Current options and recommendations for the treatment of aortic arch diseases

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Background: Diseases of the aortic arch represent some of the most complex conditions in cardiovascular medicine due to the segment's unique anatomy and its involvement in cerebral perfusion. Although isolated aortic arch pathology is relatively uncommon, the arch is frequently affected as part of more extensive thoracic aortic disease, including aneurysms, acute and chronic aortic dissections as well as penetrating aortic ulcers. The close relationship of the arch to the supra-aortic vessels and adjacent mediastinal structures contributes to the significant morbidity and mortality associated with these conditions and complicates therapeutic decision-making.

Methods: This review provides a comprehensive overview of the spectrum of diseases involving the aortic arch and summarizes contemporary treatment strategies. Particular emphasis is placed on anatomical considerations and classification systems, including the Ishimaru zones and variations in aortic arch morphology, which are essential for standardized reporting and procedural planning. Key pathological entities discussed include aortic arch aneurysms, acute and chronic dissections involving the arch, and residual aortic dissection following repair of type A dissection.

Results: Current treatment options encompass guideline-directed medical therapy, open surgical reconstruction, and evolving endovascular approaches. Open aortic arch replacement, often performed with hypothermic circulatory arrest and selective antegrade cerebral perfusion, remains the reference standard for patients with acceptable operative risk due to its durability and favorable long-term outcomes. In parallel, technological advances have enabled the development of branched thoracic endovascular aortic repair (B-TEVAR) and fenestrated thoracic endovascular aortic repair (F-TEVAR) devices, including both custom-made and off-the-shelf solutions, expanding treatment possibilities for patients at high surgical risk.

Conclusions: Careful patient selection based on anatomical characteristics, disease acuity, comorbidities, and institutional expertise is essential to optimize outcomes. Contemporary management of aortic arch disease therefore requires an individualized, multidisciplinary approach integrating both open and endovascular strategies.

Keywords: Aortic arch; endovascular arch repair; total arch repair; frozen elephant trunk (FET)

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Introduction

Diseases of the aortic arch represent some of the most complex and challenging conditions in cardiovascular medicine (1,2). Although isolated pathologies limited exclusively to the aortic arch are relatively rare, the arch is frequently involved as part of more extensive thoracic aortic disease (1). Owing to its unique anatomy and the presence of supra-aortic branch vessels supplying the brain, pathological processes affecting the aortic arch are associated with substantial morbidity and mortality, making their management of particular clinical importance (3,4).

The spectrum of aortic arch pathologies is broad and encompasses both acute and chronic conditions. These include acute and chronic aortic dissections, degenerative and post-dissection aneurysms, and atherosclerotic disease such as penetrating aortic ulcers (1). In many patients, these entities extend beyond the arch and involve adjacent segments of the ascending or descending thoracic aorta, further increasing therapeutic complexity and necessitating individualized treatment strategies (1).

Over recent decades, the treatment of aortic arch diseases has undergone substantial refinement (5,6). Advances in open surgical techniques, including improved cerebral protection strategies and hybrid approaches, have significantly enhanced procedural safety (7,8). In parallel, the rapid evolution of endovascular technologies has expanded the therapeutic armamentarium, enabling less invasive solutions for selected patients and anatomical scenarios (6). As a result, treatment selection now requires careful consideration of anatomical characteristics, disease acuity, patient comorbidities, and institutional expertise.

This article aims to provide a comprehensive overview of pathologies involving the aortic arch and to discuss current treatment options and recommendations. By reviewing both open and endovascular approaches, we seek to highlight optimal management strategies for specific anatomical and clinical scenarios and to support informed decision-making in the contemporary treatment of aortic arch disease.

Thoracic aortic pathologies involving the aortic arch

The thoracic aorta is anatomically divided into the aortic root, ascending aorta, aortic arch, and descending thoracic aorta. Among these segments, the aortic arch represents an anatomically limited yet clinically critical portion of the thoracic aorta, defined by the origins of the supra-aortic vessels. Despite its short length, the aortic arch plays a central role in complex thoracic aortic disease, frequently being involved as part of more extensive aortic pathology rather than as an isolated segment (1).

Anatomical classification and reporting standards

To standardize anatomical descriptions and facilitate precise communication in both clinical practice and research, the use of the Ishimaru zoning system is recommended. This classification divides the aorta into 12 zones (0–11 zones), enabling accurate reporting of disease extent, treatment coverage in open and endovascular repairs, extension of aortic dissections, and localization of entry tears or communications between the true and false lumens (1,9). In particular, the Ishimaru zones are indispensable for describing pathology and therapeutic strategies involving the aortic arch and adjacent segments.

In addition to longitudinal zoning, the configuration of the aortic arch itself has important anatomical and hemodynamic implications. Aortic arch anatomy is commonly categorized into types I, II, and III based on the vertical origin of the supra-aortic vessels (10). In a type I arch, all branch vessels originate at the same horizontal level. In type II, the innominate artery originates between the horizontal planes of the outer and inner curvatures of the arch, whereas in a type III arch, the innominate artery arises below the inner curvature. Notably, type III aortic arches are associated with abnormal helical flow patterns and have been shown to occur with increased prevalence in patients with type B aortic dissection, suggesting a potential mechanistic link between arch geometry and disease development (10).

Aortic arch aneurysm

Aneurysms of the aortic arch represent a relatively uncommon manifestation of thoracic aortic aneurysmal disease, with only a small proportion occurring exclusively within the arch (11) (*Figure 1A,1B*). More frequently, arch aneurysms develop as part of more extensive thoracic aortic disease. The aortic arch is anatomically defined by the origins of the supra-aortic vessels, and even modest dilation in this region may have significant clinical implications due to its proximity to critical mediastinal structures.

The clinical presentation of aortic arch aneurysms is heterogeneous. While many patients remain asymptomatic, symptoms may occur independently of aneurysm size.

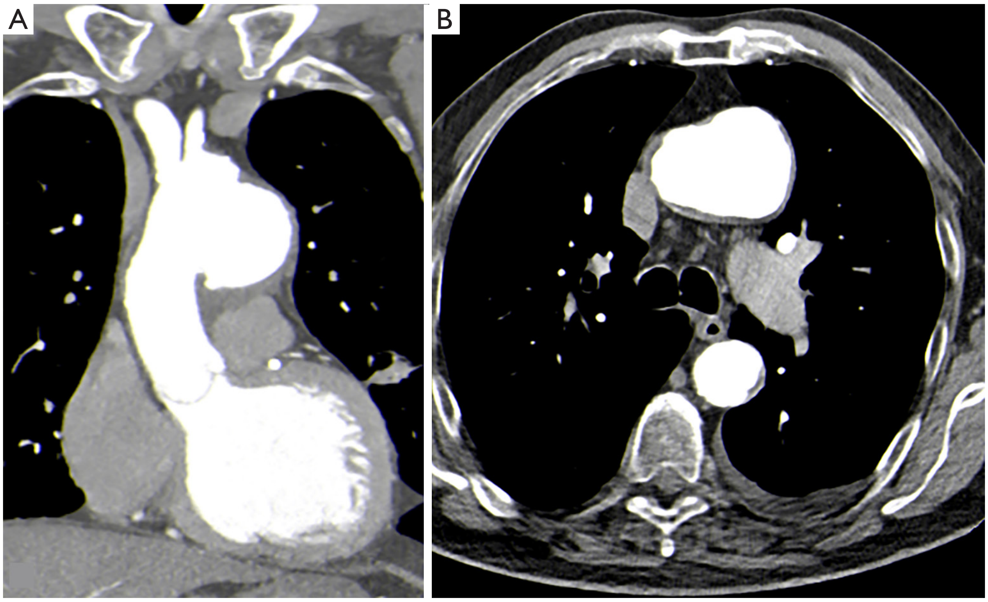

Figure 1 Preoperative computed tomography angiography of the thoracic aorta. (A) Coronal reconstruction demonstrating aortic arch pathology involving the ascending aorta and arch. (B) Axial view confirming the extent of the lesion and its relationship to the adjacent mediastinal structures.

Compression or displacement of adjacent structures can lead to dyspnea from tracheal involvement and dysphagia due to esophageal compression. In contrast to descending thoracic aneurysms, pain is a less consistent feature of arch aneurysmal disease (11).

In patients with clinically evident symptoms of aortic arch disease, aortic arch repair is recommended regardless of the size of the aneurysm. For patients with isolated aortic arch aneurysms who do not exhibit symptoms, surgical repair should be considered when the aneurysm diameter reaches or exceeds 55 mm (1).

Acute aortic dissection involving the aortic arch

Acute aortic dissection frequently involves the aortic arch, either as a primary site of intimal disruption or as a conduit for propagation between the ascending and descending thoracic aorta. Arch involvement adds substantial anatomical and prognostic complexity and is not fully captured by traditional Stanford or DeBakey classifications, underscoring the importance of contemporary descriptive frameworks in terms of the type-entry-malperfusion (TEM) classification (12).

In acute type A dissection, the aortic arch may be involved through extension of the dissection flap, direct intimal disruption within the arch, or pre-existing or concomitant aneurysmal dilation. An entry tear located in the arch represents a distinct morphological pattern, often associated with complex true- and false-lumen relationships within the supra-aortic vessels and malperfusion (12). In addition, enlargement of the arch, commonly referenced when the diameter exceeds 45 mm, reflects advanced disease and is associated with an increased likelihood of persistent false-lumen flow and later aneurysmal degeneration (13).

Acute type B aortic dissection is defined by the absence of ascending aortic involvement; however, potential treatment options frequently need to extend proximally into the aortic arch. In some patients, the arch plays a dominant role in the disease process due to the proximity of the primary entry tear to the supra-aortic vessels or due to unfavorable arch anatomy and geometry (14).

A subset of acute aortic dissections is characterized by an entry tear located in the aortic arch, with extension into the descending thoracic aorta but without involvement of the ascending aorta. Nowadays described as non-A non-B dissections, these patterns were not explicitly addressed in earlier classification systems (12). Clinically, this dissection phenotype is associated with a high incidence of adverse features, including malperfusion syndromes, rapid aortic expansion, and early rupture (12). The arch location of

Figure 2 Postoperative computed tomography angiography following total aortic repair and replacement. (A) Three-dimensional volume rendering demonstrating the FET graft with incorporation of the supra-aortic branches and the distal extension into the descending thoracic as well as abdominal aorta. (B) Maximum intensity projection confirming patency of the supra-aortic and thoracoabdominal branches with preserved distal aortic perfusion. FET, frozen elephant trunk.

the entry tear contributes to complex flow patterns and pressurization of the false lumen, which may explain the unfavorable natural history reported in earlier observational series.

Residual aortic dissection after previous type A repair

Residual aortic dissection following surgical repair of acute type A dissection represents one of the most common substrates for late aortic arch pathology. After initial repair, the dissection frequently persists in the arch and descending thoracic aorta, where ongoing false-lumen perfusion may drive negative aortic remodeling (15).

Two major pathological mechanisms dominate in this setting. The first is progressive diameter enlargement of the residual dissected aortic arch, often occurring over years and reflecting chronic false-lumen pressurization (15). The second is the development of new intimal disruptions at the distal anastomosis, commonly referred to as distal anastomotic new entry tears (16). These lesions can re-establish or augment false-lumen flow and are strongly associated with accelerated aneurysmal degeneration of the arch and distal thoracic aorta. In this scenario, subsequent, and sometimes multiple, distal reinterventions are common (*Figure 2A,2B*).

Treatment options for aortic arch pathologies

Treatment of aortic arch pathologies comprises various modalities ranging from guideline-directed medical therapy to endovascular aortic arch repair, including varying extents of supra-aortic debranching to create a sufficient proximal sealing zone, and up to open surgical partial or total aortic arch replacement using hypothermic circulatory arrest (1). While guideline-directed medical therapy is beneficial as adjunctive therapy to endovascular or open procedures, its durable long-term preventive effect in reducing aortic events is limited. Nevertheless, it plays an important role in

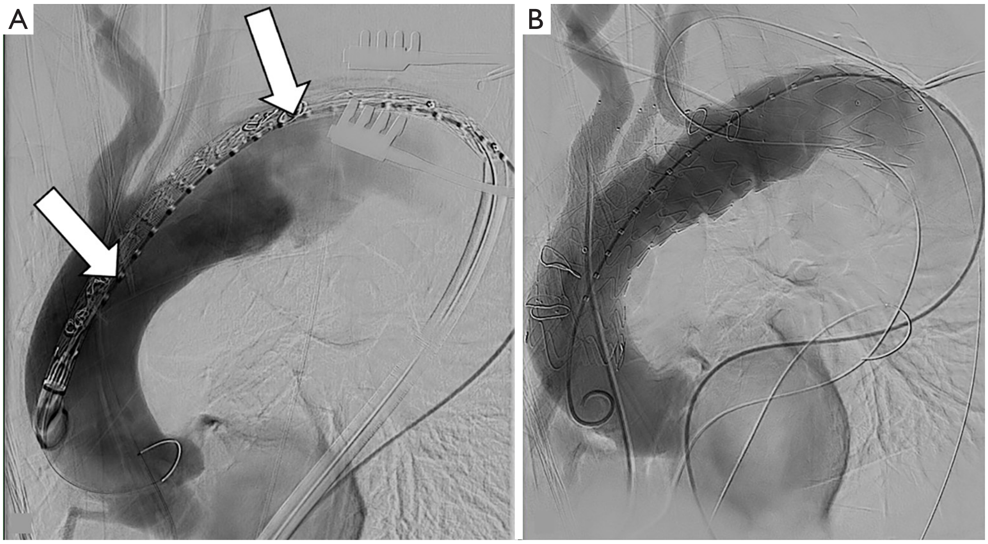

Figure 3 Pre- and post-procedural angiography of total endovascular branched aortic arch repair using the Concave EndoArch device. Pre-implantation angiography demonstrates device positioning landmarks, while final angiography confirms successful exclusion of the aortic arch pathology and patency of all supra-aortic branches through dedicated bridging stent grafts. (A) Angiography before implantation of a branched endovascular arch device (Concave, Shenzhen, China) using a Lunderquist guidewire (Cook Medical, Bloomington, IN, USA) without crossing the aortic valve. The arrows indicate the radiopaque ring markers marking the proximal and distal ends of the tunnel housing the inner branches for the supra-aortic vessels. (B) Final angiography following total endovascular branched aortic arch repair using a branched arch device with two antegrade inner branches for the innominate artery and left common carotid artery, and one retrograde branch for the LSA. The endoarch device incorporates dedicated low-profile, self-expanding bridging stent grafts. LSA, left subclavian artery.

blood pressure control and pain relief.

Endovascular aortic arch repair

Branched thoracic endovascular aortic repair (B-TEVAR)—custom-made solutions

The expertise gained from branched endografts used in the treatment of thoracoabdominal aortic pathology has been transferred to the endovascular treatment of the aortic arch. Currently, custom-made stent grafts with inner branches are used. After implantation of the main body, these devices allow continuous antegrade blood flow to the supra-aortic vessels, facilitating straightforward cannulation of the branches and implantation of bridging stent grafts (17).

Examples of devices with inner branch designs include the Cook Arch Branched Endograft (Cook, Bloomington, IN, USA) and the Relay Branch (Terumo Aortic, Sunrise, FL, USA) (17,18).

Implantation of a B-TEVAR can be performed with either two or three branches. The third branch for the left subclavian artery (LSA) represents a retrograde branch (19).

Another option for B-TEVAR in the treatment of the aortic arch is the device from Lifetech (Shenzhen, China), which can also be manufactured with three branches. In addition, this prosthesis contains a nitinol support mesh along the greater curvature of the aorta to enhance stabilization (19) (*Figure 3*).

B-TEVAR—off-the-shelf solutions

In addition to custom-made B-TEVAR devices for the aortic arch, branched endografts that are immediately available are now on the market.

One example is the Gore Thoracic Branch Endoprosthesis (Gore Medical, Newark, DE, USA), which allows landing in zone 2 without requiring a bypass from the left common carotid artery (LCCA) to the LSA. It can also be used for zone 0 landing after transposition of the LCCA and LSA to the brachiocephalic trunk (BCT). In this system, the branch for the LSA is implanted using an additional guidewire introduced into the LSA (20).

Another prosthesis with an external branch for the LSA is the Castor stent graft (MicroPort Endovastec, Shanghai, China), which also permits landing in zone 2 without the need for an LCCA-LSA bypass.

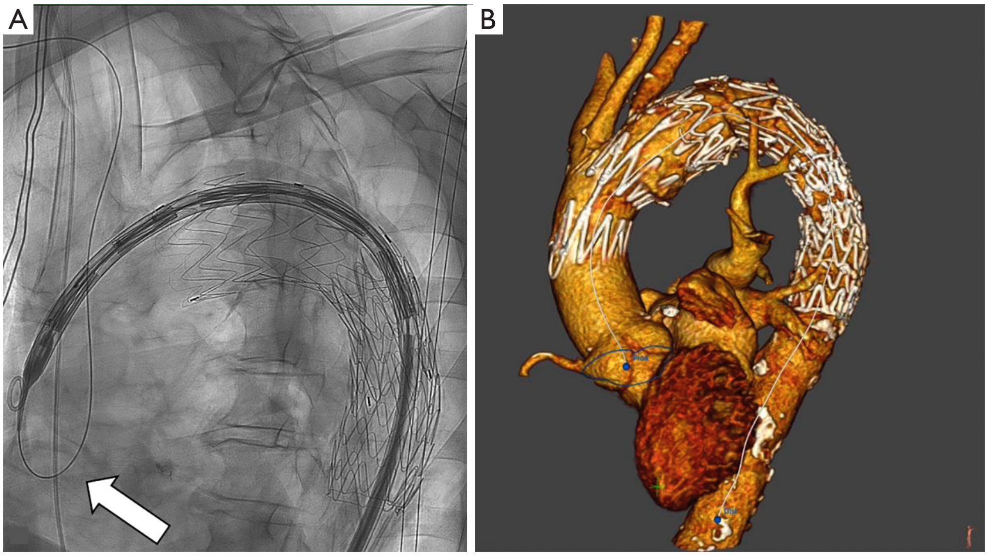

Figure 4 Fenestrated thoracic endograft implantation for total endovascular aortic arch repair. (A) High-definition angiography showing the implantation of a fenestrated thoracic endograft (Najuta, Kawasumi Laboratories, Tokyo, Japan) with the use of a loop over the aortic valve without crossing it. The arrow indicates the soft guidewire, which does not require crossing the aortic valve. (B) A three-dimensional reconstruction after endovascular aortic arch repair with a fenestrated endograft without the use of bridging stent-grafts.

An additional option for the aortic arch is the bimodular Nexus stent graft system (Artivion EMEA, Hechingen, Germany). This device consists of two components: a stent graft for the ascending aorta and a second component for the aortic arch with an external branch for the BCT (21).

Fenestrated thoracic endovascular aortic repair (F-TEVAR)—custom-made solutions

Treatment with F-TEVAR is particularly suitable for lesions located along the lesser curvature of the aorta. Compared with B-TEVAR, this technology is associated with shorter procedural times and fewer manipulations of the supra-aortic vessels. Consequently, the literature reports a lower incidence of stroke in this patient cohort (22).

Various prosthesis configurations with fenestrations and scallops have been developed, allowing landing between zones 0 and 2, thereby achieving a longer proximal landing zone.

Stent grafts from Cook Medical and Terumo Aortic incorporate fenestrations; however, these still require additional bridging stent grafts.

Terumo Aortic also offers a prosthesis with a large scallop, in which part of the graft fabric is removed along the greater curvature of the aorta to accommodate the origins of the supra-aortic vessels.

Another option for F-TEVAR in the aortic arch is the Najuta stent graft (Kawasumi Laboratories, Tokyo, Japan). This endoprosthesis has a unique design consisting of an endoskeleton covered with polytetrafluoroethylene (PTFE) and includes fenestrations for the supra-aortic vessels without the need for bridging stent grafts (23).

The device requires a different implantation technique, in which passage across the aortic valve is not necessary (*Figure 4*). In the Najuta procedure, a through-and-through wire is established between the common femoral artery and the right brachial artery. Using a loop positioned at the level of the aortic valve, optimal adaptation of the stent graft to the lesser curvature of the aorta can be achieved (23).

F-TEVAR—off-the-shelf solutions

One off-the-shelf option is *in-situ* fenestration, which is performed antegrade after deployment of the stent graft. Several techniques are available for creating the fenestration, including laser fenestration or needle fenestration (24) (*Figure 5*).

In-situ fenestration may be considered in patients who are not suitable for open surgical repair and who cannot wait for a custom-made device due to medical urgency.

However, it should be noted that this technique intentionally damages the graft fabric during fenestration, after which a bridging stent graft is implanted through the created defect. Over the long term, this may lead to

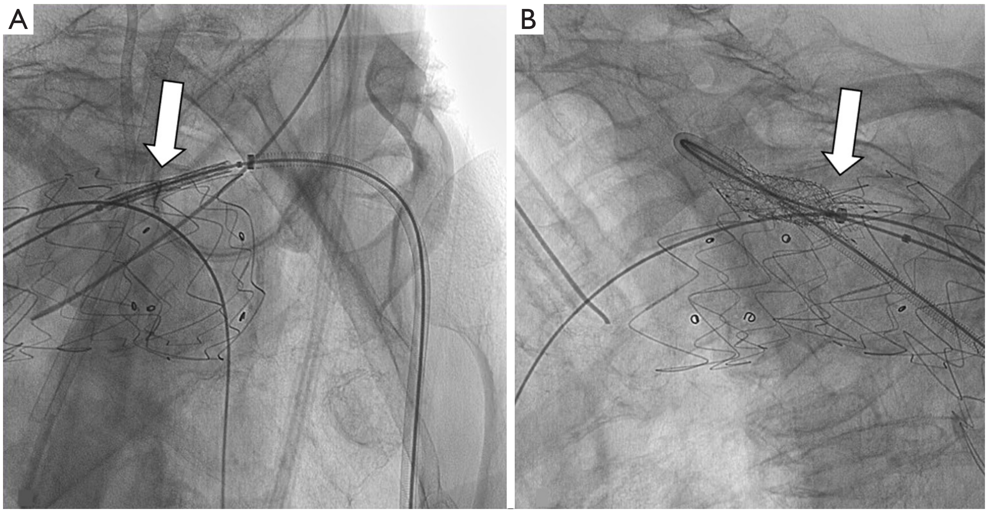

Figure 5 Guide wire-induced *in-situ* fenestration. (A) The arrow indicates the positioned bridging stent-graft through the *in-situ* fenestration. (B) The arrow indicates the flaring of the bridging stent-graft in the area of the *in-situ* fenestration.

structural failure at the connection sites. Currently, evidence regarding the long-term outcomes of this technique remains limited (25).

Open aortic arch replacement

Open surgical replacement of varying extents of the aortic arch remains the standard of care for the majority of aortic pathologies involving the aortic arch in patients who are considered operable. Patients with aortic arch aneurysms rarely present with isolated arch disease; instead, adjacent proximal and/or distal aortic segments are commonly involved. The proximal aortic arch may be affected in ascending aortopathies and is classified as the ascending extended phenotype. This phenotype is associated with stenotic right-noncoronary cusp fusion type bicuspid aortic valve (26-28). In this specific scenario, hemiarch replacement with an open distal anastomosis is sufficient in the majority of cases.

If aortic wall quality is unsatisfactory or the aortic diameter at the intended distal anastomotic site is ≥45 mm, extension of the repair to a partial arch replacement, including reimplantation of one or more supra-aortic vessels, is indicated (1). When this diameter threshold is exceeded at the level of the distal aortic arch or proximal descending aorta, total aortic arch replacement with antegrade stent-graft implantation into the descending aorta is recommended. This so-called frozen elephant trunk (FET) technique is particularly beneficial in patients with mega-aortic syndrome and extensive thoracic or thoracoabdominal aortic dilation, as it facilitates subsequent endovascular or open distal aortic interventions (29).

In complex cases, staged treatment of the downstream aortic segment using TEVAR extension, followed by open or endovascular thoracoabdominal replacement or repair, may reduce overall surgical trauma and facilitate preconditioning of the intraspinal and paraspinal collateral network, thereby lowering the risk of symptomatic spinal cord injury (29). Outcomes of the FET technique for thoracic aneurysms are generally favorable; however, stroke rates remain higher compared with other chronic settings, such as residual aortic dissection following previous type A repair (30,31).

Open versus endovascular aortic arch treatment

Open aortic arch replacement is indicated in patients with acceptable operative risk profiles, anatomies amenable to open reconstruction, and anticipated longevity sufficient to benefit from the durability of surgical repair. This strategy is preferentially applied in younger patients, those with low to intermediate perioperative risk, and individuals with connective tissue disorders or extensive and complex arch pathology. The procedure requires cardiopulmonary bypass with hypothermic circulatory arrest and adjunctive selective antegrade cerebral perfusion to ensure neurologic protection. Operative duration is considerable, with median times exceeding seven hours, and perioperative blood transfusion is routinely required. Postoperative recovery is associated with prolonged mechanical ventilation, extended intensive care unit admission, and longer overall hospitalization. In elective cases, early mortality remains acceptable, typically ranging from 2.5% to 6%, while perioperative stroke rates are reported at approximately 5–6%. Long-term outcomes favor open repair, with superior overall survival, durable exclusion of arch pathology, low rates of aorta-related mortality, and infrequent need for secondary interventions (23,32-34).

Total endovascular aortic arch repair is generally reserved for patients deemed high or prohibitive risk for conventional open surgery, most commonly elderly individuals with substantial comorbidity burden and limited physiologic reserve. Patient selection is highly dependent on detailed assessment of arch morphology, supra-aortic vessel anatomy, landing zone quality, and compatibility with branched or fenestrated endograft technology, whether custom-manufactured or off-the-shelf. Compared with open reconstruction, endovascular repair is less invasive, characterized by significantly shorter procedural times, reduced transfusion requirements, and minimal need for prolonged ventilation, intensive care, or extended hospitalization. Reported technical success rates exceed 95% with contemporary custom devices. Early mortality appears comparable to open repair; however, neurologic complications, particularly stroke, may occur at higher frequencies, with rates reported up to 14%. In addition, secondary interventions are more common, driven primarily by endoleaks, branch instability, or access-related complications. While mid-term freedom from aorta-related death at 5 years is similar between approaches, overall survival is generally inferior following endovascular repair, and reintervention rates are higher, particularly in cases requiring proximal landing in zone 0 or 1 (23,32,35).

Conclusions

In summary, open aortic arch replacement remains the reference standard for appropriately selected, medically fit patients, offering durable repair and superior long-term aorta-specific outcomes. Total endovascular arch repair represents an important complementary strategy for patients at elevated surgical risk, achieving reduced perioperative morbidity at the expense of higher reintervention rates and less predictable neurologic outcomes.

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