseudoaneurysms. Furthermore, the detailed anatomic insights provided by CT can substantially inform surgical planning and guide intraoperative strategy.[27]

Additionally, metabolic imaging modalities such as 18F-fluorodeoxyglucose positron emission tomography/computed tomography (18F-FDG PET/CT) and white blood cell (WBC) single-photon emission computed tomography (SPECT/CT) are valuable tools, particularly in prosthetic valve endocarditis (PVE) or cardiac implantable electronic device (CIED)-related infections when standard imaging is equivocal.[28,29] Moreover, brain and systemic imaging with CT, PET/CT, or MRI is recommended in symptomatic individuals and considered in asymptomatic patients to identify potential peripheral embolic lesions associated with IE.[26]

Delayed or missed diagnosis dramatically increases the risk of complications including abscess formation, perivalvular extension, heart block, systemic embolization, and irreversible hemodynamic deterioration. Multimodal imaging and early multidisciplinary involvement are critical to timely diagnosis and intervention.

Reoperation in ACHD patients

Surgical indications – perioperative considerations and timing

Reoperative surgery for IE in ACHD patients is among the most demanding scenarios in contemporary cardiovascular practice. These patients frequently present with complex anatomic substrates, prior surgical interventions, and prosthetic materials including conduits, mechanical valves, or transcatheter devices. When endocarditis occurs in this setting, it often manifests as a systemic, aggressive, and recurrent infection.[6] These characteristics significantly complicate both diagnosis and surgical management, necessitating a tailored approach guided by multimodal imaging, institutional experience, and congenital expertise.

Surgical indications for IE in ACHD patients are broadly aligned with general endocarditis guidelines but must be interpreted within the unique framework of congenital anatomy and prior interventions.[26] Clinical thresholds, timing, and operative feasibility might differ.

Indications include:

- Heart failure due to valvular destruction, fistulization, or abscess formation
- Persistent infection despite ≥7–10 days of appropriate antibiotic therapy
- Large mobile vegetations (>10–15 mm), especially on the mitral or aortic valve
- Recurrent embolization, particularly during the first 2 weeks of therapy
- Infection by highly virulent or fungal organisms, such as *S. aureus* or *Candida* spp.
- Prosthetic material infection, which almost always necessitates surgical debridement and replacement
- Perivalvular extension, including annular abscess, pseudoaneurysm, or fistula formation
- Conduction disturbances, suggestive of deep infection near the conduction system

Among ACHD patients, prior implantation of prosthetic valves or conduits increases the likelihood of complications. Foreign material not only serves as a nidus for infection but also impedes sterilization and promotes recurrence if not removed.[7]

Currently available surgical risk scores often overestimate operative mortality in patients with ACHD who develop IE. Relying solely on these generalized risk models may lead to unjustified denial of potentially life-saving surgical interventions. For this reason, it is essential that risk assessment be performed on an individual basis by a multidisciplinary team with expertise in congenital cardiology, cardiac surgery, and endocarditis. The decision to withhold surgery should only be made after a thorough evaluation by a dedicated endocarditis team within a specialized center.[30]

Non-cardiac organ involvement is a critical consideration in the preoperative and postoperative management of ACHD, as nearly all organ systems may be affected to varying degrees. Renal dysfunction is particularly prevalent: in a study involving over 1000 ACHD patients, approximately 50% exhibited an abnormal glomerular filtration rate, which carried important prognostic implications.[31] Pulmonary dysfunction is also highly prevalent, with more than 40% of ACHD adults demonstrating abnormal pulmonary function tests, a finding significantly correlated with the underlying cardiac anatomy and long-term survival.[32] Both renal and pulmonary impairments have been independently associated with increased mortality in this population. Hepatic dysfunction, especially among patients with Fontan physiology, is increasingly recognized, though its broader implications across the ACHD spectrum remain incompletely defined.[33]Additionally, cyanotic ACHD patients represent a particularly vulnerable subgroup, often exhibiting hematologic derangements and multisystem involvement. Preexisting non-cardiac comorbidities particularly chronic hepatic or pulmonary dysfunction have been shown to significantly elevate surgical risk and are associated with higher perioperative mortality in adults undergoing cardiac surgery.[34] These systemic considerations underscore the necessity of comprehensive preoperative evaluation and multidisciplinary risk stratification in ACHD patients undergoing surgical intervention.

In patients with neurologic complications, the timing becomes more nuanced. ESC 2023 guidelines[26] recommend:

- Immediate surgery following ischemic stroke if there is no hemorrhage and the patient is not comatose
- Delayed surgery (>4 weeks) following intracranial hemorrhage, unless emergent cardiac decompensation demands earlier intervention

These decisions require joint input from neurology, cardiology, cardiac surgery, infectious disease, and anesthesiology. In ACHD patients, especially those with Fontan circulation or pulmonary hypertension, anesthetic and hemodynamic considerations may further impact surgical candidacy. The importance of operating before irreversible multi-organ failure develops cannot be overstated. As shown in recent studies, patients who were declined surgery due to hemodynamic instability or comorbidities had the highest mortality, often within days of presentation.[35] This highlights the need for early referral to specialized centers and proactive surgical planning even before definitive radiologic or microbiologic confirmation is complete.

Intraoperative and technical considerations

The overarching goals of surgery for IE in ACHD are:

- Complete and radical debridement of all infected or necrotic tissues
- Anatomical reconstruction, respecting the patient's unique congenital substrate
- Hemodynamic restoration, ensuring physiological durability

While these principles align with general IE surgery, reoperations in ACHD patients are marked by extreme variability in anatomy, prior surgical reconstructions, and prosthetic exposure – factors that substantially increase operative risk. The surgical landscape is often made more hostile by the aggressiveness of the infective process, particularly in the setting of *S. aureus*, fungal, or culture-negative organisms. The risk of injury during reentry, the difficulty of complete debridement, and the technical limitations of standard valve replacement techniques all contribute to poor outcomes if not meticulously addressed. Successful reoperation demands rigorous preoperative planning and intraoperative adaptability, rooted in a deep understanding of congenital anatomy and infection pathophysiology.

Operative access and setup

Reentry into a previously operated chest mandates caution. Dense retrosternal adhesions may obscure the great vessels, prosthetic conduits, or a dilated right ventricle lying directly beneath the sternum. Preoperative contrast-enhanced CT imaging is indispensable for assessing the proximity of cardiac structures to the posterior sternum, evaluating the integrity of previous grafts or conduits, and identifying vascular access options.[36]

Selected patients may benefit from alternative surgical approaches, including right or left thoracotomy, particularly when the infection is localized to a conduit or valve accessible laterally. Hybrid procedures, while still evolving in IE management, may offer a less invasive solution in extremely high-risk patients, though experience remains limited. In high-risk patients, particularly after multiple prior sternotomies or with anteriorly located conduits, peripheral cannulation should be considered to allow for decompressed, controlled reentry under cardiopulmonary bypass.

Intraoperative TEE is essential in ACHD-IE reoperations to re-evaluate providing real-time reassessment of cardiac anatomy, extent of vegetations, presence of abscesses or fistulae and guide valve repair or replacement. Intraoperative TEE particularly critical for detecting residual intracardiac shunts before opening cardiac chambers, an essential step vital for minimizing the risk of catastrophic air embolism.

Valve and conduit management

The fundamental principal of IE surgery is radical debridement of all infected and prosthetic material. In ACHD patients, this often involves explantation of conduits, prosthetic valves, and infected patches. While native valve preservation is desirable particularly in young adults it must not compromise the completeness or durability of debridement. Valve-sparing techniques are preferred only when long-term competence and infection eradication are achievable.

Mitral valve repair is favored over replacement, particularly in localized endocarditis with leaflet perforation or vegetations provided it ensures competence and durability. Residual regurgitation or stenosis following suboptimal repair is unacceptable due to high-risk relapse or reintervention.

Aortic valve IE with annular abscess or extensive destruction may necessitate full root replacement. Homografts and stentless bioprostheses offer enhanced resistance to reinfection in this context. Although technically demanding the Ross procedure remains a viable option in young patients with suitable anatomy.

RV-PA conduits or transcatheter pulmonary valves require complete explantation. Homograft or bioprosthetic replacement is standard, tailored to anatomic considerations and infection severity.

Meta-analyses and observational registries affirm that radical explantation and valve replacement, particularly in *S. aureus* or fungal IE, often result in superior outcomes even when valve repair is technically feasible but likely suboptimal. In ACHD patients, leaving residual lesions or prosthetic material may portend early recurrence and suboptimal hemodynamics to a far greater degree than a well-placed prosthesis.[37]

Prosthetic valve and conduit selection

The selection of valve prostheses and conduits in ACHD patients with IE must consider not only anatomical and hemodynamic factors but also the infectious substrate, patient comorbidities, and likelihood of future reintervention. Bioprosthetic valves are generally favored in patients with contraindications to anticoagulation such as those with prior hemorrhagic stroke, pregnancy plans, poor compliance, or limited access to follow-up care. Conversely, mechanical valves may be appropriate in younger patients with stable anticoagulation regimens and where long-term durability is critical.[37]

When reconstructing the right ventricular outflow tract (RVOT) or aortic root in the setting of prosthetic endocarditis, cryopreserved homografts are often considered the ideal option. Their lack of synthetic material and favorable resistance to reinfection make them particularly attractive in active endocarditis. This is especially true when the infection involves the annulus, aortic root, or RV-PA conduit, or in fungal or *S. aureus* endocarditis.[38]

Homograft implantation while associated with excellent resistance to reinfection, poses considerable technical challenges and depends on the availability. Homograft degeneration is a recognized issue, particularly in younger patients.

In cases of extensive infection, complete avoidance of foreign material is theoretically ideal but in practice, this is rarely achievable. Even in aggressive debridement scenarios, prosthetic materials such as bioprosthetic valves or conduits may be required to restore anatomical continuity and function. In these situations, every effort should be made to minimize prosthetic burden, select infection-resistant materials, and ensure meticulous implantation technique.

Ultimately, anatomic radicality must be balanced with clinical pragmatism. While the goal is to avoid prosthetic material in infected fields, leaving residual lesions after an insufficient repair is often more dangerous than definitive valve or conduit replacement. The surgeon's judgment must prioritize long-term safety over theoretical ideals. Moreover, delaying surgery in high-risk but operable patients is associated with increased mortality underscoring the need for early, confident intervention by experienced congenital surgical teams.

Intraoperative support and complications

Intraoperative instability is common, driven by septic shock, coagulopathy, vasoplegia, and preexisting physiologic derangements particularly in patients with Fontan physiology, single ventricles, or pulmonary hypertension. Vasopressors are frequently

needed, and CPB strategies must be tailored to preserve end-organ perfusion and prevent right heart failure.

Mechanical circulatory support may be considered postoperatively in select cases of biventricular failure or prolonged arrest. However, its use in an infected field remains controversial and requires institutional expertise and strict exit strategies.

Conclusions

IE in ACHD patients remains a particularly complex and high-risk condition, due to prior surgeries, prosthetic materials, and unique anatomical challenges. Despite these difficulties, studies suggest that with timely diagnosis, well-planned surgical intervention, and tailored antimicrobial therapy, outcomes can be significantly improved. Key management principles include:

- Early and accurate diagnosis using TEE, blood cultures, and advanced imaging modalities such as FDG-PET/CT when needed.
- Timely surgery, balancing urgency against neurological and hemodynamic status.
- Complete removal of infected tissue, followed by appropriate reconstruction.
- Prolonged, targeted antibiotic therapy, based on culture results.
- Lifelong specialist follow-up with attention to prophylaxis and early detection.

As the ACHD population continues to expand, consistent application of these principles in specialized centers will be essential to reduce complications and improve long-term outcomes. Reoperations may be challenging, but when done properly, they offer patients a real chance at sustained recovery.

Ethical approval

None declared.

Conflict of interest

The authors declare that they have no conflict of interest.

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