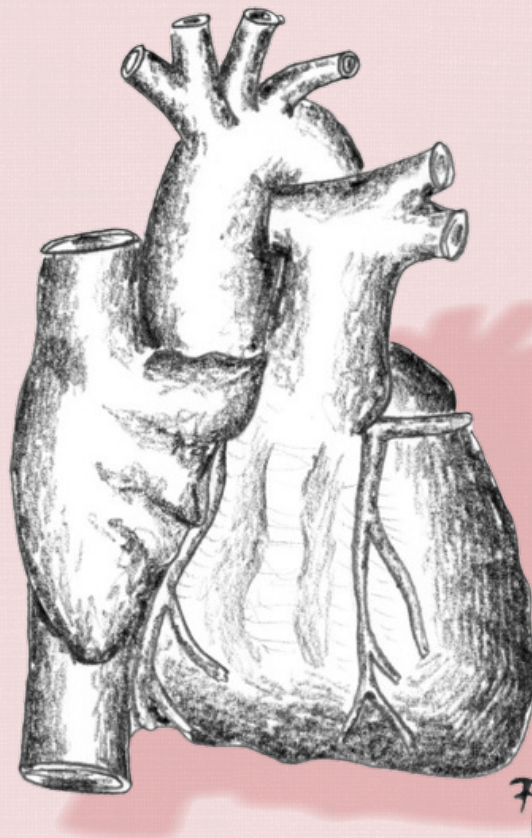


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Heart Surgery for Medical Students

Tomáš Toporcer, Adrián Kolesár



Faculty of Medicine

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Tomáš Toporcer and Adrián Kolesár

Košice 2026

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Lecture Notes

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List of Abbreviations

A1, A2, A3	- segments of the anterior mitral leaflet (A1 – anterolateral, A2 – middle, A3 – posteromedial)
AA	- axillary artery (Figure 7)
AA	- aortic arch (Figure 28)
ABO	- ABO blood group system
ACC	- American College of Cardiology
ACE	- angiotensin-converting enzyme
ACS	- acute coronary syndromes
ACT	- activated clotting time
AHA	- American Heart Association
AHF	- advanced heart failure
Ao	- aorta
AoS	- aortic stenosis
AV	- axillary vein
AVA	- aortic valve area
aVF	- augmented vector foot (limb lead of the ECG)
aVL	- augmented vector left (limb lead of the ECG)
AVNB	- atrioventricular node branch
AVR	- aortic valve replacement
BMI	- body mass index
CABG	- coronary artery bypass grafting
CAV	- cardiac allograft vasculopathy
CB	- conus branch
CCS	- chronic coronary syndromes
CCS	- Canadian Cardiovascular Society
CCTA	- coronary computed tomography angiography
CD31	- cluster of differentiation 31 (endothelial antigen)
CD34	- cluster of differentiation 34 (endothelial antigen)
CD38	- cluster of differentiation 38
CHD	- congenital heart defects
CK	- creatine kinase
CK-MB	- MB isoenzyme of creatine kinase
CMV	- cytomegalovirus
CPB	- cardiopulmonary bypass
CRT	- cardiac resynchronization therapy
CS	- unroofed coronary sinus defect (Figure 25)

CT	- computed tomography
CTA	- computed tomography angiography
DA	- descending aorta
DB	- diagonal branch
DBD	- donation after brain death
DCD	- donation after circulatory death
DHCA	- deep hypothermic circulatory arrest
DNA	- deoxyribonucleic acid
DTAA	- descending thoracic aortic aneurysm
EACTS	- European Association for Cardio-Thoracic Surgery
EBV	- Epstein–Barr virus
ECG	- electrocardiogram
ECMO	- extracorporeal membrane oxygenation
ePTFE	- expanded polytetrafluoroethylene
ESC	- European Society of Cardiology
ET	- elephant trunk
FA	- femoral artery
FBF1	- Fas Binding Factor 1
FET	- frozen elephant trunk
FFR	- fractional flow reserve
FV	- femoral vein
GRACE	- Global Registry of Acute Coronary Events (risk score)
HCV	- hepatitis C virus
HIV	- human immunodeficiency virus
HLA	- human leukocyte antigen
HRS	- Heart Rhythm Society
HTLV	- human T-lymphotropic virus
HTx	- heart transplantation
IABP	- intra-aortic balloon pump
ICD	- implantable cardioverter-defibrillator
iFR	- instantaneous wave-free ratio
IgG	- immunoglobulin G
IHD	- ischemic heart disease
INTERMACS	- Interagency Registry for Mechanically Assisted Circulatory Support
ISHLT	- International Society for Heart and Lung Transplantation
IVUS	- intravascular ultrasound
KD	- Kommerell's diverticulum

LA	- left atrium
LAB	- left atrial branch
LAD	- left anterior descending branch
LAVB	- left atrioventricular branch
LCA	- left coronary artery
LCCA	- left common carotid artery
LCX	- circumflex branch
LITA	- left internal thoracic artery
LM	- left main coronary artery
LMB	- left marginal branch
LSA	- left subclavian artery
LV	- left ventricle
LVA	- left vertebral artery
LVAD	- left ventricular assist device
LVEDP	- left ventricular end-diastolic pressure
LVEF	- left ventricular ejection fraction
LVESD	- left ventricular end-systolic dimension
LVOT	- left ventricular outflow tract
MCS	- mechanical circulatory support
MI	- myocardial infarction
MIDCAB	- minimally invasive direct coronary artery bypass
MLA	- minimal lumen area
MOMENTUM 3	Multicenter Study of MagLev Technology in Patients Undergoing - Mechanical Circulatory Support Therapy with HeartMate 3 (clinical trial)
mPG	- mean pressure gradient across the aortic valve
MR	- mitral regurgitation
MRI	- magnetic resonance imaging
mTOR	- mammalian target of rapamycin
NSTE-ACS	- non-ST-elevation acute coronary syndrome
NSTEMI	- non-ST-elevation myocardial infarction
NYHA	- New York Heart Association
OPCAB	- off-pump coronary artery bypass grafting
P1, P2, P3	- segments of the posterior mitral leaflet
PA	- pulmonary trunk (Figure 27)
pAMR	- pathologic antibody-mediated rejection
PAWP	- pulmonary artery wedge pressure
PCI	- percutaneous coronary intervention

PDB	- posterior descending branch
PGD	- primary graft dysfunction
PGE2	- prostaglandin E2
PISA	- proximal isovelocity surface area
PLB	- posterolateral branch
PMC	- percutaneous mitral commissurotomy
Prim	- primary atrioventricular defect, ostium primum (Figure 25)
PVC	- polyvinyl chloride
PVR	- pulmonary vascular resistance
Qp/Qs	- ratio of pulmonary to systemic blood flow
RA	- right atrium
RAB	- right atrial branch
RAVB	- right atrioventricular branch
RCA	- right coronary artery
RCCA	- right common carotid artery
RIM	- ramus intermedius
RIP	- ramus interventricularis posterior
RMB	- right marginal branch
RSA	- right subclavian artery
RV	- right ventricle
RVA	- right vertebral artery
RVB	- right ventricular branch
SAM	- systolic anterior motion (of the mitral valve)
SANB	- sinoatrial node branch
SAVR	- surgical aortic valve replacement
SC	- coronary sinus (Figure 27)
SCR	- supracoronary replacement
Sec	- secondary atrioventricular defect, ostium secundum (Figure 25)
SFT	- solitary fibrous tumor
STEMI	- ST-elevation myocardial infarction
SVG	- saphenous vein graft
sVi	- indexed left ventricular stroke volume
SVI	- inferior sinus venosus (Figure 25)
SVS	- superior sinus venosus (Figure 25)
SYNTAX	- scoring system quantifying the anatomical severity of coronary artery disease (Synergy between PCI with TAXUS and Cardiac Surgery)
TAAA	- thoracoabdominal aortic aneurysm
TAH	- total artificial heart

TAVI	- transcatheter aortic valve implantation
TAVR	- transcatheter aortic valve replacement (alternative term for TAVI)
TEE	- transesophageal echocardiography
TEER	- transcatheter edge-to-edge repair
TEVAR	- thoracic endovascular aortic repair
UNOS	- United Network for Organ Sharing
V1–V9	- precordial (chest) leads of the ECG
V2R–V4R	- right-sided precordial leads of the ECG
VA-ECMO	- veno-arterial extracorporeal membrane oxygenation
V-AV	- veno-arterio-venous configuration of ECMO
Vmax	- maximum velocity across the aortic valve
VSR	- ventricular septal rupture
VV-ECMO	- veno-venous extracorporeal membrane oxygenation

Preface

Cardiovascular disease remains a major cause of morbidity and mortality and one of the principal challenges of contemporary medicine. Cardiac surgery is an essential component of cardiovascular care, providing treatment for a broad spectrum of acquired and congenital heart diseases. Although highly specialized, its fundamental principles are relevant to every physician, particularly in relation to patient referral, perioperative care, recognition of complications, and long-term follow-up.

Heart Surgery for Medical Students is intended primarily for sixth-year medical students preparing for the final state examination in surgery. It provides a structured overview of the main areas of cardiac surgery, with emphasis on knowledge relevant to undergraduate medical education. The chapters cover basic pathophysiology, diagnostic principles, indications for surgical treatment, fundamental operative strategies, and clinically important aspects of postoperative management.

This English edition is a translated and revised version of the original textbook published in Slovak and is intended mainly for students enrolled in English-language medical programs. In addition to linguistic adaptation and the use of internationally accepted medical and cardiac surgical terminology, the text has been updated to reflect current concepts in cardiovascular medicine and cardiac surgery.

During preparation of this edition, selected chapters were reviewed and revised in accordance with current international clinical practice guidelines, including relevant ESC/EACTS and ACC/AHA/HRS recommendations. The structure of the textbook was also modified by introducing a new chapter dedicated to heart transplantation. This chapter incorporates and expands material that, in the original Slovak edition, was partly included within the section on mechanical circulatory support, thereby providing a more coherent presentation of advanced surgical treatment of end-stage heart failure.

The aim of this textbook is to offer a concise, systematic, and clinically oriented introduction to cardiac surgery and to support students in their preparation for the final state examination in surgery, while also providing a foundation for understanding the role of cardiac surgery in contemporary multidisciplinary cardiovascular care.

the authors

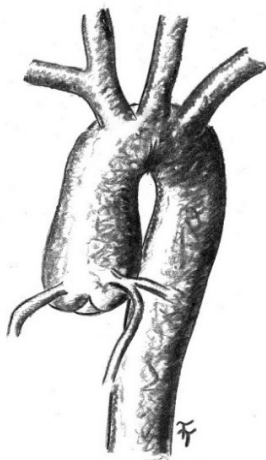


Figure 1 Sketch of the aorta.

1 Anatomy of the Heart (Tomáš Toporcer)

Functionally, the heart consists of four chambers. Physiologically, deoxygenated blood flows through the right-sided chambers and oxygenated blood through the left-sided chambers. Blood flows into the right atrium from the upper half of the body via the superior vena cava and from the lower half of the body via the inferior vena cava. In addition, deoxygenated blood from the heart itself drains into the right atrium as the coronary sinus (formed by the confluence of the great cardiac vein, the middle cardiac vein, and the small cardiac vein) along with several smaller veins from the myocardium. From the right atrium, blood flows across the tricuspid valve - valva atrioventricularis dextra seu tricuspidalis - into the right ventricle, from where it is ejected through the pulmonary valve - valva (trunci) pulmonalis - into the pulmonary trunk. After oxygen saturation in the lungs, blood reaches the left atrium, usually via four pulmonary veins. From the left atrium it flows across the bicuspid atrioventricular valve, called the valva atrioventricularis sinistra seu bicuspidalis seu mitralis, into the left ventricle. From the left ventricle, blood is then ejected through the aortic valve - valva aortae - into the ascending aorta (Figure 2).



Figure 2 Borders (margins) of the heart

Several clinically important structures are located in close proximity to the heart. Of particular relevance are the arteries of the chest wall, which can be used as conduits for coronary revascularization. The left internal thoracic artery is the arterial graft of first choice, whereas the right internal thoracic artery may also be used, particularly in younger patients, although bilateral internal thoracic artery harvesting may be associated with an increased risk of impaired sternal

wound healing. The phrenic nerves course along both sides of the pericardium and may be injured iatrogenically during cardiac surgery, particularly when their anatomical course is atypical, resulting in unilateral or, rarely, bilateral diaphragmatic paresis. At the level of the distal aortic arch, the vagus nerve and the left recurrent laryngeal nerve are of particular surgical importance; injury to the recurrent laryngeal nerve, especially during aortic arch procedures, may result in postoperative vocal cord paresis. Awareness of the close relationship between the heart and the surrounding body cavities is also essential, as these may be opened intentionally or inadvertently during cardiac surgery. This most commonly involves the pleural cavities and, less frequently, the peritoneal cavity.

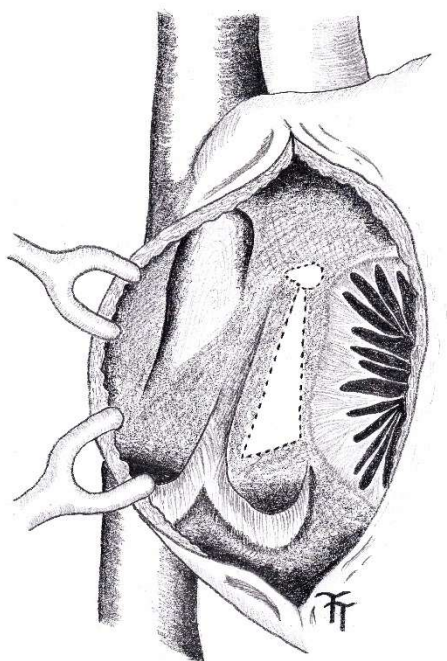


Figure 3 Anatomical location of Koch's triangle (view into the right atrium) (Koch's triangle and the atrioventricular node are marked with a dashed line and a white field)

The right atrium lies anterior and to the right of the left atrium and is generally readily accessible to the surgeon. Systemic venous blood enters the right atrium through the superior and inferior venae cavae. At the orifice of the inferior vena cava, a variably developed Eustachian valve may be present. The sinoatrial node is located near the junction of the superior vena cava and the right atrium, whereas the atrioventricular node lies within the inferior part of the interatrial septum. The atrioventricular node and the adjacent conduction tissue are particularly important from a surgical perspective, as iatrogenic injury may result in atrioventricular block. The tricuspid valve consists of three leaflets. The septal leaflet is attached along the interventricular septum, where the fibrous support of the annulus limits its tendency to dilate. Koch's triangle, which contains the atrioventricular node at its apex, is located adjacent to the septal leaflet (Figure 3). The large anterior

leaflet and the smaller posterior leaflet are attached to the more compliant portion of the tricuspid annulus along the free wall of the right ventricle. Consequently, right ventricular dilatation and chronic volume or pressure overload primarily result in annular dilatation along the anterior and posterior portions of the tricuspid annulus.

The right ventricle lies anterior to the left ventricle and therefore forms much of the anterior surface of the heart, making it particularly vulnerable to penetrating chest trauma and certain deceleration injuries. Compared with the left ventricle, the right ventricular wall is considerably thinner, reflecting the substantially lower pressures within the pulmonary circulation. This lower pressure load is associated with less mechanical stress on the right-sided heart valves and may contribute to their generally slower structural degeneration. Biological valve prostheses and homografts implanted in the right heart also tend to degenerate more slowly than comparable prostheses in the systemic circulation. In contrast, mechanical prosthetic valves in the right heart are associated with a higher risk of thrombosis, largely because of the lower pressure gradients and slower blood flow compared with the left-sided circulation. The pulmonary valve is a tricuspid semilunar valve separating the right ventricular outflow tract from the pulmonary trunk. The pulmonary trunk subsequently divides into the right and left pulmonary arteries, which carry deoxygenated blood to the lungs.

The left atrium lies posterior to the right atrium and superior to the left ventricle. Four pulmonary veins usually drain into it, with two veins arising from each lung. Anatomical variants are common; for example, the pulmonary veins from one lung may form a common trunk before entering the left atrium, thereby reducing the number of separate pulmonary venous ostia. The interatrial groove is commonly used for surgical access to the left atrium, allowing direct entry without opening another cardiac chamber. Alternative approaches may be performed through the right atrium and the interatrial septum. Given the anatomical proximity and the risk of inadvertently opening the right atrium, separate cannulation of the superior and inferior venae cavae is recommended for mitral valve procedures, allowing the atria to be excluded from the circulation during cardiopulmonary bypass and providing a bloodless operative field. The left atrial appendage is located on the left side of the heart, adjacent to the pulmonary trunk. In patients with atrial fibrillation, it represents the principal site of intracardiac thrombus formation and an important source of systemic thromboembolism. The circumflex branch of the left coronary artery courses in close proximity to the base of the left atrial appendage. The mitral valve consists of two leaflets: the larger anterior leaflet is attached to approximately one third of the mitral annulus, whereas the posterior leaflet is attached to the remaining two thirds. The coronary sinus and the circumflex branch of the left

coronary artery run in close proximity to the posterior mitral annulus and are therefore important anatomical structures during mitral valve surgery.

The left ventricle normally has a conical geometry that depends in part on the integrity of the mitral valve apparatus, including the papillary muscles and chordae tendineae. Excision of these structures alters left ventricular geometry and may adversely affect ventricular function. The anterolateral papillary muscle usually has a dual blood supply, most commonly from branches of the left anterior descending and circumflex coronary arteries, which provides a degree of protection against ischemic injury. In contrast, the posteromedial papillary muscle generally has a single dominant arterial supply, arising from either the right coronary artery or the circumflex artery depending on coronary dominance, making it more vulnerable to ischemia and infarction. The aortic valve separates the left ventricular outflow tract from the ascending aorta and consists of three cusps. The left and right coronary arteries arise from the corresponding aortic sinuses. The region adjacent to the non-coronary cusp forms part of the aortomitral continuity, where the aortic and mitral valves are in close fibrous continuity. Infective or destructive processes in this region may extend into the intervalvular fibrosa and can lead to complications such as abscess formation or fistulous communication. Near the commissure between the non-coronary and right coronary cusps lies the membranous septum, in close proximity to the atrioventricular conduction axis and the bundle of His (Figure 4). Injury to this region during surgery or as a result of pathological processes may lead to atrioventricular block. Tissue edema following aortic valve intervention may also cause transient atrioventricular conduction disturbances even in the absence of direct mechanical injury to the conduction system. The membranous septum is anatomically related to the attachment of the septal leaflet of the tricuspid valve and is divided into atrioventricular and interventricular portions. Injury to this region, particularly during transcatheter or surgical aortic valve procedures, may result in an abnormal communication between the left ventricular outflow tract and the right atrium or, less commonly, the right ventricle.

The proximal aorta is divided into the aortic root and the ascending aorta. The aortic root extends from the aortic annulus to the sinotubular junction and contains the aortic sinuses, from which the coronary arteries arise. Distal to the sinotubular junction, the ascending aorta continues to the origin of the brachiocephalic trunk, where the aortic arch begins. The aortic arch gives rise to three major branches: the brachiocephalic trunk, the left common carotid artery, and the left subclavian artery. The vertebral arteries arise from the subclavian arteries and, together with the internal carotid arteries, contribute to the cerebral arterial circulation through the circle of Willis. The internal thoracic artery arises from the inferior aspect of the subclavian artery and descends along the inner

surface of the anterior chest wall, running parallel and lateral to the sternum on both sides. Distally, the internal thoracic artery divides into the musculophrenic and superior epigastric arteries.

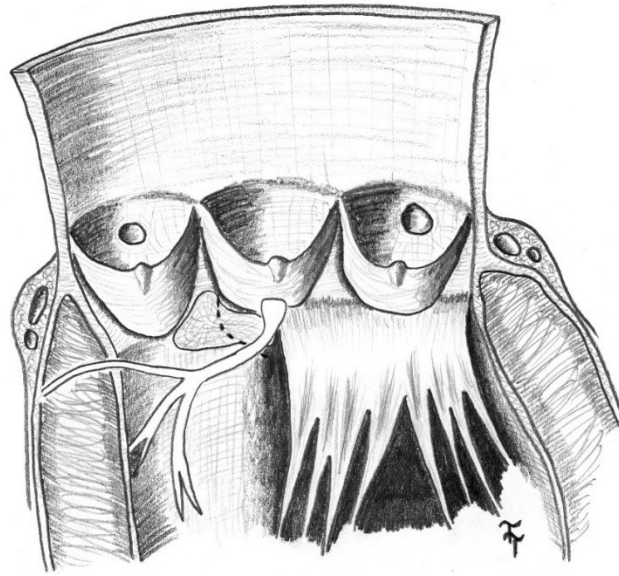


Figure 4 4 The aortic valve and its topographic anatomy in relation to surrounding structures, including the anterior leaflet of the mitral valve, the membranous septum, and the bundle of His (incision made between the right and left coronary cusps; the dashed line indicates the origin of the tricuspid valve).

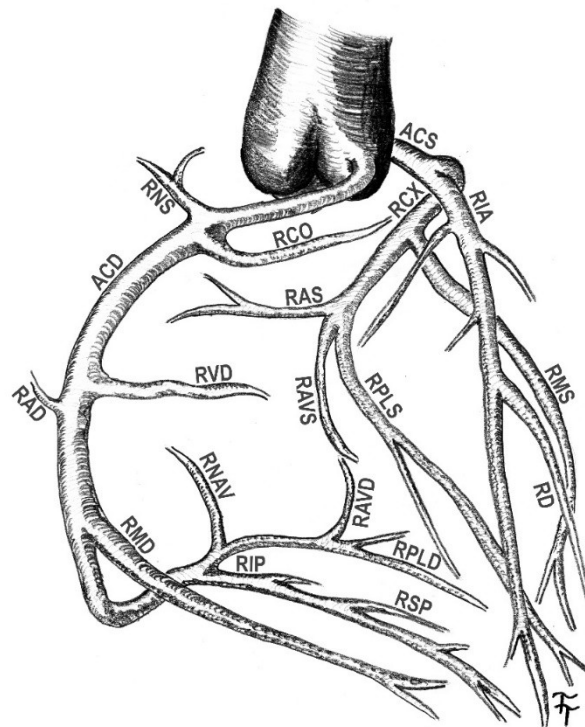


Figure 5 Diagram of coronary artery anatomy (RCA - right coronary artery; LCA - left coronary artery; RAB - right atrial branch; LAB - left atrial branch; RAVB - right atrioventricular branch; LAVB - left atrioventricular branch; CB - conus branch; LCX - circumflex branch; DB - diagonal branch; LAD - left anterior descending branch; PDB - posterior descending branch; RMB - right marginal branch; LMB - left marginal branch; AVNB - atrioventricular node branch; SANB - sinoatrial node branch; PLB - posterolateral branch; RVB - right ventricular branch).

Although only two coronary arteries arise from the aorta, coronary artery disease is commonly described clinically as single-vessel, two-vessel, or three-vessel disease. This classification considers the right coronary artery (RCA) and the two major branches of the left coronary artery - the left anterior descending artery (LAD) and the left circumflex artery (LCX) - as separate coronary territories. Atherosclerotic disease involving the left coronary artery proximal to its bifurcation is referred to as left main coronary artery disease. In some individuals, the left main coronary artery trifurcates, giving rise to an additional vessel, the ramus intermedius (RIM), which courses between the LAD and LCX and usually supplies the lateral wall of the left ventricle. The LAD gives rise to diagonal branches supplying the anterolateral left ventricular wall and septal perforating branches supplying the interventricular septum; these branches also facilitate identification of the LAD on coronary angiography. Because the LAD supplies a large proportion of the left ventricular myocardium, proximal LAD occlusion can result in extensive myocardial ischemia and severe impairment of left ventricular function, particularly in the absence of an adequate collateral circulation. The posterior descending artery, also termed the posterior interventricular artery or ramus interventricularis posterior (RIP), runs in the posterior interventricular sulcus. It most commonly arises from the right coronary artery but may originate from the circumflex artery; accordingly, the coronary circulation is described as right-dominant or left-dominant (Table 1, Figure 5).

A more detailed description of the topographic anatomy of the heart and the surrounding structures, presented in the following chapters, extends beyond the basic anatomical knowledge required to understand the fundamental principles of cardiac surgery.

1.1 Topographic Anatomy of the Heart

Geometrically, the heart represents a three-sided pyramid situated in the middle mediastinum. The three-sidedness of the pyramid is apparent when the heart is viewed from its apex. The acute margin lies on the lower side and represents a sharp border between the sternocostal surface and the diaphragmatic surface. The obtuse margin lies higher up and, at first glance, is not as clearly identifiable. The posterior margin is not clearly anatomically defined and likewise does not represent a sharp border (Figure 2).

Topographically, two-thirds of the heart's mass lies to the left of the body's median plane. The long axis of the heart runs from the left epigastrium toward the right shoulder. The short axis corresponds to the atrioventricular junction and, in general, can be said to lie more in the vertical than in the horizontal plane.

Anteriorly, the heart is protected by the sternum and its junction with the third to fifth ribs. The lungs abut the heart on its lateral sides, where the great vessels of the pulmonary circulation enter the pulmonary hila. The right lung abuts the right side of the heart and ventrally reaches as far as the median plane. This is why, during median sternotomy, unwanted opening of the right pleural cavity occurs significantly more often than opening of the left. The diaphragmatic surface represents a fairly extensive part of the heart's surface resting on the diaphragm. Dorsally, the heart lies directly against the esophagus and the tracheal bifurcation, which poses a risk of injury to these organs during cryoablation, but also offers the potential for very high-quality imaging of cardiac structures using transesophageal echocardiography (TEE).

On a posteroanterior chest X-ray, the cardiac silhouette is visible, which under normal physiological conditions is formed, from top to bottom, on the right side by the left brachiocephalic vein, the superior vena cava, the right atrium, and the inferior vena cava, and on the left side by the aortic arch, the pulmonary trunk, the left atrial appendage, and the left ventricle. Most commonly, however, the heart is examined by echocardiography, either transthoracic or transesophageal.

The heart lies within the pericardial sac, which is directly anatomically connected to the wall of the great vessels and to the diaphragm. The simplest way to picture the pericardium is as a duplicated sac into which the heart is invaginated by its apex. The inner layer of the pericardial duplication is the lamina visceralis (epicardium), which forms the outer layer of the anatomical composition of the heart wall, covers the coronary arteries and cardiac veins, and extends several centimeters onto the wall of the great vessels. The outer wall of the pericardial duplication is the lamina parietalis, a fibrous covering of the heart that is anatomically and mechanically connected to the surrounding structures. Between these individual layers of the pericardial duplication there is, physiologically, a minimal amount of lubricating fluid allowing smooth sliding of the layers against each other with every cardiac cycle. However, the transition between the visceral and parietal pericardium does not form a single line, and additional tunnels can be anatomically defined within the pericardium. The junction of the visceral and parietal pericardium separately encircles three sets of structures. First, and most caudally, is the inferior vena cava on its own. Further dorsally, the superior vena cava and all the pulmonary veins are encircled together; these two separate passages may also be joined. And most cranially, the aorta and pulmonary artery are encircled together. Sinuses, or tunnels, thus arise between these pericardial passages: between the inferior vena cava and the lower right pulmonary vein lies the oblique sinus, which may also end as a blind pouch. Further, between the pulmonary artery and the aorta on one side, and the upper pulmonary veins and the superior vena cava on the other, lies the transverse sinus. The oblique sinus is quite often incomplete. Surgically, these sinuses can be put to good use - for example, the oblique sinus when passing a snare around the inferior vena cava, and the transverse sinus as a route for the right internal thoracic artery when it is left in its native (in situ) position and anastomosed distally to marginal branches of the circumflex branch of the left coronary artery.

Both the vagus nerve and the phrenic nerve descend through the mediastinum in close proximity to the heart. The phrenic nerve enters the thorax through the thoracic inlet, running ventrally on the surface of the anterior scalene muscle, in intimate contact with the internal thoracic artery just behind it. Because of this topographic location, injury to the phrenic nerve is one of the possible complications during dissection of the internal thoracic artery for use as an arterial graft in myocardial revascularization. The right phrenic nerve further lies in intimate contact with the lateral aspect of the superior vena cava, which also makes it vulnerable during cannulation of that vessel for cardiopulmonary bypass. Both nerves then continue to descend ventrally in front of the pulmonary hilum before entering the diaphragm to provide its innervation. The nerves are also at risk intraoperatively during wide opening of the lateral pericardium and the mediastinal pleura for the purpose of mobilizing the heart, and during pericardial incision near the pulmonary trunk when the left internal thoracic artery is being harvested for revascularization. The nerves further mark the boundary of surgical pericardial excision in the treatment of constrictive pericarditis. The vagus nerve enters the thorax dorsal to the phrenic nerve, following the carotid arteries. On the right side, the vagus nerve gives off the recurrent laryngeal nerve, which loops around the right subclavian artery and ascends back cranially, away from the region of the heart. The right vagus nerve then continues caudally, dorsal to the pulmonary hilum, where it gives off its pulmonary branches, and leaves the mediastinum in close proximity to the esophagus. The left vagus nerve crosses the aortic arch, where its branch, the left recurrent laryngeal nerve, leaves the main trunk; this branch then loops around the ligamentum arteriosum and continues cranially between the pulmonary artery and the esophagus. The further course of the left vagus nerve mirrors that on the right side, passing behind the pulmonary hilum, giving off its branches, and leaving the thorax near the esophagus caudally. A further, anatomically less prominent nerve arises from fibers of the cervicothoracic (stellate) ganglion and runs close to the subclavian artery on both sides. It can therefore be damaged during dissection around that artery. It then continues cranially, innervating the region of the head and eye. Damage to this

nerve can result in Claude-Bernard-Horner syndrome, characterized by the presence of miosis, eyelid ptosis, and enophthalmos.

1.2 Right Atrium and Tricuspid Valve

The right atrium of the heart can be divided into three basic parts: the auricula dextra (right atrial appendage), the vestibule, and the terminal portion of the inferior and superior venae cavae. On external inspection, the atrial appendage and the venous component can easily be distinguished. The boundary between these parts is defined internally by a ridge known as the crista terminalis. Externally, the sinus venarum cavarum, the site where the venae cavae drain in, is separated from the atrium proper by a groove, the sulcus terminalis. The right atrial appendage is triangular in shape, with a broad attachment to both the venous inflow portion of the right atrium and to the atrium itself. The most characteristic macroscopic morphological feature of the right atrium is the muscoli pectinati, which form a network of muscular structures. Their size varies individually, from almost imperceptible structures to thick muscle bundles that can present a mechanical obstacle when cannulating the right atrium through the atrial appendage. The venous portion of the right atrium is the part lying between the crista terminalis and the tuberculum intervenosum. Another clinically important and anatomically variable structure is the valve of the inferior vena cava, also known as the Eustachian valve, which directs the flow of blood from the inferior vena cava into the right atrium and can present a mechanical obstacle when cannulating the right atrium with a two-stage cannula.

The sinoatrial node lies at the junction of the venous inflow portion of the right atrium and the right atrial appendage, in the ventral and cranial part of the sulcus terminalis. It is thus situated, in effect, at the right or lateral margin of the junction between the superior vena cava and the right atrium. In about 10% of cases its location is less well defined and extends over a wider portion of this junction. Its arterial supply comes from the sinoatrial nodal branch, which in 55% of patients arises from the right coronary artery. In a smaller percentage of the population it arises from the circumflex branch of the left coronary artery. The supplying vessel then runs through the interatrial groove toward the junction of the superior vena cava and the atrium, though the course of the supplying artery within that junction itself is highly variable. Less commonly, the supplying artery arises from a more distal segment of the right coronary artery and then crosses the right atrial appendage, which carries a higher risk of injury to it during right atriotomy. When it arises from the circumflex branch of the left coronary artery, it instead crosses the left atrium, with a risk of injury during left atriotomy and during the approach to the mitral valve. Any incisions in the region of the atria must therefore respect these anatomical variations. Some authors report that the course of the sinoatrial nodal branch is macroscopically identifiable and that the specifics of the surgical approach should take its course into account.

The most commonly used approach to the right atrium is atriotomy through the right atrial appendage, parallel to the sulcus terminalis. After incising the wall of the right atrium, the crista terminalis can be visualized; at the site where the superior vena cava enters, it continues without interruption into the septum secundum. This, in turn, forms the upper margin of the fossa ovalis. This surgical approach allows visualization of a relatively large area between the tricuspid valve and the entry of the superior vena cava, which contains both the fossa ovalis and the outlet of the coronary sinus. The tissue forming the cranial edge of the fossa ovalis, also called the secondary septum, forms the wall between the superior vena cava and the right pulmonary veins of the left atrium. The lower margin of the fossa ovalis, in turn, marks the boundary between the entry of the inferior vena cava and the coronary sinus. The area around the entry of the coronary sinus is, in fact, the atrioventricular muscular septum, i.e., the pars muscularis of the septum atrioventriculare. Only a small part of the seemingly extensive surface of the right atrium corresponds to the interatrial septum that can be used as an approach to the mitral valve. On one side, the lower margin of the coronary sinus already corresponds to the atrioventricular groove; on the other, a large portion, more cranially and ventrally, corresponds to the anterior wall of the right atrium overlying the aortic root. The transseptal surgical approach is therefore limited to the fossa ovalis and its immediate surroundings.

The atrioventricular node of the cardiac conduction system, like the sinoatrial node, is also located in the right atrium. Knowledge of its location is extremely important in practice given the surgical procedures performed in this region. The atrioventricular node is located within Koch's triangle, which is bounded by the tendon of Todaro, the septal leaflet of the tricuspid valve, and the orifice of the coronary sinus (Figure 3). The tendon of Todaro is a fibrous structure formed by the fusion of the tissue of the valve of the inferior vena cava and the valve of the coronary sinus, i.e., the Eustachian and Thebesian valves. The bundle of His passes directly through the apex of this triangle, subsequently entering the interventricular septum, where it further divides. Knowledge of the anatomical location of the heart's two most important conduction nodes, as well as their blood supply and the adjacent nerve bundles, is essential knowledge for every cardiac surgeon. Damage to these tissues represents a significant complication of the treatment process, requiring further intervention.

The tricuspid valve is anatomically best visualized from the atrial side. The circumference of the tricuspid valve averages 11.4 cm in men and 10.8 cm in women. The transition from the annular portion of each leaflet to the free edge, where the chordae tendineae attach, is continuous, and the edge does not form a completely well-defined line. The valve itself is oriented almost vertically but forms a 45-degree angle with the sagittal plane. The annulus of the valve can be divided in a triangular fashion into anterior, posterior, and septal portions, corresponding to the adjacent leaflets of the valve.

The connective tissue around the valve annulus almost completely separates the atrial and ventricular parts of the heart, except at the site where the conduction system passes through. Subendothelial fibrous fibers extend from the right fibrous trigone into the valve annulus, partially reinforcing it. The annulus is further supplemented by looser fibroelastic tissue. The density of the annular tissue itself varies with the patient's sex and age. In general, however, the tissue surrounding the tricuspid valve is significantly less firm and dense than the corresponding components of the mitral valve. Moreover, the sites of attachment of the tricuspid leaflets do not entirely correspond, in terms of strength, to the distribution of connective tissue within the annulus.

In most cases, the individual leaflets of the tricuspid valve are easily distinguishable and are located anteriorly and superiorly (anterior leaflet), inferiorly (posterior leaflet), and septally (septal leaflet). Each leaflet consists of an endothelial duplication enclosing connective tissue that extends from the annulus and into the chordae. The leaflets of the atrioventricular valves can be divided into a rough zone, a clear (translucent) zone, and a basal zone. On the ventricular side, the rough zone is where the chordae attach to the leaflet; on the atrial side, it is the area of contact between the contralateral leaflets. The clear or smooth zone represents the body of the leaflet itself, and on its ventricular side is also the site of attachment of a few additional chordae. The basal zone consists of the 2-3 mm of the leaflet nearest the valve annulus and is characterized by a higher content of connective tissue.

The anterior leaflet is the largest component of the tricuspid valve. It is firmly attached to the atrioventricular septum and continues directly onto the membranous septum, ending at the anteroseptal commissure. Its free edge usually contains several nodules. The attachment of the septal leaflet begins at the posteroseptal commissure, continues along the posterior ventricular wall across the muscular septum, and then crosses the membranous septum to the anteroseptal commissure. The septal leaflet defines one side of Koch's triangle, the location of the atrioventricular node. The posterior leaflet attaches to the diaphragmatic wall of the atrioventricular junction.

The chordae tendineae are fibrin-collagen structures that support the leaflets of the atrioventricular valves. In general, the chordae tendineae of the tricuspid valve can be classified according to their origin as fan-shaped or basal. Fan-shaped chordae arise from the apex of a papillary muscle and branch out in a fan pattern; they are typical particularly at the commissures and clefts of the leaflet. Basal chordae do not arise from the papillary muscles but from the ventricular wall, and run toward the basal zone of the leaflet. According to their site of insertion, the chordae of the tricuspid valve are divided into commissural chordae, cleft chordae, chordae inserting into the free edge of the leaflet, rough-zone chordae, and deep chordae. Commissural chordae insert into the leaflet at the site of the commissure and the adjacent leaflet. Cleft chordae are similar

but insert at sites where there are fine clefts in the leaflet. Chordae inserting into the free edge of the leaflet are usually not branched. Rough-zone chordae usually branch into three smaller chordae, one inserting into the free edge, a second into the middle of the rough zone, and a third at the boundary between the rough and clear zones of the leaflet. Sometimes the branch of a chorda that inserts at the edge of the leaflet continues subendothelially and reinforces the rough zone of the leaflet. Deep chordae are also usually branched and insert deep within the rough zone of the leaflet, sometimes reaching as far as the clear zone.

In the right ventricle there are two main papillary muscles, located anteriorly and posteriorly. On the medial side of the ventricle there is one smaller papillary muscle, together with several other inconstant muscles. The anterior papillary muscle is the largest and arises from the right anterolateral ventricular wall below the anteroposterior commissure of the valve, and has a direct anatomical relationship to the septomarginal trabecula (moderator band). The posterior, or inferior, papillary muscle arises from the myocardium below the posteroseptal commissure and is often bifid or trifid. The septal, or medial, papillary muscle is also typical and arises from the septal margin of the septomarginal trabecula. All the papillary muscles send chordae to all the adjacent portions of the tricuspid valve.

The functional complex of the tricuspid valve consists of three leaflets, the chordae, two main papillary muscles, the fibrous valve annulus, and, not least, functionally also the musculature of the right atrium and ventricle and left ventricle. The function of the valve depends on the integrity and coordinated function of each of these parts. The anterior leaflet is the largest; the posterior leaflet is characterized by multiple scallops. The septal leaflet is the smallest and arises from the region of the interventricular septum. The anterior papillary muscle predominantly supports the function of the anterior and posterior leaflets, while the medial papillary muscle mainly supports the posterior and septal leaflets. Chordae arising from the septum most often insert into the anterior and septal leaflets of the tricuspid valve. Further variable and inconstant chordae arise from the ventricular wall and insert into the leaflets. This complex and not entirely consistent mechanism between individuals is responsible, on one hand, for correct valve function, and on the other, in most cases, also for its insufficiency.

The tricuspid annulus is a complex three-dimensional structure that differs significantly from the more symmetrical annulus of the mitral valve. Studies using 3D echocardiography have mapped the shape of the tricuspid annulus in patients without tricuspid regurgitation. The authors emphasize that the functional tricuspid annulus is never a planar structure, with the posteroseptal portion lying below the plane and the anteroseptal portion above the plane of the rest of the annulus. The function of the tricuspid valve depends on a number of dynamic mechanisms of the right ventricle. The tricuspid annulus itself participates in this dynamic process in a non-negligible way and is certainly not a static component of the tricuspid valve mechanism. During each cardiac cycle, the diameter of the tricuspid annulus decreases by 19% in systole, corresponding to a 30% reduction in the area of the tricuspid valve.

The mutual interaction between the right and left ventricles plays an important role in right ventricular function. The shared interventricular septum and the considerable sharing of myocardial fibers between the right and left ventricles mean that left ventricular function affects the right ventricle not only directly through systolic motion of the interventricular septum, but also through its free wall. Experimental studies show that 20 to 40% of right ventricular systolic pressure and volume originates from contraction of the left ventricle. Furthermore, the musculature of both ventricles shares a common biochemical environment, so the nervous and hormonal, both systemic and local, control of the function of both ventricles is shared.

1.3 Left Atrium and Mitral Valve

Like the right atrium, the left atrium also has three functionally distinct parts. These parts are the auricula sinistra (left atrial appendage), the vestibule, and the venous complex. Unlike in the right atrium, here the venous complex forms the greater part of the atrium by volume, and the vestibule is not anatomically strictly separated. Furthermore, the muscoli pectinati are located exclusively in the left atrial appendage, and the wall of the vestibule, which continues directly into the region of the atrioventricular mitral valve, is relatively smooth. Given the location of the left atrium and the entry of the four pulmonary veins at its sides, this part

of the heart is surgically relatively difficult to access. The most commonly used approach is from the right side, between the interatrial groove and the entry of the right pulmonary veins. The second approach is through the dome of the left atrium. After retracting the aorta ventrally and to the right, this part of the wall is visible between the appendages of the right and left atria. When using this approach, the sinoatrial nodal branch must be respected at the point where it arises from the circumflex branch of the left coronary artery. The last approach used is through the interatrial septum from the right atrium. When the left atrium is opened, the relatively small opening of the left appendage is apparent, which, from the surgeon's viewpoint, lies to the left of the mitral valve orifice. With this approach, a large part of the left atrium goes unnoticed and remains, from the surgeon's viewpoint, below the line of incision, while the target mitral valve dominates the field of view.

The basic anatomical and functional units of the *valva atrioventricularis sinistra* (mitral valve) are analogous to those of the tricuspid valve and consist of the valve annulus, the leaflets, the chordae tendineae, and the papillary muscles. Functionally, the musculature of the entire left ventricle also belongs to the mitral valve.

The mitral orifice is a relatively precisely defined boundary between the wall of the left atrium and the origin of the mitral leaflets. In general, the diameter of the mitral annulus is smaller than that of the tricuspid annulus, and its circumference averages 9 cm in men and 7.2 cm in women. In diastole, the plane of the mitral valve lies almost vertically, forming a 45° angle with the sagittal and frontal planes. The axis of the mitral valve, in the atrioventricular direction, runs forward and to the left, slightly caudally toward the apex of the left ventricle. It nearly shares a plane with the tricuspid valve, lying posterocranial to it, and posterocaudal to the aortic valve. These three valves meet at the central fibrous body of the cardiac skeleton. On closure of the mitral valve, the leaflets meet along a single line. The mitral annulus is formed of fibrocollagenous tissue, and this fibrous component continues without interruption into the body of the leaflets themselves. This so-called cooperation between the leaflets and the annulus allows diastolic flow to be maximized while maintaining systolic competence. The valve annulus is, moreover, relatively firmly connected to the left and right fibrous trigones. The right coronary artery and the circumflex branch of the left coronary artery encircle the atrioventricular junction from both sides. The musculature of the atria and ventricles is thus separated by a layer of fibroelastic connective tissue. The anterior leaflet of the mitral valve is further connected by fibrous tissue to the left coronary and non-coronary cusps of the aortic valve.

In general, the leaflets of the mitral valve are described as a paired structure, and in the literature we also find the term "bicuspid atrioventricular valve." This term is, however, not entirely accurate and therefore less commonly used, because between the major anterior and posterior leaflets of the mitral valve we often find several small leaflets ensuring closure at the commissural sites. Given these features, some authors describe the mitral valve as a valve with a circumferential attachment of leaflets to the annulus. Despite these variations, two major commissures are recognized - the anteromedial and the posterolateral - where the anterior and posterior leaflets meet. In diastole, it is evident that the anterior leaflet represents only a quarter to a third of the mitral valve's circumference and has a semicircular or triangular shape with only minimal marginal indentations. The fibrous layer (*lamina fibrosa*) of the anterior leaflet continues without interruption from the left fibrous trigone, the right fibrous trigone, and the aortomitral continuity. The leaflet has a relatively extensive marginal area, the so-called rough zone, into which most of the chordae tendineae insert. The termination of this rough zone also represents the line of contact between the anterior and posterior leaflets in systole. The clear, translucent zone represents the part of the leaflet between the rough zone and the valve annulus, where almost no chordae tendineae insert into the leaflet, although some strands from chordae attaching at the leaflet edge do run into it. The anterior leaflet has no significant basal zone. The leaflet's attachment is located at the boundary between the inflow and outflow tracts of the left ventricle. A smooth atrial surface of the leaflet is a prerequisite for smooth, low-gradient filling of the left ventricle in diastole. Unlike the uniform anterior leaflet, the posterior leaflet usually has two or more indentations along its edge. The historical absence of a clear definition of the main commissures led to the didactic division of the posterior leaflet into several separate "scallops," even though functionally they form a single unit. Today

the posterior leaflet is defined as all the tissue lying dorsally between the anterolateral and posteromedial commissures, forming the functional counterpart to the much larger, flatter anterior leaflet. At the commissural sites, however, small so-called commissural cusps of the mitral valve are often found. For clinical localization, both the anterior and posterior leaflets are divided into three subunits. The anterior leaflet consists of segment A1, lying at the anterolateral commissure, A2 in the middle, and A3 at the posteromedial commissure. The posterior leaflet of the mitral valve is divided analogously. Each leaflet has, along its edge, a thickened rough zone where the chordae tendineae insert. Between this thickened marginal zone and the valve annulus lies a membranous, translucent zone with no chordal attachments, or only a minimal number. Two to three millimeters from the annulus, the posterior leaflet thickens again, forming the lamina fibrosa, where the basal chordae insert. The ratio of rough to translucent zone is 3:5 in the anterior leaflet and 7:5 in the posterior leaflet. Thus, in systole, more than 50% of the surface of the posterior leaflet physiologically comes into apposition with the anterior leaflet.

The chordae tendineae of the mitral valve can be divided into commissural chordae, rough-zone chordae, and basal chordae. A large proportion of rough-zone chordae branch into several strands on their way from the papillary muscle to the leaflet. Basal chordae, on the other hand, usually do not branch. There are several types of branched chordae tendineae that were described for the tricuspid valve, with analogues in the mitral valve, but given their diversity and rich variability, these subdivisions have no clinical significance. The chordae tendineae generally arise from two papillary muscles. The anterolateral papillary muscle arises from the wall of the left ventricle at the sternocostal surface, and the posteromedial muscle at the diaphragmatic surface. The chordae tendineae most often arise from the apical third of the papillary muscles. Chordae from each papillary muscle support the adjacent portion of both the anterior and posterior leaflets.

The mitral annulus is firmer than its right-sided counterpart, being formed of denser collagen. As noted, the annulus is firmly connected to the right fibrous trigone. The portion of the annulus anatomically related to this trigone is more vulnerable during surgery, given the proximity of the structures of the cardiac conduction system. The middle of the anterior mitral leaflet corresponds to the site of the commissure between the left coronary and non-coronary cusps of the aortic valve. An incision of the atrial wall at this site leads subannularly into the outflow portion of the left ventricle and is used by some authors for the purpose of enlarging the aortic annulus during aortic valve replacement. The circumflex branch of the left coronary artery also runs close to the annulus at the left half of the posterior leaflet, i.e., segments P1 and P2, and the coronary sinus runs close to the annulus at the right half of the posterior leaflet, i.e., segments P2 and P3. These structures can be endangered by excessive excision at the level of the annulus, or by placing sutures too deeply during mitral valve repair or replacement. Moreover, the more dominant the role played by the circumflex branch of the left coronary artery in myocardial supply, the more intimate its contact with the tissue of the mitral valve.

The onset of diastole is accompanied by passive opening of the mitral valve; this process is rapid, beginning as soon as the pressure in the left atrium exceeds that in the left ventricle. The initial stream of blood flows directly toward the apex of the left ventricle. The moment the blood stream reaches the aortic valve, it passively partially closes the anterior mitral leaflet. Subsequent atrial systole reopens the mitral orifice and increases the filling flow. Once the ventricle is filled, the mitral leaflets again close passively. Systole begins in the region of the papillary muscles, spreading rapidly to the wall of the entire ventricle. It is precisely this initial contraction of the papillary muscles that tensions the chordae tendineae, preventing eversion of the mitral leaflets in systole. Systole sharply increases the pressure in the left ventricle, tensioning the mitral leaflets, which causes the body of the leaflets to bulge toward the left atrium and produces perfect contact between both leaflets in the rough zone. A further functional feature of systole is a 40% reduction in the area of the mitral valve due to contraction of the annulus and the adjacent musculature. The shape of the mitral annulus also changes during systole, from circular to crescent-shaped, with the concavity at the site of the anterior leaflet.

1.4 Right Ventricle and Pulmonary Valve

Both ventricles of the heart, right and left, can be divided functionally, and partly also anatomically, into an inflow portion, an outflow portion, and an apical, trabecular portion. A strict anatomical division into sinus and conus is of less clinical and surgical use. For right ventricle, the inflow portion represents the area around the tricuspid valve, including the chordae tendineae and the muscoli pectinati. For orientation among the leaflets of the tricuspid valve, there is direct continuity between the septal leaflet and the interventricular septum. The apical trabecular portion of the right ventricle points forward, toward the apex of the whole heart. It is precisely in this location that the ventricular wall is thin enough to represent the most likely site for perforation during interventional procedures, during pacemaker implantation, or with indwelling catheters.

The outflow portion of the right ventricle consists of the infundibulum and the surrounding musculature that provides mechanical support to the pulmonary valve. Given the semilunar leaflets of the pulmonary valve, similarly to the aortic valve, the pulmonary annulus is not a flat ring. The leaflets arise from the annulus in a crescent shape, which also corresponds to a musculoarterial junction of matching shape. Anatomically, and also on echocardiography, three dimensions of the pulmonary valve are recognized, corresponding to three circles at three different levels along the axis of the pulmonary trunk. The uppermost is called the sinotubular junction and represents the highest of the rings, at the level where the valve leaflets join and separate at their attachments, the commissures. This ring is at the greatest distance from the right ventricle itself. The second ring lies at the level of the ventriculoarterial junction; the third, most proximal ring is formed by an imaginary connection at the level of the most proximal part of the semilunar leaflets, in the region of the infundibular musculature. However, none of these rings corresponds exactly to the true position of the pulmonary annulus, which follows the crescent-shaped attachments of the leaflets. Thus, at each leaflet, the annulus runs from the sinotubular junction down to the most proximal ring and back.

One of the anatomical hallmarks of the right ventricle is the muscular ridge separating the tricuspid and pulmonary valves, which is, in a sense, a continuation of the septomarginal trabecula. This muscular structure is the posterior part of the subpulmonary infundibular musculature, which provides mechanical support to the leaflets of the pulmonary valve. Surgery in this region can damage the right coronary artery, which runs in intimate proximity. Although at first glance this structure appears to belong to the outflow portion of the interventricular septum, it can be surgically removed without disrupting the integrity of the left ventricle. This confirms the fact that the outflow portions of the right and left ventricles are formed by separate, mutually independent muscular components. Similarly, the leaflets of the two valves themselves, as well as their annuli, do not share fibrous continuity, as is the case between the aortic and atrioventricular valves. It is precisely this anatomical independence of the pulmonary valve and the outflow portion of the right ventricle that allows the valve to be explanted for use in the Ross procedure. The Ross procedure is an autotransplantation of the pulmonary valve conduit into the aortic position for diseases of the aortic valve, with the pulmonary valve being replaced by an allograft, or other types of valve bioprosthesis in modified Ross procedure. After removal of the right ventricular infundibulum, the transition of the crista supraventricularis into the septomarginal trabecula becomes visible. The septomarginal trabecula divides, in its cranial course, into an anterior and a posterior component; the anterior component represents precisely the crista supraventricularis, while the posterior component continues into the inflow portion of the right ventricle. Caudally, the septomarginal trabecula continues into the apical portion of the right ventricle, where it differentiates into several smaller branches and disappears into the trabecular structures of the ventricular apex. One of these trabeculae ultimately emerges as the anterior papillary muscle of the right ventricle.

1.5 Left Ventricle and Aortic Valve

Like the right ventricle, the left ventricle can also be functionally divided into three components. The inflow portion is defined by the mitral valve and contains the chordae tendineae and muscoli pectinati. The chordae insert into two dominant muscles, anterolateral and posteromedial, with chordae from each leaflet

inserting into the heads of both papillary muscles. The mitral valve is not directly anatomically connected to the interventricular septum. The trabecular component fills mainly the apical portion of the ventricle, where, due to trabeculation, the resulting wall is locally thinned. As with the atria, trabeculation in the ventricles is also significantly less pronounced on the left side compared with the right.

The outflow portion of the left ventricle passes through and provides support to the aortic valve, and is composed of both a muscular and a fibrous part. The posterior part of the outflow tract consists of the fibrous component, which forms part of the cardiac skeleton at the site of the aortomitral continuity. The lateral side of the outflow tract is again formed by musculature, which externally corresponds to the location of the transverse sinus. Dorsal to the membranous septum, the left branch of the cardiac conduction system enters the musculature of the left ventricle; at the site of transition between the non-coronary and left coronary cusps of the aortic valve, it descends caudally into the ventricular musculature, immediately dividing into anterior, septal, and posterior divisions.

The aortic valve is likewise a semilunar valve, very similar in anatomical structure to the pulmonary valve. Similarly, it does not have a clearly defined planar annulus. Given its central position within the cardiac skeleton, the aortic valve is in contact with every chamber of the heart, and understanding these relationships is essential for understanding, and especially for the surgical correction of, almost all cardiac malformations (Figure 4).

The aortic valve consists of three semilunar cusps, which are attached to the ventriculoarterial junction in three half-arcs (scallops). Each cusp thus communicates distally with the aorta and proximally with the left ventricle. Above each cusp, on the aortic side, a sinus of Valsalva is formed. In diastole, the cusps meet centrally along the coaptation line. At the point where all three cusps meet, along the axis of the aortic valve, each cusp has a more or less prominent nodule, the *nodulus valvulae semilunaris* of Arantius. Moving laterally from the nodule toward the commissures, the coaptation line of each cusp physiologically narrows, and minimal perforations in the cusps at this lateral location are already considered physiological. During systole, the cusps are pushed by the blood stream slightly distally, but more laterally away from the axis of the aortic valve toward the wall of the aortic root, while in diastole they are passively pressed back together at the coaptation zone. Under physiological conditions, therefore, during diastole all three noduli of Arantius come into contact with each other, and the apposition of the coaptation zones of the adjacent portions of neighboring cusps creates a tight seal. Two of the three sinuses of Valsalva contain the origins of the coronary arteries, which is also reflected in their names and those of the corresponding cusps. We thus speak of the right coronary cusp (*valvula semilunaris dextra*), the left coronary cusp (*valvula semilunaris sinistra*), and the non-coronary cusp (*valvula semilunaris posterior*).

Understanding the mutual relationships between the aortic valve itself and the surrounding structures depends on understanding the shape of the attachment of each of its cusps. The commissure between the non-coronary and left coronary cusps corresponds to the site of the aortomitral continuity, with the fibrous connection itself lying beneath this commissure. The non-coronary cusp is adjacent to the posterior projection of the left ventricular outflow tract; here the aortic valve is also closest to the right atrium. At the site where the attachment of the non-coronary cusp extends into the commissure between the non-coronary and right coronary cusps lies the interatrial septum and, in particular, the atrioventricular node. The commissure between the non-coronary and right coronary cusps then lies directly above the point where the bundle of His crosses the interventricular septum. The attachment of the right cusp continues proximally over the central fibrous body of the cardiac skeleton. The commissure between the two coronary cusps, on the outer side of the aorta, corresponds to the space between the aorta and the pulmonary trunk. Both cusps adjacent to this commissure are anchored directly, and only, into the muscular outflow tract of the left ventricle. Given the independence of the outflow tracts of both ventricles, only a minimal part of the adjacent musculature forms the true interventricular septum. This independence of the outflow tract also corresponds to the fact that the attachment of the left coronary cusp is not adjacent to any other chamber of the heart.

Thorough mastery of the anatomy of the aortic valve and the topography of the surrounding structures is a prerequisite for mastering surgical procedures in this region, particularly for invasive infective endocarditis

and whenever surgical enlargement of the diameter of this valve's annulus is required. Opening and enlarging the aortic annulus at the anterior part of the annulus is called the Konno-Rastan procedure. In this case, the aortotomy is carried out longitudinally along the axis of the aorta, and the incision is directed toward the commissure between the anterior (right coronary) and non-coronary cusps. The incision then continues through the most medial part of the crista supraventricularis. After closing the right ventricle with a patch and implanting a further patch at the site of the incision in the aortic annulus, additional space is gained for implanting a larger aortic prosthesis, in order to avoid a higher postoperative transvalvular pressure gradient.

Another option for enlarging the valve annulus is to disrupt it at the site of the aortomitral continuity. In the Manouguian procedure, the aortotomy is extended through the commissure between the non-coronary and left coronary cusps as far as the anterior leaflet of the mitral valve. The Nicks variant instead directs the incision through the middle of the non-coronary cusp into the fibrous tissue beneath the aortic valve, alternatively also as far as the anterior mitral leaflet. Both techniques can breach the integrity of the left atrial wall, which must then be repaired. The Bo-Yang modification of aortic annular enlargement is performed through the commissure between the left coronary cusp and the non-coronary cusp. Just below the cusps, the incision is divided in a Y-shaped fashion, thereby avoiding direct involvement of the mitral valve apparatus. However, studies evaluating subsequent treatment options in patients after aortic annular enlargement have highlighted substantial alterations in the geometry of the left ventricular outflow tract, the aortic annulus, and the aortic root, which may considerably limit the feasibility of future transcatheter valve-in-valve procedures. In specific situations, the Ross procedure, mentioned in the description of the pulmonary valve, may also require enlargement of the aortic annulus, most commonly using the Konno–Rastan technique.

1.6 Aorta

The aorta, especially its ascending portion and the aortic arch, forms an anatomically inseparable part of cardiac surgical procedures. First, the ascending aorta and the branches of the aortic arch are the site for implanting the arterial cannula for cardiopulmonary bypass. A further part of the field consists of procedures performed at this site because of diseases of the aorta itself, in particular aneurysm, dissection, and coarctation of the aorta.

The ascending aorta begins at the aortic valve, at the level of the lower border of the third rib, and runs upward and slightly to the right, retrosternally, to the level of the upper border of the second rib. At its start, its otherwise circular shape is interrupted by the presence of three sinuses, corresponding to the three cusps of the aortic valve. This part of the ascending aorta, up to the level of the sinotubular junction, is called the aortic root and is the site from which the right and left coronary arteries arise. Most commonly, these are the only two branches of this part of the aorta. Externally, the initial segment of the aorta is covered by pericardium together with the pulmonary trunk. Between the aorta and the pulmonary trunk lie the aortopulmonary paraganglia (glomera), which serve as chemoreceptors and baroreceptors.

In the vicinity of the ascending aorta, we find anteriorly the pulmonary trunk and the right atrial appendage. Higher up, it is separated from the sternum by the pericardium, the right pleura, the anterior border of the right lung, and the thymus. Dorsal to the aorta lie the left atrium, the right pulmonary artery, and the main bronchus. To the right of the aorta lie the superior vena cava and the right atrium. To the left lie the left atrium and the pulmonary trunk.

The aortic arch is the continuation of the ascending aorta. It begins slightly to the right of the median plane, behind the second sternocostal joint. The arch first runs to the left, diagonally backward, toward the left edge of the anterior wall of the trachea. It then crosses the trachea from the left and continues caudally to the body of the fourth thoracic vertebra, where it transitions into the descending thoracic aorta. At the junction of the aortic arch and its descending portion, just past the origin of the left subclavian artery, there is a slight narrowing, the aortic isthmus. This is located at the site of the prenatal ductus arteriosus. The entire aortic arch thus lies in the superior mediastinum, arches over the left bronchus, and reaches the level of the middle of the manubrium sterni, roughly 2.5 cm below the jugular notch. The shadow of the aortic arch is also visible on a posteroanterior chest X-ray. At its start it measures 28 mm across, narrowing to 20 mm at

its end. To the left of the aortic arch lies the left pleural cavity. Along its left border, the aortic arch is crossed by the phrenic nerve and the vagus nerve, which gives off the recurrent laryngeal nerve. This nerve loops around the ligamentum arteriosum and ascends cranially along the right side of the aortic arch. To the right of, and behind, the aortic arch lie the trachea, the deep cardiac plexus, the right recurrent laryngeal nerve, the esophagus, the thoracic duct, and the vertebral bodies.

From the arch arise the brachiocephalic trunk, the left common carotid artery, and the left subclavian artery, which are crossed anteriorly by the left brachiocephalic vein. The origin of the first of these branches varies from the beginning of the arch to its highest point. More rarely, the left-sided branches may arise together as a left-sided brachiocephalic trunk. Individual origins of the internal and external carotid arteries directly from the aortic arch can also be found. The right subclavian artery normally arises from the brachiocephalic trunk behind the upper border of the sternoclavicular joint. It initially runs slightly cranially behind the clavicle, then behind the anterior scalene muscle, over the first rib into the axilla, where it becomes the axillary artery. The right-sided (aberrant) subclavian artery, when it arises from the aortic arch, usually does so individually, and likewise then passes behind the anterior scalene muscle. From the perspective of cardiac surgery, the anatomy of the subclavian artery is important both as a possible site for the arterial cannula of cardiopulmonary bypass and because of its relationship to the carotid arteries for cerebral perfusion. Furthermore, the subclavian artery and its branches are important in myocardial revascularization. One of the first branches of the subclavian artery on both sides is the internal thoracic artery, which runs dorsal to the sternocostal joints, caudally, ending as the superior epigastric artery and the musculophrenic artery. The internal thoracic artery is the most commonly used arterial graft for revascularizing the coronary arteries. Given that its proximal origin is usually left intact, successful revascularization requires adequate flow both within the internal thoracic artery itself, which very rarely undergoes atherosclerosis, and within the proximal subclavian artery. In the case of subclavian artery stenosis between its origin from the aortic arch and the origin of the internal thoracic artery, when this artery is used for myocardial revascularization, there is a postoperative risk of a "steal" phenomenon and the development of myocardial infarction with all its consequences.

The descending aorta lies in the posterior mediastinum and is a direct continuation of the aortic arch at the level of the lower border of the fourth thoracic vertebra. The end of the descending thoracic aorta is located at the level of the lower border of the twelfth thoracic vertebra, the point where the aorta passes through the diaphragm and continues as the abdominal aorta. Anterior to the descending aorta lie the left pulmonary hilum, the pericardium in direct contact with the left atrium, the esophagus, and the diaphragm. Posterior to the descending aorta lie the vertebral bodies and the hemiazygos vein. To the right of the aorta lie the azygos vein and lymphatic vessels; to the left lie the pleura and the lung. The esophagus crosses from the right side in its cranial course to lie anterior to the aorta at the midpoint of its course, and at the level where it passes through the diaphragm it is located to the left of the descending aorta. The physiological diameter of the descending aorta is 2.5 cm in its cranial portion and 2 cm in its caudal portion.

The descending thoracic aorta gives off visceral branches to the pericardium, bronchi, esophagus, and mediastinal structures, as well as parietal branches to the thoracic wall and spinal canal. The pericardial branches are small vessels supplying the posterior part of the parietal pericardium and are of limited clinical significance. The bronchial arteries usually arise at the level of the tracheal bifurcation and supply the bronchial tree and supporting pulmonary tissues. The esophageal branches supply the adjacent esophagus, while the mediastinal branches supply the surrounding connective tissue and lymph nodes. The posterior intercostal arteries comprise nine pairs, usually arising from the descending thoracic aorta and entering the third to eleventh intercostal spaces, where they anastomose anteriorly with branches of the internal thoracic and musculophrenic arteries. They also give off spinal branches that enter the vertebral canal and contribute to the segmental blood supply of the spinal cord. Of particular clinical importance is the great anterior radiculomedullary artery (artery of Adamkiewicz), which most commonly originates from a left-sided posterior intercostal or lumbar artery between T8 and L1 and provides a major contribution to the anterior spinal artery supplying the lower thoracic and lumbosacral spinal cord.

Development of the aorta begins in the third week of fetal development. It is a complex process that can give rise to a wide range of physiological variants as well as pathological malformations. The primitive aorta consists of two anterior and two posterior branches. The anterior branches subsequently form the aortic sac, and the posterior branches form the tissue of the descending aorta. Between the anterior and posterior aortic structures run six arches. The definitive anatomy of the aorta and its main branches arises through the regression of some parts of these primitive structures, while others form the definitive structures. In principle, the first arch forms the maxillary artery, and the second arch forms the stapedia and hyoid arteries, which anatomically and topographically no longer fall within the scope of this discussion. The third arches then form the common carotid arteries. The fourth arch on the left forms the definitive aortic arch, while the arch on the right forms the brachiocephalic trunk. The fifth arches regress quickly and are often not even present, and finally the sixth arches form the pulmonary arteries. On the dorsal aorta, the seventh intersegmental artery then begins, as the precursor of the subclavian artery on both sides.

The normal left-sided aortic arch arises through regression of the distal part of the right fourth primitive aortic arch, including the right-sided ductus arteriosus distal to the origin of the seventh intersegmental artery, together with the disappearance of the fifth arch on both sides. From the remnant of the right fourth arch as the brachiocephalic trunk arise the third left arch, forming the common carotid artery, and the seventh intersegmental artery, forming the subclavian artery. From the left-sided fourth arch, as the basis for the aortic arch, there remain separate origins for both the left common carotid artery and the left subclavian artery. This branching pattern is present in 70 to 80% of the healthy population. The most common variant is a common origin of the left common carotid artery together with the right common carotid artery. This type of arch is also found, for example, in cats. Further, a common origin of the left common carotid artery from the right brachiocephalic artery is present in 9 to 13% of the population. In 5 to 6% of patients we find a separate origin of the left vertebral artery from the aorta, proximal to the origin of the left subclavian artery.

There is also a broad spectrum of rarer congenital malformations which, depending on their significance, may be asymptomatic throughout the patient's life, may become symptomatic only in adulthood when a complication arises, or may be symptomatic already in early childhood, requiring surgical reconstruction. These include a left-sided aortic arch with an aberrant right subclavian artery, a left-sided aortic arch with a Kommerell diverticulum, a bilateral ligamentum arteriosum or bilateral persistence of the ductus arteriosus, and a left-sided aortic arch with a right-sided descending aorta and a persistent right-sided ductus arteriosus. A further group consists of a right-sided aortic arch, which may include an aberrant left subclavian artery arising from a retroesophageal diverticulum, a less common aberrant left subclavian artery arising from the descending aorta, an aberrant left-sided brachiocephalic trunk, or a right-sided aortic arch with a mirror-image branching pattern of all branches. Further anomalies, more or less symptomatic, include a right-sided aortic arch with an isolated left subclavian artery, a right-sided aortic arch with a left-sided descending aorta and a persistent left-sided ductus arteriosus, double aortic arch, cervical aortic arch, a persistent fifth aortic arch, and interrupted aortic arch with its three types. The last group of conditions includes hypoplasia of the aortic arch, coarctation, and pseudocoarctation.

1.7 Coronary Arteries

Both the left and right coronary arteries arise from the aortic root, just above the aortic valve, in the left and right sinuses of Valsalva. Most often, the origin is located in the upper third of the right and left aortic sinuses. Relative to the axis of the aortic root, the origin of the left coronary artery lies dorsal and slightly cranial to the origin of the right coronary artery. Clinically, we divide the coronary vascular territory into three segments, two of which fall under the left coronary artery. We thus speak of the clinically nearly equivalent right coronary artery, left anterior descending branch, and circumflex branch of the left coronary artery. This division is also the basis for the terminology of single-vessel, two-vessel, and three-vessel coronary disease. Left main coronary artery disease is classified separately. The distribution of blood supply to the heart muscle among the individual arteries and their branches is variable. Coronary dominance (right- or left-sided) depends on which vascular territory the posterior descending branch arises from, not on the

percentage of myocardium supplied. Right-sided dominance occurs in 85 to 90% of the population (Figure 5).

The left main coronary artery arises from the left sinus of Valsalva, above the left coronary cusp, and runs to the left and downward between the pulmonary trunk and the left atrial appendage. It most commonly reaches a length of 1 to 2 cm. Its length can, however, vary from a separate origin of the left anterior descending branch and the circumflex branch directly from the aorta, up to as long as 4 cm. The end of the left main coronary artery is considered to be the point where it divides into the nearly equal left anterior descending branch and circumflex branch.

From its origin at the bifurcation of the left coronary artery, the left anterior descending branch continues forward and downward in the anterior interventricular groove toward the apex of the heart. Its subepicardial course allows direct surgical revascularization. An intramuscular course is rarer and, when revascularization is needed, requires more demanding and riskier tissue dissection. Along its course it gives off diagonal branches (*rami diagonales*), branches passing into the interventricular septum, and weaker branches supplying the right ventricle. The diagonal branches supply the anterior wall of the left ventricle, and the caliber of the first or second of these may be large enough to allow surgical revascularization of that diagonal branch as well. The first diagonal branch may also arise directly at the bifurcation of the left coronary artery, in which case it is called the *ramus intermedius*. There are usually five septal branches, of which the first is the most important and usually arises from the left anterior descending branch just after the origin of the first diagonal branch. The septal branches supply the anterior two-thirds of the musculature of the interventricular septum. This right-angled origin is a distinguishing feature for identifying the left anterior descending branch on coronary angiography. Branches to the right ventricle may be absent. The left anterior descending branch itself usually terminates by dividing into two branches supplying the apex of the left ventricle.

The circumflex branch arises from the left coronary artery, usually forming a right angle with the left anterior descending coronary artery. The artery then runs along the boundary between the left atrium and ventricle and terminates at the obtuse margin. In about 10-15% of cases it ends as the posterior descending branch. The branches of the artery are called marginal branches (*rami marginales*) and, together with the posteromedial papillary muscle, supply the lateral wall of the left ventricle. Further branches supply the musculature of the left atrium and, in 40-50% of cases, also the sinoatrial node. When the posterior descending branch arises from the circumflex branch, supply to the sinoatrial node from this vessel is the rule.

The right coronary artery arises from the aorta and descends in the coronary (atrioventricular) groove. On reaching the acute margin, it bends backward. In most cases it then divides in a characteristic "U"-shaped manner into the posterior descending branch and the right posterolateral branch, which runs to the left. Branches to the atrioventricular node also arise at the site of this division. In half of cases, the sinoatrial nodal branch arises from the proximal part of the vessel. The posterior descending branch lies in the posterior interventricular groove and gives off branches into the interventricular septum. It is precisely these branches that most often supply one-third of the interventricular septum.

The posterior descending branch supplies the posterior wall of the left ventricle. The extent of the myocardium it supplies is, however, highly variable. This part of the heart may be supplied exclusively by the right coronary artery, exclusively by the circumflex branch of the left coronary artery, or by both arteries simultaneously. Before its division, the right coronary artery gives off the right marginal branch, supplying the free wall of the right ventricle. Septal branches of the right coronary artery can serve as a source of collateral circulation for the left anterior descending branch. Further, in its proximal course, the conus branch arises, which can likewise provide collateral circulation to the left anterior descending branch. The so-called Kugel's artery, another branch of the right coronary artery, provides collateral circulation to the circumflex branch of the left coronary artery.

Table 1 Relationship between individual coronary arteries, the anatomy of the corresponding left ventricular myocardium, and the electrocardiogram leads on which their occlusion is manifest.

Artery	Myocardium	ECG lead
<i>Right coronary artery</i>	inferior wall	II, III, aVF
<i>Circumflex branch</i>	lateral wall	I, aVL, V5, V6
<i>Left anterior descending branch - proximal</i>	interventricular septum	V1, V2
<i>Left anterior descending branch</i>	anterior wall	V2, V3, V4
<i>Right coronary artery</i>	right ventricle	V2R, V3R, V4R
<i>Posterior descending branch</i>	posterior wall	V7, V8, V9

Although there are substantial individual differences in coronary artery anatomy, in general it can be said that the left anterior descending branch supplies the anterior wall of the left ventricle, the anterior part of the interventricular septum, and part of the anterior wall of the right ventricle. The circumflex branch then supplies the lateral wall of the left ventricle and part of the interatrial septum, while the right coronary artery supplies most of the right ventricular myocardium and a small part of the interatrial septum. These proportions can vary individually. Given that the territory of the left anterior descending branch of the left coronary artery covers nearly half of the tissue of the left ventricle, this vessel is also called the "widowmaker." The clinical presentation of occlusion of the individual coronary arteries and their branches then varies according to the part of the myocardium affected. Partial occlusion of a coronary vessel generally leads to myocardial ischemia rather than complete infarction, manifesting clinically as angina pectoris or heart failure. To a certain degree, this state is fully reversible. Complete occlusion of a more important branch of the coronary vasculature leads to myocardial infarction in its territory, with the clinical presentation depending strictly on the anatomical structures affected and the extent of involvement of the right and left ventricular musculature (Table 1). Acute myocardial infarction may thus present as a fall in left ventricular ejection fraction when the infarct is small, as a cardiac rhythm disturbance when vessels supplying the conduction system are affected, as acute mitral regurgitation when vessels supplying a papillary muscle are affected, or as failure of the heart as a pump and death when more than 40% of the left ventricular myocardium is involved.

2 Cardiopulmonary Bypass and Mechanical Circulatory Support (Tomáš Toporcer)

2.1 Cardiopulmonary Bypass

Although the beginning of successful cardiac surgery dates back to 1896, when Ludwig Rehn successfully repaired a penetrating cardiac injury, for the following several decades only relatively simple procedures on the beating heart were possible. These included primarily closed mitral commissurotomy for rheumatic mitral stenosis, a procedure that has largely been replaced today by percutaneous balloon mitral valvuloplasty in appropriately selected patients. The major expansion of cardiac surgery required both the discovery and clinical introduction of heparin and the technical development of cardiopulmonary bypass. In 1953, John Gibbon performed the first successful open-heart operation using cardiopulmonary bypass, closing an atrial septal defect.

Cardiopulmonary bypass temporarily replaces the pumping function of the heart and the gas-exchange function of the lungs. This allows the heart to be arrested and mechanical ventilation to be discontinued during selected phases of the operation. Cardio pulmonary bypass (CPB) also permits controlled alteration of body temperature by cooling or rewarming the blood as it passes through the heat exchanger. Functionally, the CPB circuit consists of several essential components. These include the venous cannula or cannulae, for example during separate cannulation of the superior and inferior venae cavae; the venous line, which carries deoxygenated blood from the patient to the extracorporeal circuit; the venous reservoir, which receives venous blood either by gravity drainage or with the assistance of negative pressure; the main pump, usually a roller or centrifugal pump; the oxygenator, which oxygenates the blood and removes carbon dioxide; the heat exchanger, which regulates blood and body temperature; the arterial filter, which reduces the passage of gaseous and particulate embolic material; the arterial line, which carries oxygenated blood back to the patient; and the arterial cannula, which is inserted into the aorta or another large artery. Some centers may use reduced or modified extracorporeal circuits, including systems with a minimized reservoir, for selected procedures. A conventional CPB system also usually includes additional pumps for delivery of cardioplegia, suction of blood from the operative field, and ventricular venting to prevent cardiac distension, together with the corresponding cannulae and tubing. Modern CPB systems are equipped with a wide range of sensors and monitoring devices that continuously assess circuit performance as well as selected physical and biochemical parameters of the blood.

Venous blood usually enters the CPB circuit through the venous cannula by gravity drainage or with the assistance of active negative pressure and is collected in a venous reservoir positioned approximately 40–70 cm below the level of the heart. Venous cannulation may be central, with the cannulae placed in close proximity to the heart, or peripheral. Central venous cannulation can be performed in three basic configurations: bicaval cannulation, single-stage right atrial cannulation, or two-stage atriocaval cannulation. Bicaval cannulation, consisting of separate cannulation of the superior and inferior venae cavae with tourniquets or snares placed around both vessels, is required when the right-sided cardiac chambers are opened during cardiopulmonary bypass, as it prevents venous blood from entering the operative field and reduces the risk of air entering the extracorporeal circuit. This type of cannulation is commonly used for procedures involving the tricuspid valve, pulmonary valve, interatrial septum, and other intracardiac structures and may also be used during mitral valve surgery. Single-stage right atrial cannulation is generally sufficient for most aortic valve and coronary artery procedures. With two-stage atriocaval cannulation, the cannula is introduced through the right atrial appendage, with its distal portion positioned in the inferior vena

cava and its wider proximal portion, containing multiple side holes, remaining within the right atrium. Less commonly, venous cannulation may be performed peripherally, most often through the femoral or internal jugular vein, using either an open surgical or percutaneous Seldinger technique. Peripheral venous cannulation is frequently used in minimally invasive cardiac surgery, selected aortic procedures, emergency situations, and reoperations in which there is a risk of cardiac injury during repeat sternotomy or dissection of dense postoperative adhesions. Venous drainage is again achieved either by gravity or with the assistance of negative pressure, with blood flowing along the pressure gradient toward the extracorporeal circuit.

The arterial cannula, which returns oxygenated blood from the CPB circuit to the patient, may be inserted at several sites. The most commonly used sites include the ascending aorta, proximal aortic arch, brachiocephalic trunk, femoral artery, and axillary artery. The choice of cannulation site depends on the planned surgical procedure, the underlying aortic pathology, and the location and extent of atherosclerotic disease. Because of its accessibility and favorable flow characteristics, the distal ascending aorta is the most frequently used site for arterial cannulation. The cannula is typically inserted through a small arteriotomy within one or two concentric purse-string sutures. Potential complications include difficult cannula insertion, bleeding, injury or tearing of the aortic wall, atheroembolism, perforation of the posterior aortic wall, malposition resulting in inadequate or excessive cerebral perfusion, and iatrogenic aortic dissection. Femoral artery cannulation is frequently used in minimally invasive procedures, reoperations, and emergency situations, although it produces retrograde aortic perfusion. Axillary artery cannulation provides antegrade systemic perfusion and can also facilitate selective antegrade cerebral perfusion during aortic arch surgery.

The main pump maintains systemic blood flow during cardiopulmonary bypass and thereby replaces the pumping function of the heart. In most procedures, CPB is conducted using continuous, non-pulsatile flow. The two principal types of pumps are roller pumps and centrifugal pumps. A roller pump consists of flexible PVC or silicone tubing positioned against a curved raceway and intermittently compressed by two rollers mounted approximately 180° apart on a rotating head. Rotation of the rollers propels blood through the tubing at a flow rate determined primarily by the rotational speed and tubing dimensions. A centrifugal pump contains a rotating impeller or a series of smooth cones within a plastic housing. These components are magnetically coupled to an electric motor and, during rapid rotation, impart kinetic energy to the blood. This kinetic energy is converted into pressure and forward flow through the extracorporeal circuit. Unlike roller pumps, centrifugal pumps are afterload-dependent and generate flow according to both rotational speed and the resistance within the circuit.

Historically, oxygenators achieved gas exchange either by exposing a thin film of blood directly to an oxygen-rich atmosphere, as in disc oxygenators, or by bubbling oxygen directly through the blood, as in bubble oxygenators. Modern oxygenators use a membrane that separates the blood and gas phases. The same fundamental physical principles of convection and diffusion that govern gas exchange in the native lungs also apply to the artificial lung, that is, the oxygenator. Oxygen diffuses across the membrane into the blood and binds to deoxygenated hemoglobin, whereas carbon dioxide diffuses in the opposite direction from the blood into the gas phase.

The cardiopulmonary bypass circuit consists of numerous interconnected components and tubing that must be filled with fluid before CPB is connected to the cannulae already placed in the patient. This process is referred to as priming and is usually performed using a crystalloid solution supplemented with selected additives according to institutional practice. Most blood-contacting surfaces of modern extracorporeal circuits may be treated with biocompatible coatings, including heparin-based coatings, to reduce activation of coagulation and inflammation. Nevertheless, full systemic anticoagulation remains essential before cannulation and initiation of CPB. Unfractionated heparin is commonly administered at an initial dose of approximately 300 IU/kg, although institutional protocols vary. Provided that antithrombin activity is adequate, this dose generally achieves sufficient anticoagulation. The effect is monitored by measuring the activated clotting time (ACT), which is usually maintained above approximately 400–480 seconds throughout cardiopulmonary bypass, depending on the anticoagulation protocol used.

Once the extracorporeal circuit has been connected to the arterial and venous cannulae, it is essential to confirm that no air remains in any part of the arterial side of the circuit, as arterial air embolism may result in serious neurological or systemic complications. After the circuit has been checked, venous drainage is initiated and blood flows, either by gravity or assisted drainage, from the right atrium or another venous cannulation site into the venous reservoir. Once adequate venous return has been established, the main pump and oxygenator are brought into operation and oxygenated blood is returned from the extracorporeal circuit to the arterial system. As venous return to the heart decreases and blood is diverted into the CPB circuit, cardiac preload and ventricular work are markedly reduced, resulting in a substantial decrease in myocardial oxygen consumption. Under normothermic conditions, pump flow is generally maintained at a level sufficient to approximate physiological systemic perfusion requirements, commonly indexed to body surface area. The heat exchanger incorporated into the CPB circuit allows controlled systemic cooling to be induced; by reducing tissue metabolism and oxygen consumption, hypothermia permits a corresponding reduction in required pump flow while maintaining adequate organ perfusion.

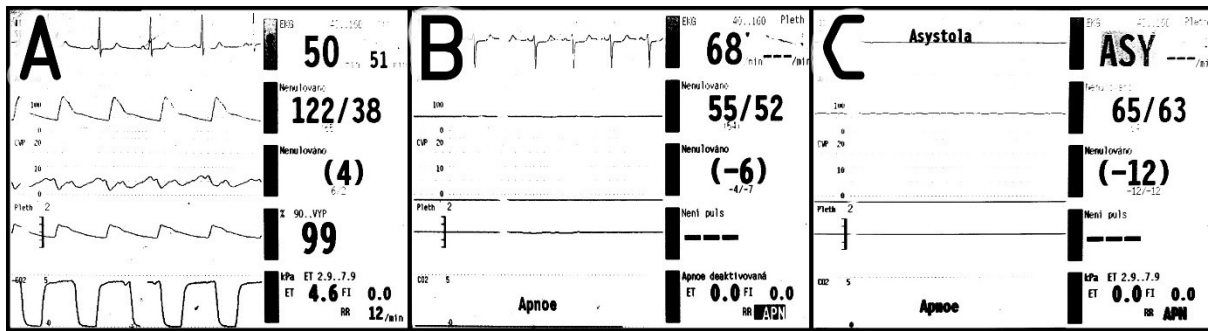


Figure 6 Vital-signs monitor during surgery using cardiopulmonary bypass (A - beating heart, B - full cardiopulmonary bypass flow before administration of cardioplegic solution - an ECG trace is visible (electrophysiological cardiac activity), but arterial blood flow is continuous at a pressure of 52-55 Torr, C - patient on cardiopulmonary bypass after administration of cardioplegic solution).

To provide a bloodless and motionless operative field, an aortic cross-clamp is applied to the ascending aorta, distal to the coronary ostia and proximal to the arterial cannula. This maneuver interrupts coronary perfusion and results in immediate myocardial ischemia. Myocardial protection is achieved by administration of a cardioplegic solution, most commonly into the aortic root proximal to the cross-clamp. When the aortic valve is competent, the cardioplegic solution is directed into the coronary arteries. Most cardioplegic solutions contain an elevated potassium concentration and are administered at a reduced temperature. Potassium is the principal agent used to induce cardiac arrest; at concentrations of approximately 16–20 mmol/L in the final crystalloid or blood solution delivered to the coronary circulation, it depolarizes the myocardial cell membrane and produces diastolic arrest. Once electrical and mechanical activity ceases, myocardial energy consumption decreases markedly. Additional cooling with hypothermic cardioplegia further reduces cellular metabolism and oxygen demand, thereby increasing myocardial tolerance to ischemia. Cardioplegia may be administered as cold crystalloid cardioplegia, cold blood cardioplegia, or warm blood cardioplegia. Blood cardioplegia incorporates the patient's own blood and provides additional oxygen-carrying and buffering capacity. Numerous cardioplegic formulations are currently available and may be broadly classified as extracellular or intracellular according to their electrolyte composition and the physiological environment they are intended to reproduce. Cardioplegia is most commonly delivered antegradely into the aortic root between the aortic valve and the applied cross-clamp. It may also be administered selectively into the coronary ostia or through venous bypass grafts after completion of the distal coronary anastomoses, allowing delivery beyond a proximal coronary stenosis. When antegrade delivery is inadequate or unsuitable, cardioplegia can be administered retrogradely through the coronary sinus, with the solution flowing from the coronary venous system through the myocardial microcirculation. A single dose of conventional St. Thomas-type cardioplegia generally provides myocardial protection for

approximately 20–30 minutes; if the period of cardiac arrest is prolonged, repeated doses are required (Figure 6).

After completion of the surgical procedure, gradual weaning from cardiopulmonary bypass begins. This process usually starts with removal of the aortic cross-clamp, after which warm, oxygenated blood with physiological electrolyte concentrations again enters the coronary circulation. Myocardial reperfusion then takes place, allowing restoration of the intracellular environment and recovery of myocardial metabolism. During the early reperfusion phase, the heart remains partially unloaded by cardiopulmonary bypass, which limits myocardial work and oxygen consumption. Adequate time should be allowed for myocardial recovery before separation from CPB. During this interval, additional surgical steps may be completed, such as construction of proximal venous graft anastomoses during coronary artery bypass surgery, thereby avoiding unnecessary prolongation of the total duration of cardiopulmonary bypass. Before weaning from CPB, the patient's acid-base status, electrolyte concentrations, hematocrit, and other relevant physiological parameters should be assessed and corrected as required. Adequate rewarming must also be achieved. Rewarming should be gradual, particularly after deeper hypothermia, in order to minimize excessive temperature gradients and the risk of cerebral hyperthermia. Once these conditions are fulfilled, mechanical ventilation is resumed and venous return to the extracorporeal circuit is progressively reduced while pump flow is decreased. The heart gradually reassumes its pumping function, with preload restored in a controlled manner. When satisfactory cardiac function and systemic hemodynamics have been achieved, cardiopulmonary bypass can be discontinued. Protamine sulfate is then administered to neutralize the anticoagulant effect of heparin and restore coagulation toward baseline. Finally, the venous and arterial cannulae are removed and the cannulation sites are secured.

2.2 Specifics of CPB in Aortic Arch Surgery - Brain Protection

One of the most important aspects of contemporary aortic arch surgery is the preservation of cerebral perfusion through the supra-aortic vessels to prevent intraoperative cerebral hypoperfusion. Patients undergoing these procedures are at considerable risk of neurological complications; therefore, the entire operative strategy must be tailored to minimize this risk. No single universally optimal approach exists, and various modifications of individual operative steps have been developed to enhance cerebral and systemic protection. Early attempts at surgery on the aortic arch and descending aorta were frequently associated with hypoxic injury to peripheral organs and neurological deficits. Systemic hypothermia was among the first methods introduced for cerebral protection during circulatory arrest. The first reported aortic arch reconstruction using deep hypothermic circulatory arrest dates back to 1975. Subsequently, selective antegrade and retrograde

cerebral perfusion techniques were introduced, allowing aortic arch surgery to be performed under less profound levels of hypothermia.

Historically, interruption of blood flow to the supra-aortic vessels and the brain was an integral part of aortic arch replacement. This period of complete interruption of systemic blood flow is referred to as circulatory arrest and provides a bloodless operative field for manipulation of the aorta and construction of the vascular anastomoses. Neurological complications associated with aortic arch surgery have traditionally been classified as permanent neurological injury and transient neurological dysfunction. Permanent neurological injury, most commonly presenting as perioperative stroke, is frequently related to cerebral embolization of particulate material, such as atherosclerotic debris, or air, resulting in focal cerebral ischemia. In contrast, transient neurological dysfunction has historically been attributed primarily to inadequate cerebral perfusion and global cerebral ischemia during the procedure, usually without a corresponding structural lesion on computed tomography or magnetic resonance imaging. To protect the brain and other organs during periods of circulatory arrest, systemic hypothermia is used to reduce tissue metabolism and oxygen consumption. The brain has particularly high metabolic requirements and is therefore especially vulnerable to interruption of blood flow. At a body temperature of approximately 15 °C, cerebral metabolic activity is reduced to approximately 15–17% of its normothermic baseline value. Nevertheless, hypothermia alone provides only a limited period of cerebral protection during complete circulatory arrest. For this reason, contemporary aortic arch surgery commonly combines systemic hypothermia with selective cerebral perfusion, most frequently selective antegrade cerebral perfusion, to provide additional protection of the brain during reconstruction of the aortic arch.

Deep hypothermic circulatory arrest (DHCA), with or without adjunctive selective cerebral perfusion, represents one of the fundamental strategies for cerebral protection during aortic arch surgery. When used as the sole method of cerebral protection, it is technically straightforward and does not require the establishment of a separate cerebral perfusion circuit. Nevertheless, appropriate monitoring of cerebral and systemic cooling remains important. After cardiopulmonary bypass is initiated, systemic cooling is achieved by reducing the temperature of blood passing through the heat exchanger. Excessive temperature gradients between the arterial blood delivered from the cardiopulmonary bypass circuit and the patient's venous blood should be avoided during both cooling and rewarming. The duration of the cooling phase depends on several factors, including cardiopulmonary bypass flow, the applied temperature gradient, body size, and the distribution of blood flow among individual tissue compartments. Monitoring of temperature, venous oxygen saturation, cooling time, and, when available, electroencephalographic activity improves the

assessment of the adequacy of systemic and cerebral cooling. Temperature is typically monitored at multiple sites, including the arterial and venous lines of the cardiopulmonary bypass circuit and at core temperature sites such as the nasopharynx, esophagus, bladder, or rectum. Nasopharyngeal temperature is commonly used as an approximate indicator of cerebral temperature during cooling. When DHCA is used without adjunctive cerebral perfusion, circulation is generally arrested after deep systemic hypothermia has been achieved, typically at temperatures of approximately 14–20 °C. Cardiopulmonary bypass is then discontinued, providing a bloodless operative field for reconstruction of the aortic arch. After completion of the required distal reconstruction and careful de-airing of the vascular system, systemic perfusion is re-established and controlled rewarming is initiated. Historically, DHCA was used as the sole method of cerebral protection during aortic arch surgery. Its protective effect, however, is time-limited. Clinical studies have demonstrated an increasing risk of neurological injury with prolonged circulatory arrest, particularly when the duration exceeds approximately 40 minutes. In a series of 394 patients undergoing surgery with DHCA alone at approximately 19 °C, the mean circulatory arrest time was approximately 31 minutes; overall mortality was 6.3% and the stroke rate was 4.8%. Among patients with circulatory arrest exceeding 40 minutes, the stroke rate increased substantially. Consequently, complex aortic arch procedures expected to require prolonged circulatory arrest are generally performed using adjunctive cerebral perfusion, most commonly selective antegrade cerebral perfusion.

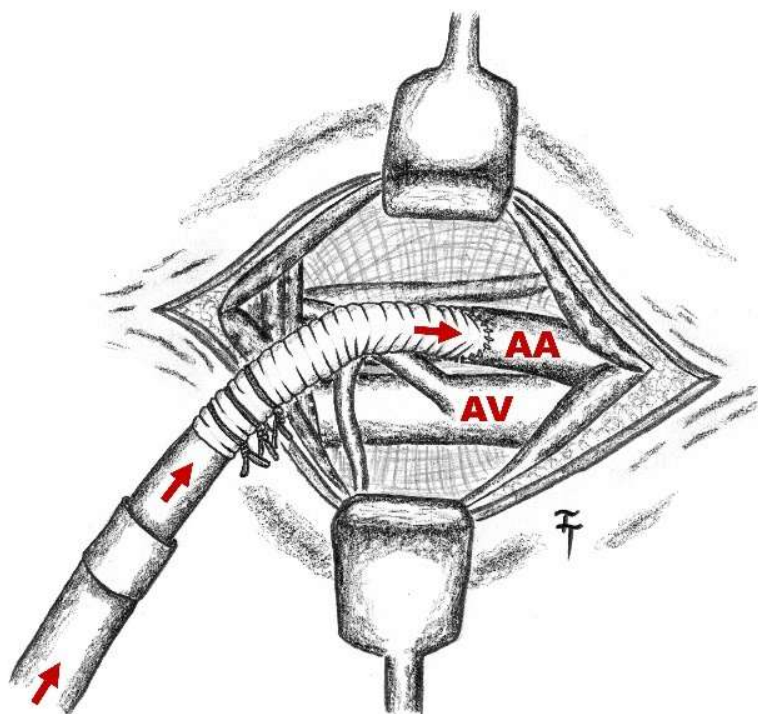


Figure 7 Cannulation of the axillary artery using an 8 mm vascular graft (AA - axillary artery, AV - axillary vein)

Retrograde cerebral perfusion was initially introduced into clinical practice for the treatment of massive iatrogenic air embolism and was subsequently adopted as part of a combined strategy for cerebral protection during aortic surgery. Retrograde cerebral perfusion is performed by delivering cold, oxygenated blood into the superior vena cava, thereby establishing retrograde flow through the cerebral venous system and the upper half of the body. It is generally administered at a flow rate of approximately 200–400 mL/min while maintaining superior vena caval pressure at 15–25 mmHg. Adequate perfusion can be confirmed by the return of dark, deoxygenated blood from the arterial vessels of the upper body. This technique offers several potential advantages. Most importantly, it may facilitate the clearance of air and particulate embolic material from the cerebral arterial circulation; additional benefits include maintenance of cerebral hypothermia and, to a more limited extent, delivery of oxygenated blood to cerebral tissue. The combined use of deep hypothermia and retrograde cerebral perfusion has therefore been associated with improved cerebral protection during aortic arch surgery compared with deep hypothermic circulatory arrest alone. Nevertheless, the technique does not completely eliminate the risk of neurological complications or mortality, and reported operative mortality with this combined approach remains approximately 3.4%.

Another strategy used to reduce the risk of neurological complications during aortic arch surgery is selective antegrade cerebral perfusion, which is currently employed in most cardiac surgical centers. Antegrade cerebral blood flow provides more physiological and effective oxygen delivery to brain tissue than retrograde perfusion through the superior vena cava. Technically, selective antegrade cerebral perfusion can be performed using several modifications. Bilateral perfusion is commonly achieved by delivering oxygenated blood directly into the left common carotid artery and the brachiocephalic trunk after opening the aortic arch. Potential disadvantages include the risk of air embolism and embolization of particulate material released from atherosclerotic plaques during cannula insertion, while the cannulas themselves may also interfere with visualization of the operative field. For these reasons, many centers prefer antegrade cerebral perfusion through the right axillary (continuation of the subclavian artery) artery or the brachiocephalic trunk. When the right axillary artery is used, the procedure typically begins with end-to-side anastomosis of an approximately 8-mm vascular graft to the artery, followed by insertion of the arterial cannula of the cardiopulmonary bypass circuit into the graft (Figure 7). Venous drainage is established either through the femoral vein or, more commonly, by central cannulation. During circulatory arrest, the brachiocephalic trunk, left common carotid artery, and left subclavian artery are occluded, and cardiopulmonary bypass flow is reduced to approximately 10 mL/kg/min, with further adjustment according to the degree of hypothermia and cerebral perfusion requirements. Perfusion pressure can be monitored through a catheter placed in the right radial artery. With unilateral antegrade cerebral

perfusion, the primary flow is directed to the right cerebral hemisphere, while perfusion of the left hemisphere depends on collateral circulation through the circle of Willis (Figure 8). If collateral flow is inadequate, the left hemisphere may be at risk of hypoperfusion, and cerebral perfusion should be supplemented by direct cannulation of the left common carotid artery. In patients with an intact circle of Willis and adequate collateral circulation, additional left carotid cannulation is usually unnecessary. Moreover, unnecessary manipulation of the arch vessels may itself increase the risk of cerebral embolization and perioperative stroke.

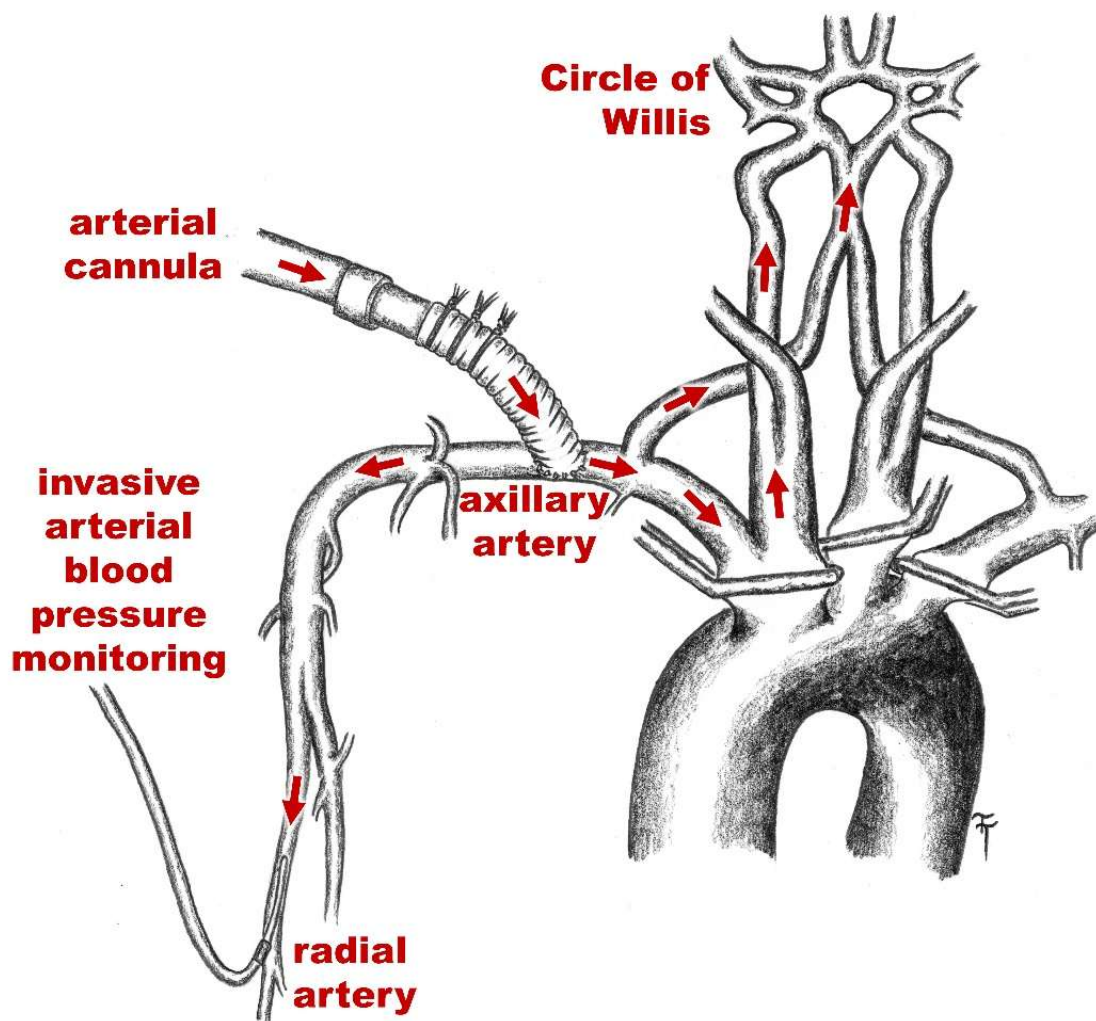


Figure 8 Blood flow during selective antegrade cerebral perfusion during surgery in the region of the aortic arch.

The use of selective cerebral perfusion allows deep hypothermia to be replaced by a more moderate degree of systemic cooling. With selective cerebral perfusion, circulatory arrest is effectively limited to the lower body, while cerebral blood flow is maintained. Systemic hypothermia is therefore primarily required to protect the remaining organs, peripheral tissues, and the spinal cord during the period of distal circulatory arrest. Because these tissues generally have

lower metabolic demands than the brain, less profound hypothermia is usually sufficient to reduce their oxygen requirements to a tolerable level. The combination of moderate hypothermia and selective antegrade cerebral perfusion is currently one of the most widely used strategies for cerebral and systemic protection during aortic arch surgery. For hemiarch replacement, target core temperatures of approximately 28–29 °C are commonly used, whereas total arch replacement is often performed at somewhat lower temperatures, approximately 25–26 °C, depending on the expected duration of lower-body circulatory arrest and institutional practice. Contemporary practice therefore most commonly involves either deep hypothermia combined with retrograde cerebral perfusion or moderate hypothermia combined with selective antegrade cerebral perfusion, most frequently using a unilateral approach.

Based on the available evidence, the combination of systemic hypothermia with selective cerebral perfusion provides more effective cerebral protection than hypothermia alone. Comparative studies have not consistently demonstrated a significant difference in the incidence of permanent stroke between antegrade and retrograde cerebral perfusion. However, antegrade cerebral perfusion has been associated with a lower incidence of transient postoperative neurological dysfunction in some studies. Cannulation of the right axillary artery combined with continuous monitoring of cerebral oxygen saturation is currently a widely used strategy. If cerebral oxygenation is inadequate during unilateral perfusion, bilateral selective antegrade cerebral perfusion can be established by adding direct perfusion of the left common carotid artery. When right axillary artery cannulation is not feasible, for example because of an aberrant right subclavian artery, proximal extension of an aortic dissection, significant stenosis, or other anatomical limitations, an alternative arterial inflow site and cerebral perfusion strategy must be selected.

2.3 Specifics of CPB in Descending Aorta Surgery - Spinal Cord Protection

Hypothermia also remains an important adjunct for protection of peripheral tissues during surgical procedures on the descending thoracic aorta. The patient is placed on cardiopulmonary bypass, most commonly using peripheral cannulation of the femoral artery and vein, and systemic cooling is initiated after establishment of extracorporeal circulation. A left thoracotomy is typically used for surgical access. Once the target temperature has been reached, selected according to the anticipated duration of circulatory interruption and the planned operative strategy, pump flow may be reduced and vascular clamps applied proximally and distally to the segment of aorta undergoing reconstruction. Because interruption of proximal aortic flow also compromises perfusion of the upper body, the duration of this phase is limited unless an additional arterial inflow is established to maintain cerebral and upper-body perfusion. Pump flow must then be adjusted according to the requirements of the different perfusion territories. After completion of the aortic reconstruction, the

clamps are removed and full systemic perfusion is restored. Rewarming is then performed in a controlled manner, and separation from cardiopulmonary bypass should be undertaken only after adequate restoration of body temperature and satisfactory systemic hemodynamics.

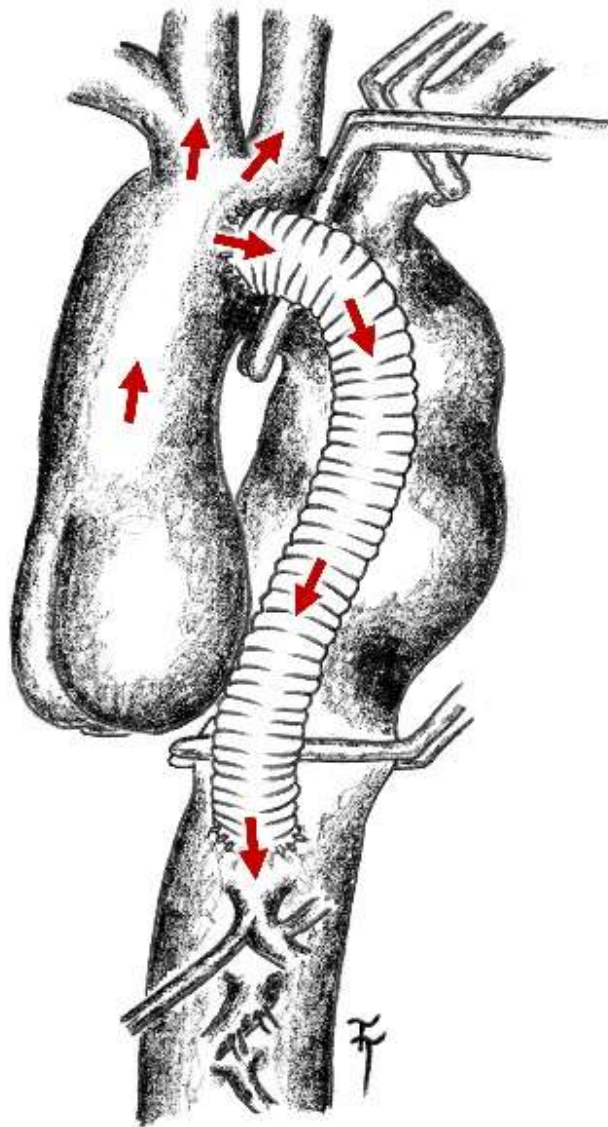


Figure 9 Gott shunt.

Surgery on the descending aorta can also be performed using a temporary extra-anatomic bypass, which diverts blood around the segment of the aorta undergoing reconstruction while maintaining distal perfusion as effectively as possible. Historically, the Gott shunt was used for this purpose, connecting the proximal aorta, usually at the level of the aortic arch or proximal descending aorta, to the descending aorta distal to the operative segment. Several technical modifications of this shunt were described (Figure 9). By maintaining blood flow around the clamped aortic segment, the shunt allowed reconstruction of the aorta between its proximal and distal connections.

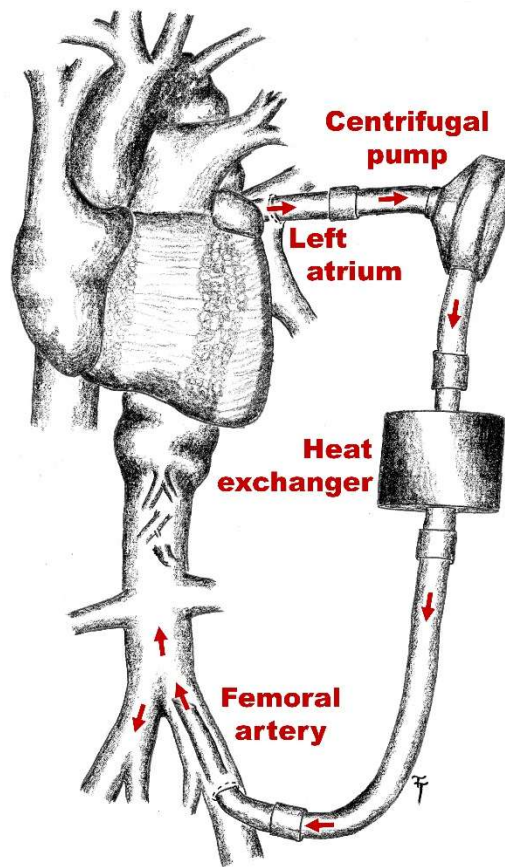


Figure 10 Left heart bypass.

Left heart bypass is a more contemporary technique used during surgery on the descending thoracic aorta. Blood is drained from the left atrium, usually through a pulmonary vein, and is returned by a centrifugal pump to the arterial circulation distal to the aortic cross-clamp, most commonly through the femoral artery (Figure 10). Because oxygenated blood is obtained directly from the left heart, a venous reservoir and oxygenator are generally not required. Systemic anticoagulation can therefore be reduced compared with conventional cardiopulmonary bypass. This is largely possible because the circuit does not contain an oxygenator, which represents one of the most thrombogenic components of a conventional CPB circuit because of its large artificial blood-contacting surface. Nevertheless, the required heparin dose and target activated clotting time depend on the specific circuit and institutional protocol. During left heart bypass, careful monitoring of proximal and distal arterial pressures, bypass flow, cardiac filling pressures, oxygenation, and overall hemodynamic status is essential. Left heart bypass provides controlled distal aortic perfusion and may reduce the risk of ischemic injury to the spinal cord and abdominal organs compared with simple aortic cross-clamping or passive shunting. Reported rates of spinal cord injury vary considerably according to the underlying aortic pathology, extent of reconstruction, and use of

adjunctive protective strategies, and should therefore not be attributed solely to the type of bypass technique used.

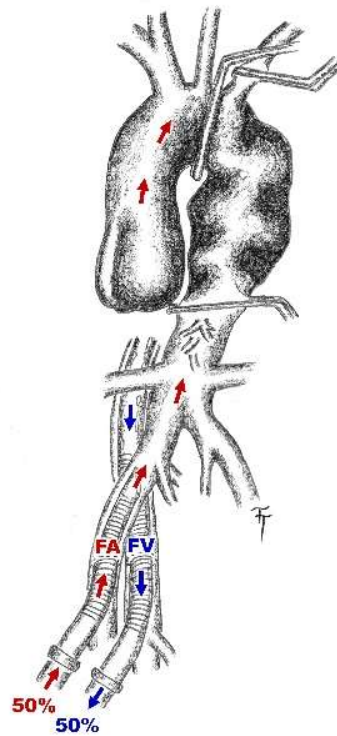


Figure 11 Partial cardiopulmonary bypass (FA - femoral artery; FV - femoral vein)

Partial cardiopulmonary bypass represents another controlled method of maintaining perfusion of the lower body during surgery on the descending thoracic aorta (Figure 11). Its use can reduce the risk of ischemic injury to distal organs, particularly the spinal cord. Venous drainage is most commonly established by peripheral cannulation of the femoral vein, with the cannula advanced into the right atrium. Blood is withdrawn into the extracorporeal circuit, oxygenated, and then returned to the arterial circulation of the lower body, usually through the femoral artery. This creates two parallel circulations: the native heart and lungs maintain perfusion and oxygenation of the upper body, while the extracorporeal circuit provides distal perfusion below the aortic cross-clamp. Ventilation of the lungs is maintained, often using one-lung ventilation to facilitate surgical exposure. By adjusting venous drainage and pump flow, the perfusionist can modulate cardiac preload and systemic hemodynamics under continuous invasive arterial pressure monitoring, typically from an upper-extremity artery. Flow through the arterial cannula of the bypass circuit determines perfusion of the lower body. This technique may also be useful in patients with variant arch anatomy, including an aberrant subclavian artery, and represents a practical option in centers experienced with peripheral cardiopulmonary bypass. Its principal disadvantage is the need for full systemic anticoagulation because the extracorporeal circuit includes an oxygenator.

3 Heart transplantation (Tomáš Toporcer)

The concept of replacing a failing heart with a healthy donor organ was explored experimentally long before clinical transplantation became feasible. The first experimental cardiac transplantation is generally attributed to Alexis Carrel and Charles Guthrie, who in 1905 at the University of Chicago performed heterotopic heart transplantation in dogs. From the early 1950s, Vladimir Demikhov further developed experimental heterotopic heart transplantation in dogs and contributed substantially to the technical foundations of the field. During the same period, several principles that remain fundamental to donor-organ preservation were introduced, including hypothermic protection of the donor and recipient and the use of potassium-containing solutions for myocardial preservation.

The surgical technique also evolved rapidly during this period. In 1959, Goldberg performed an experimental orthotopic heart transplantation in dogs at the University of Maryland, while Cass and Brock at Guy's Hospital described an early bicaval approach. At Stanford University, Norman Shumway and his colleagues developed a reproducible technique for orthotopic heart transplantation using cardiopulmonary bypass. In 1960, Lower and Shumway reported prolonged survival after orthotopic cardiac transplantation in dogs using immunosuppressive therapy with corticosteroids and azathioprine. The first cardiac xenotransplantation in a human was performed by James Hardy in 1964 using a chimpanzee heart; however, the graft was unable to provide sufficient circulatory support for the recipient.

The first successful heart transplantation between two humans was performed by Christiaan Barnard in 1967; the recipient survived for 18 days. Early outcomes were limited primarily by acute rejection and infectious complications. Subsequent advances in immunosuppressive therapy, initially based on corticosteroids and antiproliferative agents and later transformed by the introduction of calcineurin inhibitors such as cyclosporine and tacrolimus, together with improvements in donor selection, organ preservation, surgical technique, and postoperative care, established heart transplantation (HTx) as a standard treatment for selected patients with advanced heart failure. Today, HTx remains the definitive therapeutic option for eligible patients with end-stage heart disease when optimal medical, interventional, and surgical therapies are no longer sufficient.

Patients with advanced heart failure (AHF) have a poor prognosis despite optimal medical therapy. Major advances in surgical treatment have been achieved through the development of mechanical circulatory support (MCS) and heart transplantation. In carefully selected patients with AHF, heart transplantation (HTx) remains the gold standard of treatment, while left ventricular assist device (LVAD) therapy has become an important alternative. HTx offers the potential for

excellent functional recovery and freedom from device-related complications, but its use is fundamentally limited by the availability of suitable donor organs. As the global prevalence of heart failure continues to increase, the demand for transplantation remains substantially greater than the supply of donor hearts.

For this reason, the management of advanced heart failure is closely linked to mechanical circulatory support. Patients awaiting heart transplantation are frequently stabilized with temporary or durable MCS as a bridge to transplantation or bridge to decision, while durable support may serve as destination therapy in patients who are not candidates for transplantation. Because the availability of suitable donor hearts remains limited and waiting times may be prolonged, mechanical circulatory support is an integral component of the management of patients with advanced heart failure.

3.1 Extracorporeal Short-Term and Medium-Term Mechanical Support Systems

A wide range of short-, intermediate-, and long-term mechanical circulatory support devices is currently available to temporarily support or replace the function of a failing heart. Short-term mechanical circulatory support is primarily indicated in patients with severe or refractory cardiogenic shock, characterized by inadequate cardiac output and tissue hypoperfusion despite appropriate medical therapy. Typical hemodynamic findings include a cardiac index below approximately 2.2 L/min/m², systolic arterial pressure below 90 mmHg, elevated cardiac filling pressures, and the need for substantial inotropic or vasopressor support. Patients with critical cardiogenic shock and rapidly deteriorating hemodynamics correspond most closely to INTERMACS profile 1 (Interagency Registry for Mechanically Assisted Circulatory Support) (Table 3). The principal objective of mechanical circulatory support is to maintain adequate systemic perfusion and oxygen delivery while allowing myocardial recovery or providing a bridge to a definitive therapeutic strategy.

The development of mechanical circulatory support in humans dates back to the work of Kantrowitz, who introduced the concept of aortic counterpulsation and subsequently developed the intra-aortic balloon pump (IABP). The IABP later became the most widely used temporary mechanical circulatory support device for several decades. It consists of a balloon positioned in the descending thoracic aorta, with its tip usually located just distal to the origin of the left subclavian artery. The balloon inflates during diastole and deflates immediately before systole. Diastolic inflation increases aortic diastolic pressure and may improve coronary perfusion, whereas rapid deflation before systole reduces left ventricular afterload and myocardial oxygen demand. The hemodynamic support provided by IABP is relatively modest, and its routine use in cardiogenic shock, particularly after acute myocardial infarction, has markedly declined because randomized

studies failed to demonstrate a survival benefit. Nevertheless, IABP remains useful in selected clinical situations, including temporary stabilization of some mechanical complications of acute myocardial infarction and selected cases requiring short-term ventricular unloading or perioperative circulatory support.

Table 2 INTERMACS classification (INTERMACS - Interagency Registry for Mechanically Assisted Circulatory Support, LVAD - left ventricular assist device, NYHA - New York Heart Association).

	Simplified description	Indication for mechanical cardiac support
I	Patient in cardiogenic shock	Immediate extracorporeal support
II	Patient hemodynamically unstable despite inotropic therapy	LVAD implantation within days
III	Patient unable to be weaned from inotropes	LVAD implantation indicated
IV	Patient with inadequate response to maximized diuretic therapy	LVAD implantation indicated
V	Patient unable to leave the house due to risk of decompensation	Patient can tolerate deferral and reassessment
VI	Patient at risk of decompensation with even minimal exertion	Patient can tolerate deferral and reassessment
VII	Patient with NYHA IV	LVAD not indicated

Extracorporeal membrane oxygenation (ECMO) is one of the most widely used forms of temporary extracorporeal cardiopulmonary support. It can be established in either a veno-venous (VV-ECMO) or veno-arterial (VA-ECMO) configuration. In both configurations, blood is drained from the venous system through a centrally or peripherally placed cannula, passes through an extracorporeal circuit containing a pump, membrane oxygenator, and heat exchanger, and is then returned to the patient through a second cannula. In VV-ECMO, oxygenated blood is returned to the venous circulation and subsequently passes through the right heart and pulmonary circulation. This configuration provides respiratory support and is therefore used primarily in patients with severe respiratory failure and preserved or adequate cardiac function. In VA-ECMO, oxygenated blood is returned directly to the arterial circulation, thereby providing both respiratory and circulatory support and making this configuration suitable for patients with severe cardiac failure or cardiogenic shock. Anticoagulation during ECMO is generally less intense than during conventional cardiopulmonary bypass, but the required heparin dose and target activated partial thromboplastin time vary according to institutional protocols, bleeding risk, circuit characteristics, and the patient's clinical condition rather than being defined by a single universal target. Cannulation-related vascular complications are common, particularly limb ischemia during peripheral femoral VA-ECMO caused by obstruction of arterial flow by the return cannula. This

complication can be reduced by placement of a smaller distal perfusion cannula into the superficial femoral artery distal to the primary arterial cannulation site. Other important complications include thrombosis within the extracorporeal circuit, intracardiac thrombosis, bleeding, and differential hypoxemia, traditionally termed Harlequin or North-South syndrome. Differential hypoxemia occurs particularly during peripheral femoral VA-ECMO when recovering native left ventricular output ejects poorly oxygenated blood from severely impaired lungs into the proximal aorta, while well-oxygenated ECMO blood perfuses the distal aorta and lower body. As a result, the coronary and cerebral circulations may receive inadequately oxygenated blood. Management is directed primarily at improving pulmonary oxygenation and modifying the balance or configuration of native and extracorporeal blood flow; options include optimization of ventilation, adjustment of ECMO flow, repositioning or modification of cannulation, addition of a venous return limb to create a V-AV configuration, or conversion to axillary or central arterial return. Left ventricular venting may be required when VA-ECMO causes ventricular distension, pulmonary edema, or impaired ventricular unloading, and it can also reduce the risk of intracardiac stasis and thrombosis, but it is not the primary treatment for differential hypoxemia. Despite the substantially greater complexity of patient management compared with IABP, VA-ECMO has become an important and widely used form of short-term mechanical circulatory support in patients with severe or refractory cardiogenic shock. Its use should nevertheless be individualized, because routine application in all patients with cardiogenic shock has not been shown to provide a consistent mortality benefit.

Another widely used temporary mechanical circulatory support device is the Impella. The Impella is a catheter-mounted microaxial flow pump that actively draws blood from one chamber and expels it across the pump into the downstream circulation. Because it does not contain an oxygenator, the left-sided Impella provides circulatory support without directly supporting gas exchange. It is usually introduced through a peripheral artery and positioned across the aortic valve, with its inflow located in the left ventricle and its outflow in the ascending aorta. In this configuration, blood is actively pumped from the left ventricle into the aorta, thereby unloading the ventricle and increasing systemic blood flow. The Impella can also be used for left ventricular unloading during VA-ECMO as an alternative or adjunct to other unloading strategies. Because the device itself does not oxygenate blood, adequate pulmonary gas exchange or additional extracorporeal oxygenation is required when severe respiratory failure is present. A right-sided version, the Impella RP, has been developed for temporary right ventricular support. It is introduced through the venous system, with blood drained from the inferior vena cava or right atrium and delivered into the pulmonary artery across the pulmonary valve. Numerous other temporary and durable mechanical circulatory support devices are available worldwide, using continuous or

pulsatile flow and different pump technologies, with either peripheral or central cannulation. In patients with acute myocardial infarction complicated by cardiogenic shock, the use of intravascular microaxial flow pumps should be individualized according to hemodynamic status, shock severity, bleeding and vascular risk, and the anticipated therapeutic strategy rather than applied routinely. Observational studies have reported higher rates of bleeding and vascular complications with Impella compared with less invasive support strategies, while randomized evidence for a consistent mortality benefit remains limited.

At the time mechanical circulatory support is considered, the therapeutic objective of implantation and the anticipated “exit strategy” should also be defined, including whether meaningful myocardial recovery, transition to durable support, or transplantation is realistically achievable. Short-term mechanical support is frequently used as a bridge to recovery in potentially reversible conditions such as acute myocardial infarction with myocardial stunning or postcardiotomy cardiogenic shock, with the expectation that myocardial function may recover sufficiently to allow device explantation within days or weeks. Other strategies include bridge to transplantation, for which durable mechanical circulatory support is generally more appropriate when prolonged support is anticipated; bridge to destination therapy; bridge to bridge, in which temporary support is used until implantation of a durable device is possible; and bridge to decision, which provides time to assess neurological status, end-organ function, transplant eligibility, and overall prognosis before a definitive strategy is selected. The initial strategy may change during follow-up as the patient’s clinical condition and transplant candidacy evolve. In INTERMACS data, 43.5% of patients initially implanted as bridge to transplantation were no longer listed for transplantation at 2 years, illustrating that treatment intent is not necessarily fixed. Conversely, some patients initially considered unsuitable for transplantation may become eligible after clinical optimization, including improvement in pulmonary vascular resistance and end-organ function. Similarly, a bridge-to-recovery strategy may need to be converted to durable mechanical support if adequate myocardial recovery does not occur. Mechanical circulatory support should not be initiated when there is no realistic prospect of myocardial recovery, transplantation, durable support, or another meaningful definitive treatment strategy and when the expected burden of treatment clearly outweighs the possibility of achieving an acceptable clinical outcome.

3.2 Intracorporeal Long-Term Mechanical Support Systems

The indication for durable mechanical circulatory support is advanced heart failure that remains refractory to optimal medical and device therapy and is associated with severe functional limitation, recurrent decompensation, and evidence of impaired systemic perfusion. Typical features include a markedly reduced left ventricular ejection fraction, often below 25%, low cardiac output or cardiac

index, elevated filling pressures, low peak oxygen consumption, dependence on inotropic support, and recurrent hospitalizations for heart failure. Repeated hospitalization, for example three or more admissions for decompensated heart failure within one year, is an important marker of advanced disease but should not be regarded as an isolated indication for device implantation. Durable mechanical circulatory support is most commonly considered in patients corresponding to INTERMACS profiles 2–4, although the exact timing depends on clinical trajectory and end-organ function. It is usually implanted as a left ventricular assist device (LVAD), whereas isolated durable right ventricular support is uncommon; selected patients with severe biventricular failure may require biventricular support or a total artificial heart (TAH). Contemporary implantable LVAD systems consist of an intracorporeal pump connected to an external controller and power source through a percutaneous driveline. Fully implantable systems using transcutaneous energy transmission, which would eliminate the driveline and potentially reduce infection risk, remain investigational rather than established clinical standard. First-generation durable devices included pulsatile systems such as the HeartMate XVE. The second generation was represented by continuous-flow axial pumps such as the HeartMate II, in which blood is withdrawn from the left ventricle through an apical inflow cannula and returned to the ascending aorta through an outflow graft. Because of its size, the pump was commonly positioned in a preperitoneal or intraperitoneal pump pocket. Third-generation devices use centrifugal continuous-flow technology with magnetic levitation. The HeartMate 3 is the principal contemporary example: it is implanted within the pericardial space, draws blood from the left ventricular apex, and returns it to the ascending aorta. The HeartWare HVAD was another compact centrifugal-flow device of this generation, but its sale and distribution were discontinued in 2021 because of higher rates of neurological adverse events and mortality compared with other available LVADs, together with concerns regarding pump failure to restart. Adequate right ventricular function is a major determinant of outcome after LVAD implantation. LV unloading alters interventricular septal geometry and increases venous return to the right ventricle, which may precipitate or worsen right ventricular failure. Assessment and management of right ventricular function are therefore central to perioperative and long-term LVAD care. When medical treatment is insufficient, temporary right ventricular mechanical support may be required; in selected patients with irreversible biventricular failure, durable biventricular support or total artificial heart replacement may be considered.

The modern history of the total artificial heart (TAH) includes several important milestones. In 1969, Liotta and Cooley implanted a TAH as temporary support before transplantation, whereas in 1982 William DeVries implanted the Jarvik-7 in Barney Clark as the first permanent total artificial heart replacement. Implantation of a TAH requires excision of both native ventricles while

preserving portions of the atria, followed by connection of the device to the right and left atrioventricular valve annuli and to the pulmonary artery and ascending aorta. The device contains two separate artificial ventricles that replace the pumping function of the native right and left ventricles. TAH implantation is primarily considered in patients with severe irreversible biventricular failure in whom isolated ventricular assist-device support is unsuitable, and in selected situations in which extensive cardiac replacement is required. The SynCardia temporary Total Artificial Heart is a pneumatically driven system derived from the Jarvik-7 concept; its pumps are implanted within the thorax, while pneumatic drivelines connect them to an external driver. The AbioCor represented a different concept of a fully implantable TAH with transcutaneous energy transfer and is primarily of historical importance in the development of fully implantable artificial hearts.

Mechanical circulatory support represents an advanced therapeutic option for patients with severe or end-stage heart failure when optimal pharmacological, interventional, and conventional surgical treatment is insufficient. It should be considered within the broader spectrum of guideline-directed heart failure therapy, which may also include cardiac resynchronization therapy (CRT) in appropriately selected patients. CRT uses coordinated biventricular pacing to improve ventricular electrical and mechanical synchrony, particularly in patients with ventricular conduction delay, and may improve left ventricular function, symptoms, and clinical outcomes. Mechanical circulatory support is therefore not simply a final treatment after all other therapies have failed, but rather a component of an advanced heart failure strategy that may be used as a bridge to recovery, bridge to transplantation, bridge to decision, or destination therapy, depending on the patient's clinical condition and long-term therapeutic options.

3.3 Indications and Contraindications for Heart Transplantation

The principal indication for heart transplantation is advanced heart failure refractory to optimal medical, interventional, and device-based therapy. Other selected indications include recurrent life-threatening ventricular arrhythmias and severe refractory angina that is not amenable to further revascularization. Important contraindications or major limiting factors include active systemic infection, active or recently treated malignancy with an unacceptable risk of recurrence, severe peripheral vascular disease, irreversible dysfunction of other major organs, and severe obesity. Contemporary guidelines emphasize individualized assessment rather than the use of single variables in isolation. Several donor- and recipient-related factors influence post-transplant outcomes, including ABO compatibility, immunological compatibility and sensitization, donor-recipient size matching, ischemic time, donor and recipient age, infectious risk, and donor characteristics and cause of death.

3.4 Donor Selection, Allocation and Graft Preservation

Donor selection requires balancing graft quality against the persistent shortage of suitable organs. Increasing donor age is associated with a higher prevalence of coronary atherosclerosis, myocardial fibrosis, valvular disease, and greater susceptibility to ischemic injury, and is therefore associated with less favorable long-term outcomes. Nevertheless, carefully selected older donor hearts may be appropriate, particularly for older recipients or when expected ischemic time is short. Donor suitability must therefore be assessed individually rather than according to a strict age threshold alone. Acceptable causes of donor death include traumatic brain injury, intracranial hemorrhage, anoxic brain injury, and selected primary brain tumors without a significant risk of extracranial transmission. Particular caution is required when the cause of death or preceding clinical course raises concern for occult cardiovascular disease, myocardial injury, or systemic toxicity, such as after carbon monoxide poisoning.

Potential donors undergo extensive screening for transmissible infections, including human immunodeficiency virus (HIV), hepatitis B virus (HBV), hepatitis C virus (HCV), cytomegalovirus (CMV), Epstein–Barr virus (EBV), human T-cell lymphotropic virus (HTLV), and syphilis, together with microbiological assessment when clinically indicated. Active bloodstream infection is not necessarily an absolute contraindication if the causative organism is identified, appropriate antimicrobial therapy is instituted, and recipient treatment can be continued after transplantation. Hearts from HCV-viremic donors may now be transplanted into HCV-negative recipients provided that informed consent and a clear plan for post-transplant direct-acting antiviral therapy are established. Donor cardiac function is assessed by clinical evaluation, electrocardiography, echocardiography, laboratory testing, and, when appropriate, coronary angiography. Transient left ventricular dysfunction should be reassessed after donor hemodynamic optimization because reversible myocardial dysfunction is common after brain death. Coronary angiography is considered particularly in older donors or in those with risk factors for coronary artery disease, while significant obstructive coronary disease may limit graft suitability depending on its extent, recipient characteristics, and the possibility of surgical revascularization.

Donor-recipient matching includes ABO compatibility, immunological assessment, and appropriate graft sizing. Because allocation time is limited and complete HLA matching is rarely feasible, prospective HLA matching is not routinely required in heart transplantation; however, recipient sensitization and the presence of donor-specific antibodies remain clinically important. Graft size should preferably be assessed using predicted heart mass rather than body weight, height, BMI, or body surface area alone, particularly in recipients with elevated pulmonary vascular resistance or other conditions in which an undersized graft may be poorly tolerated. Ischemic time

should be minimized whenever possible. Traditionally, a cold ischemic time of less than approximately 4 hours has been considered desirable, although the effect of ischemic time is continuous rather than defined by a strict threshold and interacts with donor age, graft quality, and recipient risk.

Allocation systems determine which recipient receives an available donor heart. In October 2018, the United Network for Organ Sharing (UNOS) in the United States replaced the previous three-tier allocation system (Status 1A, 1B, and 2) with a six-tier system designed to provide more granular prioritization according to clinical urgency. The revised system gives the highest priority to patients with the most severe hemodynamic compromise, particularly those requiring temporary mechanical circulatory support, as well as selected patients with life-threatening ventricular arrhythmias or dependence on high-dose inotropic therapy. It also expanded the geographical sharing of donor organs. The new allocation policy reduced waiting-list time and mortality but was accompanied by increased use of temporary mechanical circulatory support and longer donor ischemic times because organs are transported over greater distances. Post-transplant outcomes have generally remained comparable after adjustment, although some early analyses reported higher rates of selected postoperative complications.

Several recent developments aim to expand the donor pool and increase access to transplantation. Donation after circulatory death (DCD) has emerged as an important source of donor hearts, with contemporary studies demonstrating post-transplant survival comparable to that achieved with donation after brain death (DBD). The introduction of DCD programs has increased the probability of transplantation and reduced the risk of death or clinical deterioration while patients remain on the waiting list. Advances in donor-heart preservation and transport represent another important development. Ex vivo normothermic perfusion systems allow the donor heart to remain perfused and beating during transport and have demonstrated clinical outcomes non-inferior to conventional static cold storage. Temperature-controlled hypothermic preservation systems have also shown encouraging results and may facilitate the safe transport of donor hearts over longer distances while minimizing the risks associated with uncontrolled cold ischemia.

3.5 Surgical Techniques and Anatomical Options of Heart Transplantation

The standard surgical procedure is orthotopic heart transplantation, in which the recipient's native heart is excised and the donor heart is implanted in the normal anatomical position. The operation is usually performed through a median sternotomy with cardiopulmonary bypass. After aortic cross-clamping, the recipient heart is excised while preserving appropriate atrial and vascular cuffs for implantation. In the bicaval technique, the superior vena cava and inferior vena cava are divided separately, the aorta and pulmonary artery are transected, and the left atrium is divided so

that a posterior cuff containing the pulmonary venous orifices remains in the recipient. The donor heart is prepared by trimming the great vessels to the required length and fashioning the left atrium into a single cuff around the pulmonary venous confluence. Two principal implantation techniques are used (Table 2).

In the biatrial technique, the donor left atrium is first anastomosed to the recipient left atrial cuff, followed by anastomosis of the donor right atrium to a retained cuff of the recipient right atrium; the pulmonary artery and aorta are then anastomosed. In the bicaval technique, the left atrial anastomosis is followed by separate anastomoses of the inferior and superior venae cavae, thereby preserving the anatomical integrity of the donor right atrium, after which the pulmonary artery and aorta are connected. Compared with the biatrial technique, the bicaval technique is associated with better preservation of right atrial geometry, a lower incidence of significant tricuspid regurgitation, and a lower requirement for permanent pacemaker implantation. After completion of the vascular anastomoses, the cardiac chambers are carefully de-aired, the aortic cross-clamp is removed, and the donor heart is reperfused. Temporary epicardial pacing wires are usually placed on the right atrium and right ventricle, and the patient is subsequently weaned from cardiopulmonary bypass once satisfactory graft function and hemodynamic stability have been achieved.

Table 3 Principal implantation techniques of heart transplantation (HTx - heart transplantation).

Technique	Principle	Characteristics and indications
Orthotopic, biatrial	Recipient heart removed; left and right atria anastomosed (atrial sutures end at the interatrial septum), then pulmonary artery and aorta.	Part of the recipient's right atrium remains in place.
Orthotopic, bicaval	Recipient heart removed; left atrial cuff, then separate anastomoses of the superior and inferior vena cava, pulmonary artery and aorta.	Donor right atrium remains intact; lower need for a pacemaker and shorter hospital stay.
Heterotopic	Donor heart placed alongside the recipient's own heart, which remains in situ; end-to-side aorto-aortic anastomosis and additional atrial/caval and pulmonary arterial connections.	Considered in pulmonary hypertension, small grafts, prolonged ischemic time and support during severe rejection.

ABO compatibility between donor and recipient remains an important principle in heart transplantation because preformed anti-A or anti-B antibodies can cause hyperacute antibody-mediated rejection with rapid graft dysfunction and thrombosis of the graft microvasculature. ABO-compatible transplantation is therefore standard practice in adults. However, ABO-incompatible heart transplantation can be performed successfully in selected recipients, particularly infants and young children, whose naturally occurring anti-A and anti-B antibody levels are low or absent. With appropriate recipient selection, antibody monitoring, and immunosuppressive management,

outcomes in pediatric ABO-incompatible transplantation have been shown to be comparable with those of ABO-compatible transplantation.

3.6 Immunosuppression and Postoperative Complications

Standard maintenance immunosuppression after heart transplantation usually consists of triple therapy combining a calcineurin inhibitor, most commonly tacrolimus or cyclosporine, an antiproliferative agent, usually mycophenolate mofetil or azathioprine, and corticosteroids. Induction therapy with lymphocyte-depleting or non-depleting antibodies may be used in selected patients to reduce the risk of early rejection and to permit delayed initiation or lower doses of calcineurin inhibitors. Corticosteroids are used routinely during the early post-transplant period and for the treatment of rejection episodes. Because of their long-term adverse effects, withdrawal may be considered within the first year in recipients at low immunological risk, although some patients require prolonged therapy. Calcineurin inhibitors remain a cornerstone of maintenance immunosuppression, but nephrotoxicity is one of their most important adverse effects. Mammalian target of rapamycin (mTOR) inhibitors, such as sirolimus and everolimus, may reduce the progression of cardiac allograft vasculopathy and allow reduction of calcineurin inhibitor exposure, but are associated with adverse effects including dyslipidemia, impaired wound healing, proteinuria, and cytopenias. Several newer immunomodulatory strategies are being investigated for selected patients with difficult-to-treat antibody-mediated rejection or sensitization, including anti-cluster of differentiation 38 (CD38) therapy, costimulation blockade with belatacept, interleukin-6 pathway inhibition, and immunoglobulin G-cleaving enzymes (IgG-cleaving enzymes).

Rejection is broadly classified as acute cellular rejection, antibody-mediated rejection, and hyperacute rejection. Acute cellular rejection is mediated predominantly by T lymphocytes and remains an important form of graft rejection, whereas antibody-mediated rejection results from donor-specific antibodies and associated microvascular injury. Antibody-mediated rejection is graded according to the International Society for Heart and Lung Transplantation (ISHLT) pathological classification from pathologic antibody-mediated rejection (pAMR) 0 to pAMR 3 on the basis of histopathological and immunopathological findings. Hyperacute rejection occurs immediately or very early after transplantation as a consequence of preformed recipient antibodies directed against donor antigens and may result in catastrophic graft failure. Endomyocardial biopsy remains the reference standard for diagnosing acute cellular rejection and is also important in the assessment of antibody-mediated rejection. Non-invasive surveillance methods, including gene-expression profiling and measurement of donor-derived cell-free deoxyribonucleic acid (DNA), are increasingly used in selected patients to reduce the need for routine biopsies, while additional

molecular and immunological tools for detecting both cellular and antibody-mediated rejection continue to evolve.

Primary graft dysfunction (PGD) is a major cause of early morbidity and mortality after heart transplantation. It is defined as severe ventricular dysfunction developing within the first 24 hours after transplantation in the absence of an identifiable secondary cause such as hyperacute rejection, pulmonary hypertension, or a surgical complication. Its pathophysiology is multifactorial and includes ischemia-reperfusion injury, donor-related myocardial dysfunction, recipient factors, and procedural factors such as prolonged ischemic time. Increasing donor age, prolonged ischemic time, and adverse recipient characteristics have been associated with a higher risk of PGD. Severe PGD may involve the left ventricle, right ventricle, or both of them and frequently requires temporary mechanical circulatory support. Veno-arterial ECMO is widely used for severe PGD and can provide effective temporary support while myocardial recovery occurs.

Cardiac allograft vasculopathy (CAV) is one of the major causes of late graft dysfunction and mortality after heart transplantation. Its pathogenesis involves both immunological mechanisms, including chronic endothelial injury and immune-mediated vascular inflammation, and non-immunological risk factors such as dyslipidemia, diabetes, obesity, donor age, and ischemic injury. Because the transplanted heart is denervated, myocardial ischemia is frequently clinically silent and patients may not develop typical angina. Conventional coronary angiography can underestimate early diffuse disease because CAV often produces concentric intimal thickening without focal stenosis; intravascular ultrasound therefore provides greater sensitivity for detecting early structural changes. Infectious complications remain an important cause of morbidity and mortality, particularly during periods of intense immunosuppression. CMV is among the most clinically important viral infections, while EBV is particularly relevant because of its association with post-transplant lymphoproliferative disease; bacterial, fungal, and other viral infections also contribute substantially to post-transplant complications. Malignancy is an important long-term complication of chronic immunosuppression, with skin cancers and post-transplant lymphoproliferative disease among the most frequent forms. Cardiac retransplantation is performed only in highly selected patients because of limited donor availability and generally less favorable outcomes than primary transplantation.

3.7 Outcomes and Future Directions

Contemporary outcomes after heart transplantation are favorable, with approximately three quarters or more of adult recipients surviving at least 5 years, although survival varies according to recipient characteristics, transplant era, and registry population. Durable LVAD therapy has also achieved substantial improvements in long-term outcomes; with the contemporary HeartMate 3

device, approximately 58% of patients were alive at 5 years in extended follow-up of the MOMENTUM 3 trial. Further advances in the treatment of advanced heart failure are expected from fully implantable mechanical circulatory support systems, improved donor-heart preservation, and strategies aimed at expanding the donor pool. An important recent milestone has been clinical cardiac xenotransplantation using hearts from genetically modified pigs. The first living human recipient received a genetically modified porcine heart in 2022 and survived for approximately two months. A second living recipient underwent transplantation in 2023 and also survived for approximately six weeks. These procedures demonstrated that genetic modification and modern immunosuppression can prevent immediate hyperacute xenograft rejection, but important immunological, infectious, and physiological barriers remain, and cardiac xenotransplantation is still an experimental therapy.

4 Ischemic Heart Disease (Tomáš Toporcer)

Ischemic heart disease results from an imbalance between myocardial oxygen supply and demand. In most cases, it is caused by atherosclerotic disease of the coronary arteries, although other mechanisms may also impair coronary blood flow or increase myocardial oxygen demand. Atherosclerosis is a chronic inflammatory disease of large and medium-sized arteries characterized by lipid accumulation within the arterial intima and the formation of atherosclerotic plaques that may progressively narrow the vascular lumen or cause acute arterial occlusion. It should be distinguished from the broader term arteriosclerosis, which encompasses several forms of arterial wall disease, including atherosclerosis, Mönckeberg medial calcific sclerosis, and arteriolosclerosis. Mönckeberg sclerosis is characterized predominantly by calcification of the media and internal elastic lamina and generally does not directly obstruct the arterial lumen, whereas arteriolosclerosis affects small arteries and arterioles and is characterized by thickening and remodeling of their walls. The distribution of atherosclerosis throughout the arterial system is heterogeneous. The aorta and coronary arteries are frequently affected, whereas some arterial territories, including the internal thoracic arteries (internal mammary artery), are relatively resistant to clinically significant atherosclerotic disease. A mature atherosclerotic plaque typically consists of a fibrous cap containing smooth muscle cells and extracellular matrix components, including collagen and proteoglycans, covering a lipid-rich necrotic core containing cholesterol, cellular debris, and inflammatory cells. Inflammation and neovascularization are important features of advanced plaques and contribute to plaque progression, intraplaque hemorrhage, and vulnerability. Many atherosclerotic plaques remain clinically silent and may progressively calcify, with the overall coronary atherosclerotic burden being assessable by imaging techniques such as coronary

artery calcium scoring. Plaques may become complicated by surface erosion or rupture, exposing thrombogenic material to circulating blood and resulting in thrombus formation. The thrombus may partially or completely occlude the vessel or may embolize distally, producing acute ischemia or infarction. Intraplaque hemorrhage may rapidly increase plaque volume and contribute to luminal obstruction and plaque instability. In contrast, gradual progression of a stable stenotic lesion may produce chronic myocardial ischemia, particularly when coronary blood flow can no longer increase sufficiently to meet myocardial oxygen demand. Acute plaque disruption with superimposed thrombosis is the principal mechanism underlying most acute coronary syndromes.

4.1 Clinical Presentations of Ischemic Heart Disease

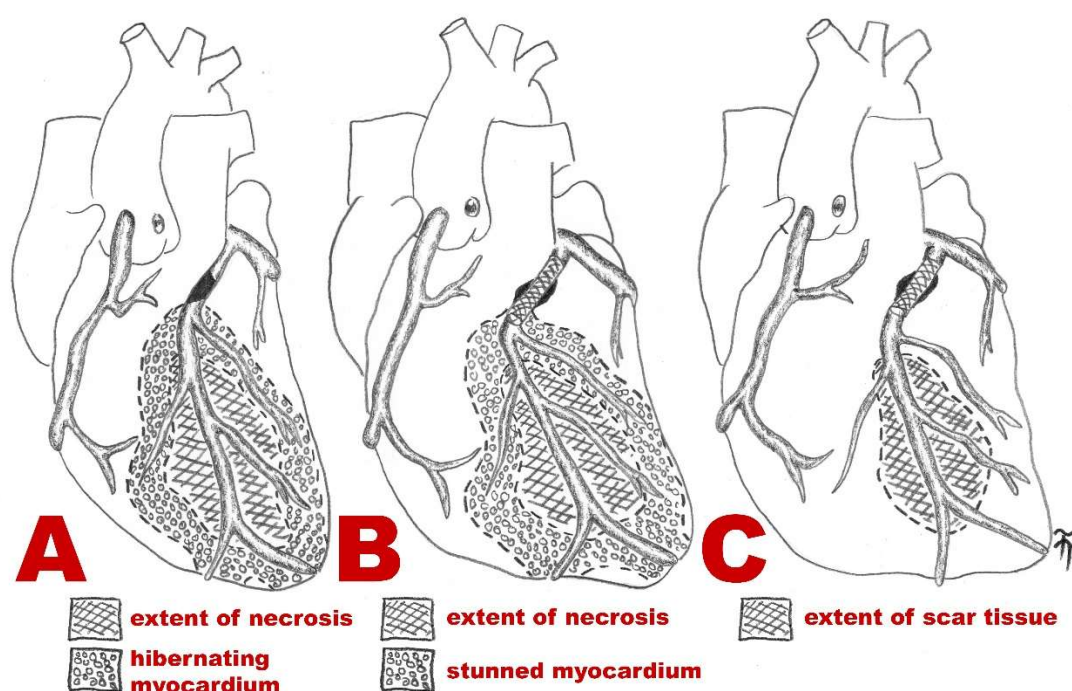


Figure 12 Dynamics of the extent of the akinetic and hypokinetic myocardial region during acute myocardial infarction (A - immediately after vessel lumen occlusion; B - early after revascularization; C - definitive state after healing of the lesion).

Ischemic heart disease (IHD) can present in various forms, spanning a broad spectrum of clinical severity. One of its most serious manifestations is sudden cardiac death, which in the setting of acute myocardial ischemia is most commonly caused by malignant ventricular arrhythmias, particularly ventricular fibrillation, or by severe acute pump failure resulting from extensive myocardial injury. When myocardial infarction is considered the likely cause of cardiac death but death occurs before diagnostic testing can confirm the diagnosis, the current Fifth Universal Definition of Myocardial Infarction no longer assigns the event to a separate numerical category; instead, the infarction should be classified, whenever possible, as primary, secondary, or procedure-related according to the clinical circumstances or post-mortem findings. Another important presentation of IHD within the spectrum of acute coronary syndromes (ACS) is ST-elevation

myocardial infarction (STEMI). On the electrocardiogram (ECG), ST-segment elevation is defined as new elevation at the J point in at least two contiguous leads of ≥ 1 mm in all leads except V2–V3, where the thresholds are ≥ 1.5 mm in women, ≥ 2.0 mm in men aged 40 years or older, and ≥ 2.5 mm in men younger than 40 years, in the absence of left ventricular hypertrophy or bundle branch block. Reciprocal ST-segment depression may also be present. A newly developed left bundle branch block alone is no longer considered diagnostic of acute myocardial infarction; in patients with left bundle branch block or ventricular pacing, additional ECG criteria, such as the Sgarbossa criteria, together with the clinical presentation and other diagnostic findings, should be used to identify acute coronary occlusion. The current universal definition also recognizes pathological Q waves as evidence of myocardial infarction, defined as a Q-wave duration of at least 40 ms and/or a depth of at least 25% of the corresponding R wave in two contiguous leads. Myocardial infarction results in the release of cardiac biomarkers into the circulation, with cardiac troponin I or T representing the preferred and most specific biomarker for the diagnosis of myocardial injury; creatine kinase (CK) and creatine kinase-MB (CK-MB) have largely lost their central diagnostic role when cardiac troponin assays are available. STEMI frequently results from acute thrombotic occlusion of an epicardial coronary artery, but the extent and transmural extent of myocardial necrosis depend on the duration of ischemia, collateral circulation, myocardial oxygen demand, and the timing of reperfusion. The principal therapeutic objective is therefore rapid restoration of blood flow in the infarct-related artery. Primary percutaneous coronary intervention (PCI) is the preferred reperfusion strategy when it can be performed within 120 minutes of STEMI diagnosis. When timely primary PCI cannot be achieved, fibrinolytic therapy is indicated within 12 hours of symptom onset in patients without contraindications, followed by transfer to a PCI-capable center and subsequent coronary angiography. In patients presenting within 12 hours of symptom onset, immediate reperfusion is recommended. In patients presenting later, between 12 and 48 hours after symptom onset, an invasive strategy should still be considered, particularly when a substantial amount of myocardium remains at risk. Irrespective of the time from symptom onset, immediate revascularization is indicated when there is persistent ischemia, cardiogenic shock or hemodynamic instability, acute severe heart failure, or life-threatening arrhythmia. Conversely, routine PCI of a persistently occluded infarct-related artery in an asymptomatic and clinically stable patient presenting late after myocardial infarction does not provide benefit. In hemodynamically stable patients with STEMI and multivessel coronary artery disease, complete revascularization of significant non-culprit lesions is generally recommended either during the index procedure or as a staged intervention, whereas routine immediate multivessel PCI should be avoided in patients presenting with cardiogenic shock. Emergency or urgent coronary artery bypass grafting (CABG)

should be considered in patients with acute myocardial infarction when PCI of the infarct-related artery is not feasible or has been unsuccessful and a large area of viable myocardium remains at risk, particularly in the presence of ongoing ischemia. In STEMI, current ACC/AHA guidance considers emergency or urgent CABG reasonable in this setting, whereas CABG after failed PCI is not recommended when there is no substantial myocardium at risk, when suitable surgical targets are absent, or when the expected operative risk exceeds the potential benefit. In patients with cardiogenic shock, emergency revascularization of the culprit vessel is indicated, with CABG reserved primarily for patients in whom PCI is not technically feasible or cannot provide adequate revascularization. The timing of surgery should be individualized according to hemodynamic stability, ongoing ischemia, coronary anatomy, the amount of jeopardized myocardium, and the anticipated surgical risk. Temporary mechanical circulatory support may be considered in selected patients with severe or refractory cardiogenic shock, but routine use of VA-ECMO solely as a prerequisite to PCI is not currently recommended. The myocardium affected by acute coronary occlusion is heterogeneous: the central region may undergo irreversible necrosis, whereas surrounding ischemic but still viable myocardium may be salvaged by timely reperfusion. This should be distinguished from myocardial hibernation, which refers to a state of chronically reduced regional contractile function associated with persistently reduced blood flow and preserved viability, with the potential for functional recovery after revascularization. Myocardial stunning, in contrast, is a transient but potentially prolonged reduction in contractile function that persists after restoration of adequate coronary blood flow despite preserved myocardial viability. Recovery may take hours to days or, in some circumstances, longer. The presence of extensive stunned myocardium after acute myocardial infarction or surgical revascularization may therefore contribute to temporary postoperative ventricular dysfunction and adversely affect early hemodynamic recovery (Figure 12).

The third major clinical presentation within the spectrum of acute coronary syndromes is non-ST-elevation myocardial infarction (NSTEMI). In contrast to STEMI, NSTEMI is not defined by the presence or absence of complete coronary occlusion or by the transmural extent of myocardial necrosis. It results from acute myocardial ischemia with myocardial necrosis in the absence of persistent ST-segment elevation fulfilling STEMI criteria. According to the current Universal Definition of Myocardial Infarction, the diagnosis requires acute myocardial injury, demonstrated by a rise and/or fall in cardiac troponin with at least one value above the sex-specific 99th percentile upper reference limit, together with evidence of myocardial ischemia, such as ischemic symptoms, new ischemic ECG changes, development of pathological Q waves, imaging evidence of a new regional wall-motion abnormality or loss of viable myocardium, or identification of an acute

coronary pathology on coronary imaging. ECG findings may include ST-segment depression, transient ST-segment elevation, T-wave inversion, or other dynamic ischemic changes, although the ECG may also be normal. Management is guided by clinical risk stratification. According to the 2023 ESC guidelines, patients with NSTEMI-ACS are stratified primarily according to the presence of very-high-risk or high-risk features. Very-high-risk features include hemodynamic instability or cardiogenic shock, recurrent or ongoing chest pain refractory to medical treatment, life-threatening arrhythmias or cardiac arrest after presentation, mechanical complications of myocardial infarction, acute heart failure presumed to be caused by ongoing myocardial ischemia, and recurrent dynamic ECG changes suggestive of ischemia, particularly intermittent ST-segment elevation. These patients should undergo an immediate invasive strategy. High-risk features include a confirmed diagnosis of NSTEMI, a Global Registry of Acute Coronary Events (GRACE) risk score greater than 140, transient ST-segment elevation, or dynamic ST-segment or T-wave changes. In these patients, an inpatient invasive strategy is recommended, and an early invasive strategy within 24 hours should be considered. When coronary anatomy favors surgical revascularization, the timing of CABG should be individualized according to the patient's clinical stability, ongoing ischemia, coronary anatomy, antiplatelet treatment, and overall procedural risk rather than determined by a fixed waiting period.

Another form of acute coronary syndrome is unstable angina. It is characterized by symptoms of myocardial ischemia without acute myocardial injury, that is, without the rise and/or fall in cardiac troponin required for the diagnosis of myocardial infarction. With the widespread use of high-sensitivity cardiac troponin assays, many patients who would previously have been classified as having unstable angina are now diagnosed with NSTEMI. The need for and timing of invasive assessment depend on the clinical presentation, recurrence or persistence of ischemic symptoms, ECG findings, and overall risk. When revascularization is indicated, the choice between PCI and CABG is based on coronary anatomy, disease complexity, ventricular function, comorbidities, surgical risk, and the expected completeness and durability of revascularization.

Stable angina is now considered within the broader spectrum of chronic coronary syndromes (CCS). It typically occurs when coronary blood flow is sufficient to meet resting myocardial oxygen requirements but becomes inadequate when oxygen demand increases, most commonly during physical exertion or emotional stress. Symptoms are usually reproducible and improve with rest or antianginal therapy. Chronic coronary syndromes encompass a broader range of clinical presentations than stable angina alone, including obstructive epicardial coronary artery disease, ischemia caused by coronary microvascular dysfunction or vasospasm, and patients who remain asymptomatic after previous coronary events or revascularization.

Myocardial ischemia may also be clinically silent, particularly in patients with diabetes, autonomic dysfunction, previous myocardial infarction, or advanced coronary artery disease. Silent ischemia does not necessarily lead to a progressive decline in left ventricular ejection fraction, although recurrent or extensive ischemia and previous silent myocardial infarction may contribute to ischemic left ventricular dysfunction and heart failure. Patients with chronic coronary syndrome should undergo appropriate diagnostic and risk assessment, and the decision regarding revascularization should be based on symptoms, extent of ischemia, coronary anatomy, left ventricular function, prognostic benefit, and patient preference. Heart Team discussion is particularly important when both PCI and CABG are reasonable options or when coronary anatomy and comorbidities make the optimal strategy uncertain.

In the management of ischemic heart disease, the first priority is to determine whether the patient presents with an acute coronary syndrome or with a chronic coronary syndrome. In acute coronary syndromes, particularly when acute coronary occlusion is present, myocardial injury may progress rapidly and timely restoration of coronary blood flow is essential to limit infarct size, preserve left ventricular function, and improve clinical outcomes. At the other end of the spectrum, patients with chronic coronary syndromes are generally hemodynamically stable, allowing the revascularization strategy to be selected according to the expected long-term benefit of PCI or CABG, taking into account the anatomical extent and complexity of coronary artery disease, left ventricular function, comorbidities, procedural risk, and patient preference.

4.2 Indications for Myocardial Revascularization

The indication for myocardial revascularization, whether by PCI or CABG, is determined by the patient's symptoms, the anatomical extent and functional significance of coronary artery disease, left ventricular function, and the expected prognostic or symptomatic benefit of the intervention, while taking the patient's comorbidities and procedural risk into account. The severity of symptoms can be quantified using standardized functional classifications. The Canadian Cardiovascular Society (CCS) classification is used to grade the severity of angina, whereas the New York Heart Association (NYHA) classification describes the functional limitation caused by heart failure symptoms, particularly exertional dyspnea (Table 4).

In symptomatic patients with suspected chronic coronary syndrome, the first step is to assess the clinical likelihood of obstructive coronary artery disease and to exclude alternative causes of the symptoms. Contemporary ESC recommendations use a risk factor-weighted clinical likelihood model incorporating age, sex, symptoms, and cardiovascular risk factors rather than relying on symptom classification alone. Patients with a very low or low clinical likelihood of obstructive coronary disease may not require further testing, whereas those with an intermediate or high

likelihood generally undergo non-invasive anatomical or functional imaging. Coronary computed tomography angiography (CCTA) is particularly useful for excluding obstructive coronary artery disease and for detecting non-obstructive atherosclerosis, whereas functional imaging, including stress echocardiography, myocardial perfusion imaging, or stress cardiac magnetic resonance, is preferred when assessment of inducible myocardial ischemia, viability, or the functional significance of a coronary lesion is required. Exercise ECG may still provide useful information on symptoms, exercise capacity, arrhythmias, blood pressure response, and ischemic ECG changes, but it is no longer the preferred first-line diagnostic test when modern imaging techniques are available. Invasive coronary angiography is indicated in patients with a very high clinical likelihood of obstructive disease, severe symptoms despite guideline-directed medical therapy, angina at a low level of exercise, high-risk findings on non-invasive testing, or when the results of non-invasive investigations remain uncertain. When angiographically intermediate stenoses are identified, their functional significance should generally be assessed using fractional flow reserve (FFR) or instantaneous wave-free ratio (iFR) before revascularization. Both methods quantify the hemodynamic significance of a coronary artery stenosis by invasive local measurement of blood pressure proximal and distal to the stenotic lesion.

Table 4 CCS and NYHA classifications.

CCS (Canadian Cardiovascular Society)	
0	No angina pectoris
1	Angina with prolonged exertion
2	Angina on walking quickly (or climbing more than 2 flights of stairs)
3	Angina on walking slowly for at least two blocks (or climbing less than 2 flights of stairs)
4	Angina with ordinary activities
NYHA (New York Heart Association)	
1	No limitation of activity, patient can climb ≥ 2 flights of stairs
2	Dyspnea after one flight of stairs
3	Patient unable to climb one flight of stairs
4	Dyspnea at rest

Table 5 European Society of Cardiology (2024) recommendations for myocardial revascularization (RCA – right coronary artery, LCX – circumflex branch of the left coronary artery, LAD – left anterior descending branch of the left coronary artery; Single-vessel disease – involvement of one of the three vessels LAD, LCX, RCA; Two-vessel disease – involvement of two of the three vessels LAD, LCX, RCA; Three-vessel disease – involvement of all three vessels LAD, LCX, RCA; SYNTAX – scoring system quantifying the anatomical severity of coronary artery disease)

		CABG	PCI
Single-vessel disease	Without proximal LAD	IIb	I
	With proximal LAD	I	I
Two-vessel disease	Without proximal LAD	IIb	I
	With proximal LAD	I	I
Left main coronary artery (LM) stenosis	SYNTAX 0-22	I	I
	SYNTAX 23-32	I	IIa
	High complexity / high surgical risk	I	IIb
Three-vessel disease	Low-to-intermediate complexity	I	I
	High complexity	I	CABG preferred
Three-vessel disease in a patient with diabetes mellitus	Standard/high surgical risk	I	CABG preferred
	Very high surgical risk	I	IIa

In simplified terms, myocardial revascularization should be considered when a coronary stenosis is both anatomically relevant and functionally significant, particularly when it supplies a substantial myocardial territory. Angiographic diameter stenosis alone is an imperfect predictor of functional significance, especially for intermediate lesions. When the angiographic finding is equivocal, additional invasive methods can be used to determine whether the stenosis is clinically relevant. Intravascular ultrasound (IVUS) provides detailed cross-sectional imaging of the coronary artery and allows measurement of the minimal lumen area (MLA) and assessment of plaque morphology. Its diagnostic role is particularly important in intermediate left main coronary artery stenosis. An MLA of approximately 6 mm² or greater is generally considered a reasonable threshold for safely deferring left main revascularization, whereas values between approximately 4.5 and 6 mm² usually require integration with invasive physiological assessment and other clinical and anatomical findings. For intermediate stenoses elsewhere in the coronary circulation, functional assessment using fractional flow reserve (FFR) or instantaneous wave-free ratio (iFR) is preferred. As stated above, both techniques use a pressure-sensing coronary guidewire to compare the pressure distal to

the stenosis with the proximal aortic pressure. FFR is measured during pharmacologically induced maximal hyperemia, most commonly using adenosine. A value ≤ 0.80 indicates a hemodynamically significant stenosis. iFR is measured without pharmacologically induced hyperemia, during a specific period of diastole when microvascular resistance is naturally low. A value ≤ 0.89 is considered hemodynamically significant. Both FFR and iFR are well-validated methods and are recommended for the physiological assessment of intermediate coronary stenoses before revascularization.

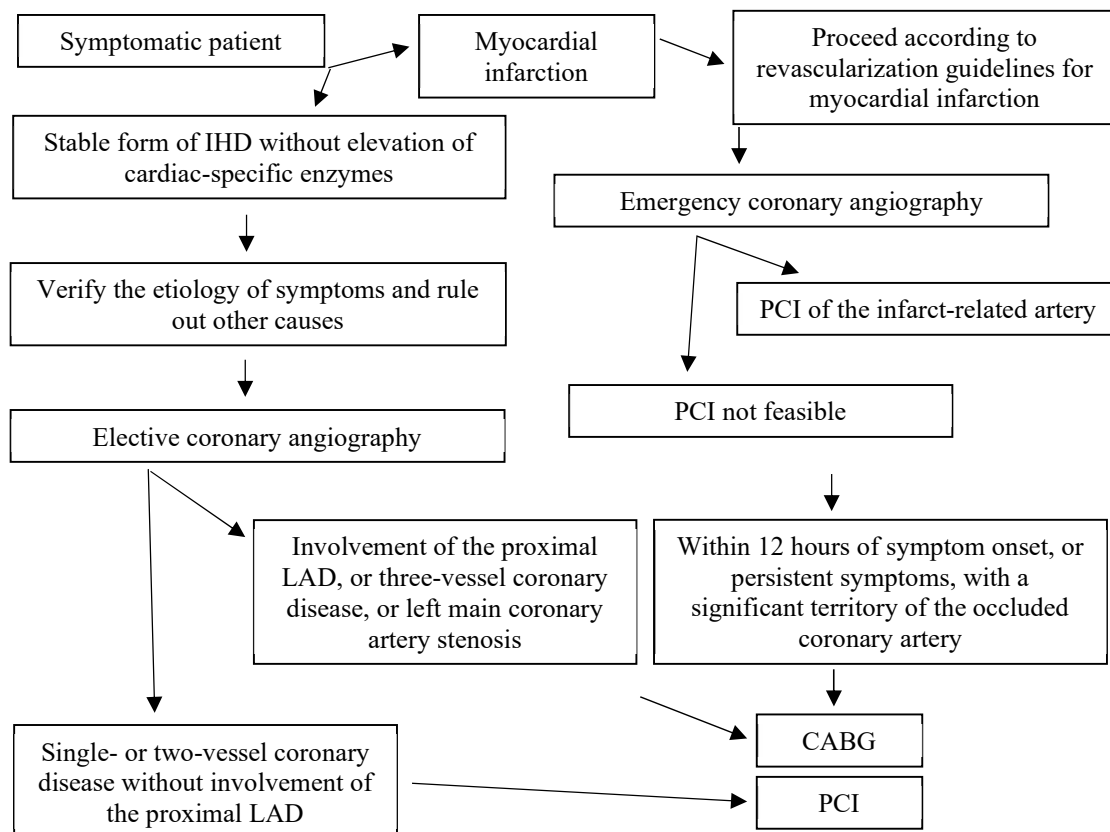


Figure 13 Simplified algorithm for the treatment of ischemic heart disease (CABG - coronary artery bypass grafting; PCI - percutaneous coronary intervention; LAD - left anterior descending branch of the left coronary artery).

When a clinically important stenosis is demonstrated in a vessel supplying a sufficiently large myocardial territory, the indication for revascularization is determined by symptoms, evidence of ischemia, prognostic benefit, coronary anatomy, and the expected completeness of revascularization. The choice between PCI and CABG should follow the current 2024 ESC Guidelines for the management of chronic coronary syndromes and should take into account the anatomical distribution and complexity of coronary artery disease, left ventricular function, diabetes and other comorbidities, surgical and interventional risk, and patient preference. The SYNTAX score may be used to quantify the anatomical complexity of coronary artery disease, particularly in patients with left main or multivessel disease, although it should not be used in isolation because it

does not incorporate physiological lesion significance or important clinical variables. Local expertise and institutional outcomes may influence the selected strategy, but clinical practice should remain consistent with current guideline recommendations.

In simplified terms, CABG is generally preferred over PCI in patients with anatomically complex multivessel coronary artery disease, particularly in the presence of diabetes mellitus, and in patients with significant left main coronary artery disease when the coronary anatomy is complex or complete revascularization by PCI is unlikely to be achieved. In patients with left main disease who are suitable for either revascularization strategy, CABG remains the overall preferred approach because it is associated with lower rates of spontaneous myocardial infarction and repeat revascularization during long-term follow-up; however, PCI is a reasonable alternative in patients with low-complexity left main disease when it can achieve completeness of revascularization comparable to CABG. In patients with complex multivessel disease, particularly those with diabetes mellitus and acceptable surgical risk, CABG is associated with better long-term survival than PCI and is therefore generally preferred. In contrast, in patients with single- or two-vessel coronary artery disease, with or without involvement of the proximal LAD, PCI and CABG provide broadly comparable outcomes in terms of death, myocardial infarction, and stroke, although PCI is less invasive and is therefore frequently preferred when coronary anatomy is suitable. CABG may nevertheless be favored in patients with complex LAD disease because PCI is associated with a higher long-term risk of repeat revascularization. When both PCI and CABG are technically feasible, the choice of revascularization strategy should be based on coronary anatomy and complexity, the likelihood of achieving complete revascularization, left ventricular function, diabetes and other comorbidities, surgical and interventional risk, and patient preference. In general, PCI is associated with faster recovery and lower early procedural morbidity, whereas CABG provides more durable revascularization and a lower need for subsequent repeat procedures. Both techniques continue to evolve. Contemporary drug-eluting stents have substantially reduced restenosis and repeat revascularization after PCI, while the use of arterial grafts improves the long-term durability of surgical revascularization compared with saphenous vein grafts. Minimally invasive and less invasive surgical techniques may further reduce postoperative morbidity and accelerate recovery in appropriately selected patients. Because the comparative outcomes of PCI and CABG continue to evolve with advances in technology, pharmacological treatment, and procedural techniques, revascularization decisions should follow current evidence-based guidelines and, particularly in complex cases, be made by a multidisciplinary Heart Team (Figure 13).

4.3 Surgical Myocardial Revascularization

The first attempts to surgically treat the symptoms of ischemic heart disease date back to the turn of the twentieth century. In 1899, François-Franck proposed interruption of the cardiac sympathetic pathways as a means of relieving angina. In 1916, Thomas Jonnesco performed left-sided cardiac sympathetic denervation for severe angina and associated ventricular arrhythmias, with symptomatic improvement. Various forms of cardiac sympathectomy and denervation were subsequently used during the first half of the twentieth century, although their principal effect was symptomatic relief rather than restoration of myocardial blood flow.

In 1910, Alexis Carrel described one of the earliest experimental forms of direct coronary revascularization, creating an aortocoronary bypass in a dog using an arterial graft. The experiment demonstrated the technical feasibility of directly bypassing an obstructed coronary artery but also highlighted the major limitation of the era: coronary anastomosis on the beating heart had to be completed within only a few minutes before severe myocardial ischemia and ventricular fibrillation developed. Carrel's pioneering work on vascular suturing and transplantation was recognized with the Nobel Prize in Physiology or Medicine in 1912 and provided the technical foundations for many later developments in cardiovascular surgery.

During the 1930s and 1940s, most surgical attempts at myocardial revascularization were based on indirect methods intended to stimulate collateral vessel formation or increase extracardiac blood supply to the myocardium. Claude Beck developed procedures in which vascularized tissues and inflammatory adhesions were brought into contact with the epicardium in an attempt to promote neovascularization. Other approaches included transposition of vascularized tissues such as omentum or muscle onto the heart and procedures designed to redirect blood through the coronary venous system. The Beck II procedure used an arterial graft to create an aorta-to-coronary-sinus fistula in an attempt to establish retrograde myocardial perfusion. In 1939, Davide Fieschi introduced bilateral ligation of the internal thoracic arteries, based on the hypothesis that this would increase flow through collateral branches supplying the heart; although symptomatic improvement was initially reported, the physiological rationale was subsequently questioned. A major advance in indirect myocardial revascularization was introduced by Arthur Vineberg, who implanted the internal thoracic artery directly into a tunnel within the left ventricular myocardium. Experimental work began in the 1940s, and the first clinical procedures were performed around 1950. Subsequent coronary angiography demonstrated the development of communications between the implanted internal thoracic artery and the native coronary circulation, providing objective evidence that the technique could augment myocardial perfusion. The Vineberg procedure became widely used during the 1950s and 1960s, with approximately 10,000–15,000 procedures performed worldwide

before it was replaced by direct coronary artery bypass grafting. Direct coronary revascularization began to develop rapidly during the late 1950s and 1960s. Goetz performed a successful internal thoracic artery-to-coronary bypass in 1960, Sabiston performed a hand-sewn saphenous vein coronary bypass in 1962, Garrett and colleagues performed another saphenous vein bypass in 1964, and Kolesov introduced clinically successful internal thoracic artery-to-coronary anastomosis. In 1967, René Favaloro systematically developed and popularized aortocoronary saphenous vein bypass grafting at the Cleveland Clinic, establishing the basis of modern CABG. Because several surgeons independently contributed essential technical developments during this period, the emergence of contemporary coronary bypass surgery is best regarded as an evolutionary process rather than the achievement of a single pioneer.

The objective of coronary artery bypass grafting (CABG) is to create a conduit that delivers blood to a coronary artery distal to a hemodynamically significant stenosis or occlusion, thereby restoring myocardial perfusion. CABG can be performed using several surgical strategies, and the preferred technique varies among institutions and according to patient characteristics. Conventional on-pump CABG with cardioplegic cardiac arrest remains the standard approach in many centers. The goal of surgical revascularization is generally to achieve complete and durable revascularization of all clinically relevant coronary territories whenever technically feasible. Although CABG has traditionally been based predominantly on anatomical assessment of coronary disease, contemporary decision-making increasingly incorporates the functional significance of individual lesions as well. In conventional on-pump CABG, the conduits are first harvested, cardiopulmonary bypass is established, and the heart is arrested using cardioplegia. Distal coronary anastomoses are then constructed during cardiac arrest. After completion of the distal anastomoses, the aortic cross-clamp is removed, myocardial reperfusion is restored, and any required proximal anastomoses are constructed, either during the same cross-clamp period or on the beating heart depending on the surgical strategy. The patient is subsequently weaned from cardiopulmonary bypass once satisfactory cardiac function and hemodynamic stability have been achieved. An alternative technique is off-pump coronary artery bypass grafting (OPCAB), in which the procedure is performed on the beating heart without cardiopulmonary bypass. After preparation of the conduits, the target coronary artery and surrounding myocardium are locally stabilized, allowing construction of the distal anastomosis while the remainder of the heart continues to beat. The principal technical challenge is maintaining adequate hemodynamics while the heart is displaced to expose the target coronary vessel, particularly during revascularization of the lateral and posterior walls. Off-pump CABG can nevertheless be used for multivessel revascularization in experienced centers and is also frequently incorporated into minimally invasive and hybrid coronary

revascularization strategies. Compared with conventional on-pump CABG, off-pump surgery may reduce some complications related to cardiopulmonary bypass in selected high-risk patients, but several studies have reported a higher risk of incomplete revascularization and lower graft patency when the procedure is performed by less experienced teams.

For every coronary revascularization procedure, the choice of the optimal conduit must be considered according to the characteristics of the patient, the target coronary artery, and the expected long-term benefit. In clinical practice, coronary bypass conduits are predominantly venous or arterial. The great saphenous vein is the most commonly used venous conduit, whereas the small saphenous vein and, much less frequently, other superficial veins may be used when suitable. An important advantage of saphenous vein grafts (SVGs) is their relatively simple and rapid harvesting, which can be performed simultaneously with preparation of the chest. They are also comparatively resistant to perioperative vasospasm and can therefore provide reliable early graft flow even in patients requiring substantial vasoactive support. Their principal limitation is inferior long-term durability compared with arterial grafts. Early SVG failure is most commonly related to endothelial injury and thrombosis, whereas subsequent failure results from intimal hyperplasia and progressive graft atherosclerosis. Atherogenesis becomes increasingly important several years after implantation and represents the dominant mechanism of late SVG failure. Intensive secondary prevention, particularly lipid-lowering therapy, reduces the progression of graft atherosclerosis. Historically, approximately half of conventional saphenous vein grafts remain patent at 10 years, although contemporary harvesting techniques, medical therapy, and secondary prevention may improve these outcomes. Arterial conduits generally provide superior long-term patency. The left internal thoracic artery (LITA) is the preferred conduit for revascularization of the left anterior descending artery because of its exceptional long-term durability and resistance to atherosclerosis. These properties are related to several structural and biological characteristics of the vessel wall, including preservation of endothelial function, a relatively resistant internal elastic lamina, and production of vasoactive and antithrombotic mediators such as nitric oxide and prostacyclin. The right internal thoracic artery may be used as an additional arterial conduit, although bilateral internal thoracic artery harvesting increases the risk of sternal wound complications, particularly in patients with diabetes, obesity, or other risk factors for impaired wound healing. Skeletonized harvesting can reduce this risk. The radial artery is another important arterial conduit and is currently preferred over the saphenous vein for grafting the second most important coronary target after the LAD when anatomy is suitable. Before harvesting, adequate collateral perfusion of the hand through the ulnar artery and palmar arch should be confirmed. The radial artery is particularly sensitive to competitive flow and should therefore preferentially be used for coronary vessels with severe or functionally

significant stenosis, ideally subocclusive lesions. Earlier recommendations commonly used angiographic thresholds of greater than 70% stenosis for left-sided targets and approximately 90% for the right coronary artery; contemporary guidance emphasizes severe stenosis and avoidance of substantial competitive native flow rather than relying solely on a single numerical threshold. A disadvantage of arterial grafts, particularly the radial artery, is their greater susceptibility to vasospasm, which may compromise early graft flow; pharmacological antispasmodic therapy is therefore commonly used. Grafts selection should consequently be individualized according to the patient's age and expected survival, coronary anatomy, degree of target-vessel stenosis, ventricular function, comorbidities, risk of wound complications, availability and quality of individual grafts, and the likelihood of achieving complete and durable revascularization. Younger patients with a long expected lifespan may benefit most from multiple arterial grafts or, in selected cases, total arterial revascularization, whereas saphenous vein grafts remain appropriate and frequently necessary in patients in whom arterial conduits are unsuitable, unavailable, or unlikely to provide additional long-term benefit.

For venous grafts, the distal anastomosis is constructed on the target coronary artery beyond the significant stenosis, while the proximal anastomosis is usually created on the ascending aorta, but may also be connected to another venous graft or, less commonly, to an arterial conduit as part of a composite graft configuration. When the internal thoracic artery is used, the distal anastomosis is likewise constructed on the coronary artery distal to the stenosis. Whenever possible, its proximal origin from the subclavian artery is preserved, allowing the conduit to function as an in situ graft. If the native origin cannot be retained because of graft length, anatomical constraints, or the planned revascularization strategy, the internal thoracic artery may be used as a free graft and connected proximally to the ascending aorta or to another arterial conduit, most commonly as part of a composite Y- or T-graft. The radial artery is generally used as a free graft and therefore requires both proximal and distal anastomoses. In addition to a terminal end-to-side distal anastomosis, sequential grafting may be performed by constructing one or more side-to-side anastomoses between the conduit and additional coronary targets along its course, followed by a terminal distal anastomosis. This technique can be used with both arterial and venous conduits and allows a single graft to revascularize several coronary territories. Adequate graft flow depends on appropriate conduit length and geometry, the absence of tension or kinking, the severity of native coronary stenosis, and a sufficiently large and well-developed distal vascular bed. Poor runoff, competitive native coronary flow, technical anastomotic problems, or conduit injury may reduce graft flow and increase the risk of early graft thrombosis. With appropriate conduit selection, graft configuration,

and surgical technique, CABG provides durable myocardial revascularization and excellent long-term outcomes in appropriately selected patients.

5 Most Common Mechanical Complications of Myocardial Infarction (Tomáš Toporcer)

Mechanical complications of myocardial infarction (MI) usually develop during the first several days after the onset of infarction and may occur regardless of whether coronary revascularization has been performed. Delayed or absent reperfusion increases their risk because a larger amount of myocardium undergoes irreversible necrosis. The principal mechanical complications include ventricular free-wall rupture, contained ventricular rupture, ventricular septal rupture, and acute mitral regurgitation caused by papillary muscle rupture. The term contained rupture is currently preferred by the AHA/ACC for a lesion traditionally referred to as a post-infarction ventricular pseudoaneurysm. Mitral regurgitation after myocardial infarction may also develop without papillary muscle rupture as a consequence of ischemic left ventricular remodeling and papillary muscle displacement, resulting in secondary ischemic mitral regurgitation. In contrast, acute severe mitral regurgitation usually results from partial or complete papillary muscle rupture. Patients with suspected mechanical complications require immediate echocardiographic assessment and should be managed in a center with cardiac surgical expertise. Short-term mechanical circulatory support may be used for hemodynamic stabilization as a bridge to definitive treatment when required. Definitive management depends on the specific complication and the patient's hemodynamic condition. Ventricular free-wall rupture and acute severe mitral regurgitation caused by papillary muscle rupture generally require emergency surgical treatment. In post-infarction ventricular septal rupture, surgical repair remains the definitive treatment, but the optimal timing is individualized. Emergency surgery is required in patients with refractory cardiogenic shock or progressive hemodynamic deterioration, whereas in selected patients who can be stabilized, temporary mechanical circulatory support may permit delayed repair. Delaying surgery beyond the acute phase may allow recovery of end-organ dysfunction and maturation and fibrosis of the infarcted myocardium, which can facilitate a more durable repair; however, this strategy carries the competing risk of sudden clinical deterioration while awaiting surgery. Observational data show lower operative mortality when repair is delayed beyond approximately 7 days, although this finding is affected by substantial survivor and selection bias and should not be interpreted as a universal recommendation to postpone surgery.

5.1 Cardiac Wall Rupture

Ventricular free-wall rupture was described at autopsy as early as the seventeenth century, and successful surgical repair became possible only with the development of modern cardiac surgery. Today, ventricular free-wall rupture is recognized as one of the most severe mechanical complications of acute myocardial infarction. It usually occurs within the first several days after infarction and is associated predominantly with transmural myocardial necrosis. Its incidence has declined substantially in the era of early reperfusion therapy. Clinically, free-wall rupture may present catastrophically with sudden circulatory collapse and electromechanical dissociation, or more gradually with pericardial effusion, cardiac tamponade, and cardiogenic shock. In some patients, rupture is contained by adherent pericardium, thrombus, or scar tissue, producing what has traditionally been termed a ventricular pseudoaneurysm; current AHA/ACC guidance prefers the term contained rupture. Unlike a true ventricular aneurysm, a contained rupture does not contain the full normal layers of the ventricular wall and therefore corresponds more closely to the histopathological definition of a pseudoaneurysm; it also carries a substantially greater risk of subsequent rupture. It is frequently located in the inferior or posterolateral region of the left ventricle. In patients with acute free-wall rupture and cardiac tamponade, emergency surgical treatment is generally required, and echocardiography is the principal diagnostic modality. Diagnostic coronary angiography should not delay life-saving surgery in a hemodynamically unstable patient. In contained rupture, the indication and timing of intervention depend on clinical presentation, size, location, evidence of expansion, time from myocardial infarction, and operative risk. Acute and subacute post-infarction contained ruptures are generally considered for urgent repair because the risk of rupture is greatest early after infarction, whereas management of small, stable, chronic lesions may be individualized. Contemporary reviews most often regard lesions presenting within approximately 3 months after myocardial infarction, particularly large or symptomatic lesions, as candidates for surgical treatment rather than applying a fixed 6-month threshold. Coronary angiography is usually performed before surgery when the patient is hemodynamically stable, and concomitant CABG may be undertaken when significant revascularizable coronary artery disease is present.

The objective of surgery is to exclude the rupture cavity from the left ventricular circulation and restore structural integrity of the ventricular wall. The operative technique depends on the size, location, morphology, and quality of the surrounding myocardium. Small defects with sufficiently firm tissue may be closed directly using reinforced sutures, whereas larger defects are commonly repaired with a patch anchored to viable myocardium. Patch repair may be performed externally or from within the ventricular cavity, depending on the anatomy of the lesion. In selected cases of

oozing-type free-wall rupture, sutureless patch techniques using biological or synthetic material and surgical adhesive have also been described. Particular care is required to avoid distortion of ventricular geometry and the subvalvular mitral apparatus, especially when the defect is located close to the posteromedial papillary muscle. Excessive alteration of ventricular geometry or papillary muscle displacement may result in new or worsened mitral regurgitation.

5.2 Ventricular Septal Defect

The first pathological descriptions of post-infarction ventricular septal rupture (VSR) date back to the nineteenth century. The first successful surgical repair was reported by Denton Cooley and colleagues in 1957. In the pre-reperfusion era, VSR complicated approximately 1–2% of acute myocardial infarctions, whereas with contemporary reperfusion therapy its incidence has fallen to well below 1%. Anterior VSR usually follows infarction in the territory of the left anterior descending artery and is often associated with a relatively direct defect involving the anterior or apical septum. Posterior VSR more commonly follows inferior myocardial infarction caused by occlusion of the right coronary artery or, less frequently, the circumflex artery and may have a more complex, irregular, or serpiginous morphology. Posterior defects are also more frequently associated with right ventricular dysfunction and concomitant ischemic mitral regurgitation. The hemodynamic consequences depend on the size of the defect, ventricular function, pulmonary and systemic vascular resistance, and the magnitude of the left-to-right shunt. A large defect may result in marked pulmonary overcirculation, right ventricular volume and pressure overload, reduced effective systemic cardiac output, and rapidly progressive biventricular failure or cardiogenic shock. Clinically, patients may develop sudden hemodynamic deterioration several days after myocardial infarction, frequently accompanied by a new harsh pansystolic murmur and a palpable precordial thrill. Echocardiography with color Doppler is the principal diagnostic modality and allows identification of the defect, estimation of shunt severity, and assessment of ventricular function and associated valvular abnormalities. In selected hemodynamically stable patients, cardiac CT or magnetic resonance imaging may provide additional anatomical information, although these modalities should not delay definitive treatment.

Historically, delayed repair was advocated because acutely infarcted myocardium is friable and poorly suited to secure suturing, whereas several weeks after infarction the margins of the defect become more fibrotic and mechanically stronger. Contemporary management, however, does not use a fixed minimum interval between myocardial infarction and surgery. Surgical repair remains the definitive treatment for most patients, but the timing should be individualized according to hemodynamic stability, the degree of end-organ dysfunction, shunt magnitude, ventricular function, and the feasibility of temporary mechanical circulatory support. Patients with refractory cardiogenic

shock or progressive hemodynamic deterioration generally require urgent or emergency intervention, whereas selected patients who can be stabilized may benefit from delayed repair, allowing recovery of end-organ function and maturation of the infarcted tissue. Observational studies consistently report lower operative mortality when surgery can be delayed beyond the first several days, particularly beyond approximately 7 days, although these findings are strongly affected by survivor and selection bias. Without definitive repair, post-infarction VSR carries an extremely poor prognosis, with mortality approaching 80–90% within the first one to two months in historical series. Medical stabilization aims to reduce left-to-right shunting and maintain systemic perfusion by optimizing preload, reducing excessive systemic vascular resistance when tolerated, and supporting cardiac output. Intra-aortic balloon counterpulsation can reduce left ventricular afterload and shunt volume and remains a useful temporary stabilization strategy in selected patients. Current guidelines also consider short-term mechanical circulatory support reasonable as a bridge to surgery when necessary. More advanced support, including VA-ECMO, may be used in profound or refractory shock, but device selection should be individualized because VA-ECMO increases systemic arterial pressure and left ventricular afterload and may therefore increase shunting unless appropriate ventricular unloading is achieved. Evidence comparing different support strategies remains observational, and routine use of any single device cannot currently be recommended for all patients with post-infarction VSR.

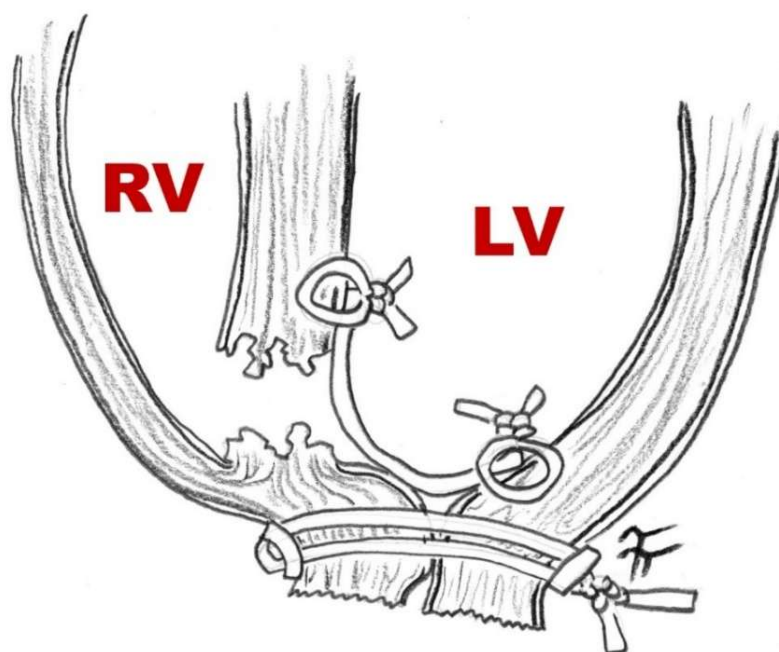


Figure 14 Exclusion technique for suturing a ventricular septal defect according to David, Colley, and Ross (LV - left ventricle, RV - right ventricle).

Once the infarcted myocardium begins to organize and firmer tissue develops around the defect, surgical repair may become technically more favorable. However, contemporary management does

not require a fixed waiting period of 5–7 days after the development of ventricular septal rupture (VSR). Patients with refractory cardiogenic shock or progressive hemodynamic deterioration require urgent or emergency repair, whereas in selected patients who can be stabilized, surgery may be delayed for several days to allow partial maturation of the infarcted myocardium and recovery of end-organ function. In hemodynamically unstable patients in whom immediate surgery is considered excessively high risk, temporary VA-ECMO support may be used as a bridge to delayed repair, allowing stabilization and, when clinically feasible, postponement of surgery for approximately 5–7 days. This interval may permit further organization and fibrosis of the infarcted myocardium, providing firmer and better-demarcated tissue for surgical reconstruction. Repair performed in this delayed setting may therefore be technically easier and may reduce the risk of sutures tearing through friable myocardium and the subsequent development of residual or recurrent septal defects. From our clinical experience, the most unfavorable situation is an acute post-infarction VSR in a hemodynamically unstable patient in whom VA-ECMO or another form of temporary mechanical circulatory support cannot be established, making emergency repair in freshly infarcted tissue unavoidable. The surgical approach and repair technique depend on the location, size, and morphology of the defect and on the extent of the surrounding infarction. A left ventriculotomy through the infarcted myocardium is commonly used to access the defect. In apical defects, infarctectomy with resection of the affected apex and direct or patch reconstruction may be performed according to principles associated with the Daggett technique. Anterior defects may also be repaired by direct septal reconstruction or patch closure, whereas posterior defects are technically more demanding because of their frequently complex, irregular, or serpiginous anatomy and their proximity to the mitral apparatus. Traditional techniques involved infarctectomy followed by reconstruction of the ventricular septum and free ventricular wall with reinforced sutures or patches. A major development was the infarct-exclusion technique introduced by Komeda and David and subsequently refined by David and colleagues. In this method, a pericardial patch is sutured to viable left ventricular endocardium around the infarcted area, thereby excluding both the septal rupture and the infarcted myocardium from the functional left ventricular cavity without excising the necrotic tissue. The left ventriculotomy is then closed over the excluded infarcted area. This technique may preserve ventricular geometry, reduce stress on friable infarcted tissue, and limit injury to the right ventricle and has become one of the most widely used approaches for surgical repair of post-infarction VSR. Transcatheter closure has also emerged as an option in selected patients with suitable anatomy, particularly when surgical risk is prohibitive, as a bridge to surgery, or for treatment of a residual postoperative shunt. Device closure is technically more favorable when

the defect has well-defined margins and sufficient surrounding tissue for secure device anchoring; Amplatzer-type occluders are most commonly used for this purpose.

The postoperative course is determined largely by the extent of the underlying myocardial infarction and the degree of preoperative hemodynamic compromise. Important complications include low cardiac output syndrome, persistent or recurrent VSR, right or left ventricular failure, bleeding, renal and respiratory failure, infection, arrhythmias, stroke, and progressive ventricular remodeling with secondary mitral regurgitation. Small residual shunts may be managed conservatively when hemodynamically insignificant, whereas clinically relevant residual defects should be considered for repeat surgical or transcatheter closure according to the magnitude of the shunt, symptoms, ventricular loading, and overall clinical condition. A pulmonary-to-systemic blood flow ratio (Q_p/Q_s) ratio greater than approximately 1.5 may support intervention when accompanied by significant volume overload or clinical consequences, but should not be used as the sole criterion. Despite advances in surgical technique, mechanical circulatory support, and perioperative care, post-infarction VSR remains associated with substantial early morbidity and mortality, particularly in patients presenting with cardiogenic shock, right ventricular dysfunction, posterior defects, or multiorgan failure. In patients who survive successful repair, however, long-term functional status and quality of life may be satisfactory rather than uniformly poor.

5.3 Mitral Regurgitation in Myocardial Infarction

The introduction of early reperfusion therapy has substantially reduced the incidence and severity of mechanical complications after myocardial infarction, including post-infarction mitral regurgitation (MR). Nevertheless, MR remains relatively common after acute MI and may result from transient ischemic papillary muscle dysfunction, left ventricular remodeling with leaflet tethering, or structural disruption of the mitral apparatus. Mild or moderate ischemic MR may improve after successful revascularization as myocardial ischemia resolves and stunned myocardium recovers, whereas MR caused by established ventricular remodeling or structural damage may persist. This distinction is clinically important because the indications for intervention differ between acute structural MR and chronic secondary ischemic MR, and because the expected results of mitral valve surgery also differ according to the underlying mechanism.

Acute severe mitral regurgitation caused by papillary muscle rupture is a true mechanical complication of myocardial infarction and represents an irreversible structural lesion. It most often develops within the first week after MI and typically presents with sudden hemodynamic deterioration, pulmonary edema, and low cardiac output; a prominent systolic murmur may be present but can be soft or even absent in severe acute MR because of rapid equalization of left ventricular and left atrial pressures. The posteromedial papillary muscle is more vulnerable because

it usually has a single dominant arterial supply, most commonly from the posterior descending artery arising from either the right coronary artery or, in a left-dominant circulation, the circumflex artery. In contrast, the anterolateral papillary muscle generally has a dual blood supply from branches of the left anterior descending and circumflex arteries and is therefore less frequently affected. Echocardiography is the diagnostic method of choice and demonstrates severe acute MR, flail leaflet motion, and, in many cases, partial or complete papillary muscle rupture. According to the Carpentier classification, this lesion produces type II leaflet motion (the Carpentier classification is presented in the following chapter). Emergency mitral valve surgery is the standard treatment. Mitral valve replacement is most commonly performed because the infarcted and friable papillary muscle tissue often makes durable repair difficult; however, in selected patients with partial papillary muscle rupture and stable hemodynamics, valve repair may be feasible in experienced centers. Concomitant coronary revascularization should be performed when indicated by coronary anatomy and myocardial viability.

Chronic ischemic mitral regurgitation is a form of secondary MR caused by adverse left ventricular remodeling after myocardial infarction, with papillary muscle displacement, leaflet tethering, and impaired leaflet coaptation despite structurally normal leaflets. Clinical presentation ranges from predominant ischemic symptoms to progressive heart failure. Revascularization alone may improve MR when viable myocardium recovers and reverse remodeling occurs, but this response is unpredictable, particularly when significant ventricular remodeling and leaflet tethering are already present. In patients with mild ischemic MR undergoing CABG, concomitant mitral intervention is generally not indicated. In moderate ischemic MR, randomized data have shown that adding mitral repair to CABG reduces the prevalence of residual MR but does not improve survival or left ventricular reverse remodeling and is associated with additional perioperative morbidity; therefore, routine concomitant mitral repair is not recommended for every patient and the decision should be individualized. In severe secondary ischemic MR requiring surgery, either restrictive annuloplasty repair or chordal-sparing valve replacement may be considered according to ventricular geometry, leaflet tethering, scar distribution, and the predicted risk of recurrent MR. Randomized trials have demonstrated substantially higher recurrence of moderate or severe MR after repair than after chordal-sparing replacement, although survival was similar between strategies. Replacement is therefore often favored when echocardiographic or anatomical features suggest a high risk of recurrent MR after repair, whereas repair remains appropriate in carefully selected patients with favorable valve and ventricular geometry.

5.4 Left Ventricular Aneurysm

A further late complication of myocardial infarction is the development of a left ventricular aneurysm in the region of transmural myocardial injury. The affected ventricular wall becomes thinned, fibrotic, and akinetic or dyskinetic, resulting in adverse remodeling, increased wall stress, and a reduction in effective left ventricular stroke volume. Unlike ventricular free-wall rupture, ventricular septal rupture, or acute mitral regurgitation, a left ventricular aneurysm usually develops gradually and does not typically require emergency treatment. Over time, however, it may contribute to progressive heart failure, ventricular arrhythmias, thrombus formation with systemic embolization, and recurrent ischemic events. Surgical resection of a post-infarction left ventricular aneurysm using cardiopulmonary bypass was first reported by Cooley and colleagues in 1958. Ischemic heart disease is by far the most common cause of acquired left ventricular aneurysm, although aneurysmal remodeling may also occur in other conditions, including trauma, Chagas disease, and inflammatory or infiltrative myocardial disease. Post-infarction aneurysms most commonly involve the anterior or apical left ventricular wall following infarction in the territory of the left anterior descending artery. During the healing process, necrotic myocardium is progressively replaced by fibrous scar tissue, producing a non-contractile or paradoxically moving segment. Early reperfusion therapy substantially reduces infarct size and therefore also reduces the likelihood of subsequent aneurysm formation. The clinical consequences of a left ventricular aneurysm depend mainly on its size, location, effect on global ventricular geometry and function, associated mitral regurgitation, presence of ventricular arrhythmias, and formation of intracavitary thrombus.

Diagnosis is based primarily on echocardiography, while cardiac magnetic resonance provides more precise assessment of ventricular volumes, scar distribution, myocardial viability, thrombus, and the distinction between a true aneurysm and contained rupture. Left ventriculography may also demonstrate aneurysm morphology but is now used less frequently solely for this purpose. Surgical ventricular reconstruction should be considered in selected symptomatic patients with a large left ventricular aneurysm associated with refractory heart failure, persistent angina despite appropriate revascularization, recurrent malignant ventricular arrhythmias, or systemic embolization from an intracavitary thrombus. Ventricular volumes and the extent of dyskinetic or akinetic myocardium are important components of the assessment, but no single end-systolic or end-diastolic volume threshold should be used as an absolute indication for surgery. The aim of surgical treatment is to exclude the non-functional aneurysmal segment, restore a more physiological left ventricular size and geometry, reduce wall stress, and preserve the mitral subvalvular apparatus. Small selected aneurysms may be treated by linear closure or plication, whereas larger lesions usually require

ventriculotomy through scarred myocardium followed by aneurysmectomy and linear or patch reconstruction. Endoventricular patch reconstruction can be used when preservation or restoration of ventricular geometry is particularly important. Care must be taken to avoid excessive reduction of the ventricular cavity and displacement of the papillary muscles, as both may impair left ventricular filling or induce or worsen mitral regurgitation.

6 Acquired Valvular Heart Disease (Tomáš Toporcer)

The aortic valve is the final valve in the cardiac circulation and lies at the junction between the left ventricular outflow tract and the aorta. Aortic valve disease most commonly results from pathology of the valve cusps themselves and less frequently from alterations in the geometry of the aortic root. The most common aortic valve lesion is aortic stenosis, which classically presents with a triad of symptoms: angina pectoris, dyspnea or manifestations of heart failure, and syncope. On physical examination, a systolic murmur is typically present. Aortic regurgitation is less common and is characteristically associated with a diastolic murmur.

6.1 Aortic Stenosis

Aortic stenosis (AoS) is the most common acquired valvular heart disease and affects approximately 2% of individuals over 65 years of age. Its prevalence increases substantially with age and is approximately twice as high in patients aged 75–85 years as in those aged 65–75 years. Etiologically, AoS most commonly develops as a degenerative process caused by chronic mechanical stress on the valve cusps. This process is associated with inflammation, lipid accumulation, macrophage infiltration, progressive cusp thickening, and calcification. Less commonly, aortic stenosis may result from Paget disease, alkaptonuria, congenital valve abnormalities, or rheumatic disease.

A normal aortic valve has an effective orifice area greater than 2.5 cm², typically approximately 3–4 cm². Aortic stenosis increases left ventricular afterload and leads to compensatory hypertrophy of the left ventricular myocardium. This process may be partly modified by treatment with angiotensin-converting enzyme (ACE) inhibitors. Progressive hypertrophy is associated with reduced ventricular compliance and an increase in left ventricular end-diastolic pressure (LVEDP), while adequate ventricular filling becomes increasingly dependent on preserved sinus rhythm and atrial contraction. The increased pressure load across the stenotic aortic valve prolongs left ventricular systolic ejection and may shorten the effective duration of diastole. Prolonged and excessive systolic work increases myocardial oxygen and energy requirements. Diastolic dysfunction, increased LVEDP, reduced diastolic coronary perfusion time, and the increased metabolic demands of the hypertrophied myocardium may therefore result in myocardial ischemia

even in the absence of obstructive coronary artery disease. AoS may consequently present with angina pectoris and symptoms of heart failure despite normal epicardial coronary arteries. Progressive ventricular dysfunction leads to a reduction in cardiac output, an increase in left atrial and pulmonary venous pressures, reflected by an elevated pulmonary artery wedge pressure, and eventually to pulmonary hypertension. A less common clinical association is Heyde syndrome, characterized by gastrointestinal bleeding caused by the combination of aortic stenosis, acquired von Willebrand factor dysfunction, and bleeding from intestinal angiodysplasia.

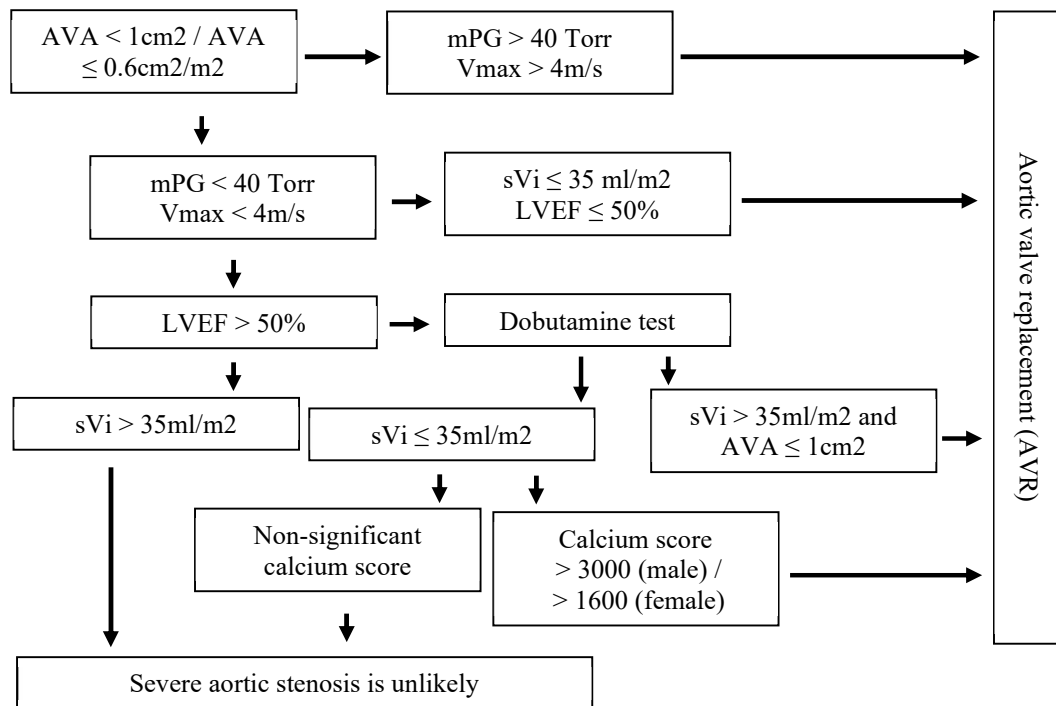


Figure 15 Diagnostic strategy for severe aortic stenosis (AVA - aortic valve area, LVEF - left ventricular ejection fraction, mPG - mean pressure gradient across the aortic valve, sVi - indexed left ventricular stroke volume, Vmax - maximum velocity across the aortic valve.)

The severity of aortic stenosis is assessed primarily using echocardiographic parameters. Severe aortic stenosis is typically characterized by an aortic valve area of less than 1.0 cm^2 , or less than $0.6\text{ cm}^2/\text{m}^2$ when indexed to body surface area, a mean transvalvular pressure gradient greater than 40 mmHg , and a peak transvalvular velocity greater than 4.0 m/s . In a symptomatic patient who meets these criteria, intervention on the aortic valve is indicated. As left ventricular dysfunction progresses, with a reduction in left ventricular ejection fraction (LVEF) below 50% and a stroke volume index below 35 mL/m^2 , the ventricle may no longer be able to generate sufficient flow to produce the expected pressure gradient across the stenotic valve. In such patients, severe aortic stenosis may still be present despite lower transvalvular gradients and velocities, provided that the valve area remains below 1.0 cm^2 , or $0.6\text{ cm}^2/\text{m}^2$ when indexed. This condition is referred to as low-

flow, low-gradient aortic stenosis. In patients with reduced LVEF, dobutamine stress echocardiography can be used to distinguish true severe aortic stenosis from pseudo-severe stenosis by assessing changes in stroke volume, transvalvular gradient, and calculated valve area during pharmacological augmentation of flow. If stroke volume increases while the aortic valve area remains severely reduced, true severe aortic stenosis is likely. If flow reserve is absent or the findings remain inconclusive, computed tomography (CT) assessment of aortic valve calcification can provide additional diagnostic information. CT quantifies valve calcification using an aortic valve calcium score, with sex-specific thresholds helping to confirm the diagnosis of severe aortic stenosis. If none of these findings supports severe valve obstruction, the diagnosis of severe aortic stenosis becomes unlikely (Figure 15).

Current guidelines also define indications for intervention in asymptomatic patients with severe aortic stenosis. Once a patient meets the criteria for aortic valve intervention, several treatment options are available. The 2025 ESC/EACTS guidelines base the choice between surgical and transcatheter treatment primarily on age, surgical risk, anatomy, life expectancy, and the feasibility of transfemoral access. Transcatheter aortic valve implantation (TAVI, or TAVR) is recommended as the preferred approach in patients aged 70 years or older when transfemoral access is feasible and valve anatomy is suitable, whereas surgical aortic valve replacement (SAVR) is recommended in patients younger than 70 years who are at low surgical risk. In patients who do not fall clearly into either category, the treatment strategy should be determined by the Heart Team after consideration of individual clinical, anatomical, and procedural factors, as well as estimated life expectancy and the expected durability of the prosthesis. These interventions should be performed in dedicated Heart Valve Centres with on-site cardiac surgery and interventional cardiology. If TAVI is otherwise indicated but transfemoral access is not feasible, an alternative access route may be considered; however, contemporary practice increasingly favors transvascular alternatives rather than transapical or direct transaortic access whenever possible. TAVI may also be considered in selected patients with bicuspid aortic valve stenosis who are at increased surgical risk and have suitable anatomy. TAVI is associated with favorable periprocedural outcomes, and randomized trials have demonstrated good results in intermediate and low-surgical-risk populations, contributing to the progressive expansion of its indications. During TAVI, a transcatheter bioprosthetic valve mounted within a stent frame is positioned within the native stenotic aortic valve. Expansion of the prosthesis displaces the native calcified cusps outward and establishes a new functional valve orifice.

In patients with severe aortic stenosis who are selected for surgical treatment, aortic valve replacement can be performed through a median sternotomy, partial sternotomy, or right

thoracotomy. The latter two approaches belong to the group of minimally invasive cardiac surgical techniques. After cardiopulmonary bypass has been established and cardioplegic arrest achieved, the aortic valve is exposed through an aortotomy. The native stenotic valve is excised and replaced with a prosthetic valve, which may be either mechanical or biological. Mechanical prostheses offer excellent durability but require lifelong anticoagulation with a vitamin K antagonist. They are therefore generally more suitable for younger patients with a long expected lifespan and no contraindication to long-term anticoagulation. Biological prostheses do not require lifelong vitamin K antagonist therapy but have limited durability because of structural valve degeneration and are therefore usually preferred in older patients. In intermediate age groups, prosthesis selection should be individualized according to expected lifespan, bleeding risk, the likelihood of future reintervention, and the patient's informed preference. Certain patient groups require additional consideration. In patients receiving chronic dialysis, structural degeneration of biological prostheses may occur more rapidly, whereas in women planning pregnancy the need for vitamin K antagonist therapy with a mechanical prosthesis represents an important limitation. In such patients, a biological prosthesis may therefore be preferred despite the higher likelihood of future reintervention. The availability of transcatheter valve-in-valve implantation for degenerated surgical bioprostheses has further influenced prosthesis selection, particularly in younger patients who wish to avoid lifelong anticoagulation, although this strategy requires careful consideration of the size of the original prosthesis, coronary anatomy, and the feasibility of future transcatheter interventions.

Some centers also offer alternative surgical strategies for the treatment of aortic valve disease. The Ozaki procedure consists of reconstructing the aortic valve using new cusps fashioned from the patient's own pericardium. This technique can provide excellent postoperative hemodynamics, including in patients with a small aortic annulus, although long-term durability data remain more limited than for established prosthetic valves. Another option is the Ross procedure, in which the diseased aortic valve is replaced with the patient's pulmonary autograft and the right ventricular outflow tract is reconstructed using a pulmonary homograft. Because the homograft does not require immunosuppressive therapy and is exposed to the lower-pressure pulmonary circulation, its durability is generally better than that of a homograft implanted in the systemic position. The pulmonary autograft in the aortic position provides a living, autologous valve substitute with excellent hemodynamic properties and the potential for long-term durability. The Ross procedure is technically more demanding and converts single-valve disease into a two-valve operation, but in carefully selected young patients it offers very favorable long-term outcomes. It may be particularly

attractive in young adults and in women planning pregnancy, in whom avoidance of lifelong anticoagulation is an important consideration.

Aortic valve surgery is associated with low operative mortality in appropriately selected patients and is performed routinely in cardiac surgical centers worldwide. In patients with symptomatic severe aortic stenosis who are selected for surgical treatment, valve replacement relieves the fixed obstruction to left ventricular outflow and thereby reduces the pathological pressure overload responsible for progressive ventricular hypertrophy and heart failure. The extent of this hemodynamic improvement depends in part on implantation of a prosthesis with an adequate effective orifice area. If the prosthesis is too small relative to the patient's body size and flow requirements, prosthesis-patient mismatch may occur, resulting in persistently elevated transprosthetic gradients and flow velocities after surgery. When the native aortic annulus is too small to accommodate an appropriately sized prosthesis, several surgical techniques can be used to enlarge the annulus or aortic root and thereby permit implantation of a prosthesis with a larger effective orifice area. In patients with favorable postoperative hemodynamics, regression of left ventricular hypertrophy and improvement in diastolic function are commonly observed during follow-up. Delaying intervention after the development of a clear indication may allow irreversible myocardial fibrosis, ventricular dysfunction, and heart failure to progress, thereby limiting the extent of functional recovery after valve replacement. Patients should therefore undergo intervention once guideline-based criteria are fulfilled, provided that the expected benefit outweighs the procedural risk.

6.2 Aortic Regurgitation

Aortic regurgitation is considerably less common in the general population than aortic stenosis. Pathophysiologically, it may result from primary disease of the valve cusps, including degenerative or myxomatous changes, infective endocarditis, rheumatic disease, and selected congenital abnormalities. A second mechanism involves pathology of the aortic root, in which progressive dilatation displaces the commissures and impairs coaptation of the aortic cusps, resulting in regurgitation.

With chronic progressive aortic regurgitation, volume overload leads to eccentric left ventricular hypertrophy and progressive ventricular dilatation. This compensatory remodeling initially allows the left ventricle to maintain an adequate forward stroke volume despite the regurgitant volume returning during diastole. Once these compensatory mechanisms become insufficient, left ventricular systolic function declines, with a reduction in ejection fraction and an increase in left ventricular end-systolic dimension (LVESD).

Clinical signs may include a widened pulse pressure and a bounding pulse (pulsus celer et altus, or Corrigan pulse), de Musset sign with head bobbing synchronous with the pulse, prominent carotid pulsations, pulsatile sensations in the head, palpitations, fatigue, Müller sign with pulsation of the uvula, and Quincke sign with visible capillary pulsation in the nail bed.

Diagnostic assessment is based primarily on echocardiography. Findings supporting severe aortic regurgitation include holodiastolic flow reversal in the descending aorta with an end-diastolic velocity greater than 20 cm/s, a vena contracta width greater than 6 mm, a pressure half-time below 200 ms, an effective regurgitant orifice area of at least 30 mm², and a regurgitant volume of at least 60 mL.

Surgery is indicated in symptomatic patients with severe aortic regurgitation and in asymptomatic patients with severe regurgitation who show evidence of left ventricular decompensation, such as a reduction in left ventricular ejection fraction or progressive increase in LVESD. When aortic valve surgery is indicated and significant dilatation of the ascending aorta is also present, concomitant surgical treatment of the ascending aorta should be considered according to the diameter, underlying aortic pathology, and individual patient characteristics (Figure 16).

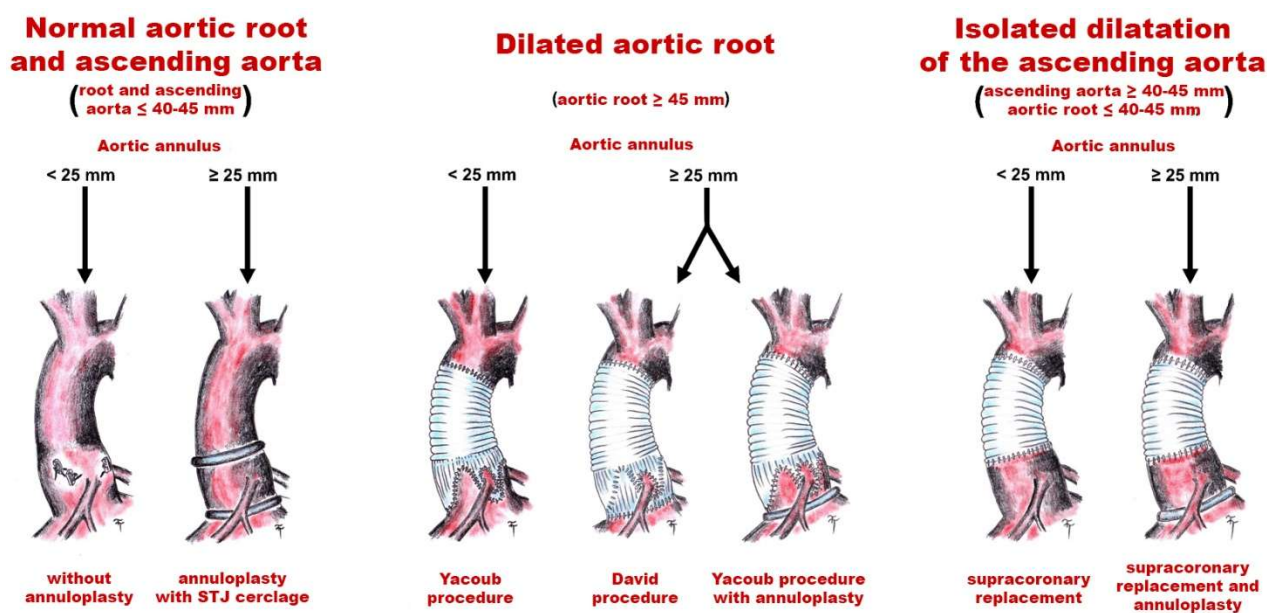


Figure 16 Aortic procedures in aortic-valve-sparing surgery for aortic regurgitation (SCR - supracoronary replacement - the Wheat procedure).

The standard surgical treatment for aortic regurgitation is aortic valve replacement with either a mechanical or biological prosthesis, with prosthesis selection following the same general principles as in surgery for aortic stenosis. When pathology also involves the aortic root, valve-sparing surgery may be considered, particularly when the valve cusps themselves remain structurally preserved. If the aortic root is not dilated and the mechanism of regurgitation is primarily valvular, simple valve replacement is generally preferred, and the indication for a valve-sparing procedure is less well

established. In patients with a dilated aortic root and preserved cusp tissue, several valve-sparing techniques are available, including aortic valve reimplantation (the David procedure), aortic root remodeling (the Yacoub procedure), and the Florida Sleeve procedure (Figure 17). In the David procedure, the aortic valve is reimplanted within a vascular graft that is anchored to the aortic annulus, restoring and stabilizing the geometry of the aortic root. The coronary ostia are then reimplanted into the graft. In the Yacoub procedure, the diseased aortic root is replaced while preserving the native aortic valve, with the graft tailored to recreate the sinuses of Valsalva; the annulus may additionally be stabilized with an external annuloplasty ring. The coronary arteries are again reimplanted into the graft. In the Florida Sleeve procedure, the native aortic root and coronary origins are preserved, and the root is externally supported within a vascular graft that is fenestrated to accommodate the coronary arteries. When appropriately indicated and performed in experienced centers, valve-sparing root replacement can provide excellent long-term valve durability while avoiding lifelong anticoagulation. Careful patient selection is essential, particularly with regard to cusp quality, cusp geometry, and the mechanism of regurgitation. When valve anatomy is unfavorable or surgical experience with valve-sparing techniques is limited, conventional valve replacement remains the more predictable option.

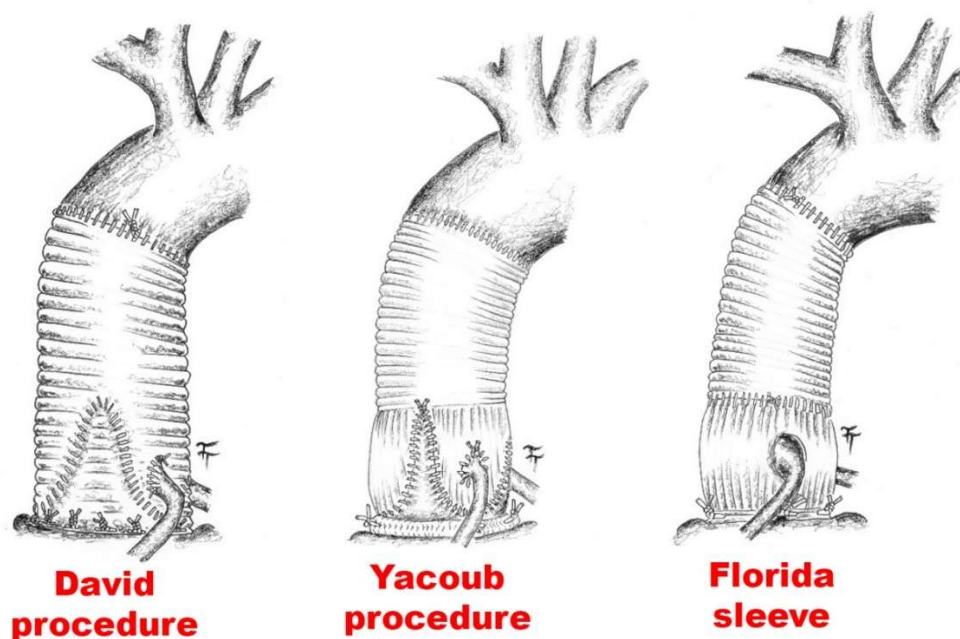


Figure 17 Aortic-valve-sparing procedures.v

When the heart is affected by infective endocarditis, the diagnosis is established according to the Duke criteria. The major criteria include positive blood cultures fulfilling specific microbiological conditions and imaging evidence of endocardial involvement. Minor criteria include the presence of a predisposing cardiac condition or intravenous drug use, fever, vascular phenomena, immunological phenomena, and microbiological evidence that does not meet the requirements for

a major criterion. A definite diagnosis of infective endocarditis is established by the presence of two major criteria, one major criterion together with three minor criteria, or five minor criteria. Once the diagnosis is established, targeted antimicrobial therapy is initiated according to the identified or suspected pathogen. Surgical treatment is indicated when infective endocarditis causes heart failure, most commonly as a result of severe valvular regurgitation due to destruction of the valve cusps or leaflets, when the infection cannot be adequately controlled, or when there is a high risk of systemic embolization. The surgical strategy is determined by the extent and anatomical location of infection and by the cardiac structures involved.

6.3 Mitral Stenosis

Before the widespread availability of antibiotic therapy, mitral valve disease, particularly mitral stenosis, was among the most common forms of valvular heart disease and was predominantly caused by rheumatic involvement. Before the introduction of cardiopulmonary bypass, closed digital mitral commissurotomy was one of the earliest cardiac surgical procedures performed on the beating heart. Today, rheumatic valvular disease is considerably less common in developed countries. The most frequent contemporary mitral valve disorder is mitral regurgitation, particularly secondary mitral regurgitation associated with ischemic or dilated cardiomyopathy. Functionally, the mitral valve represents a complex apparatus in which several anatomical structures and physiological mechanisms act together. Normal mitral valve function depends on the integrity and coordinated function of the mitral annulus, valve leaflets, chordae tendineae, papillary muscles, and left ventricular myocardium.

Mitral stenosis is defined by narrowing of the mitral valve orifice, with clinically significant stenosis generally present when the valve area is reduced below approximately 1.5 cm². Its causes include rheumatic valvular disease, degenerative calcification of the leaflets and annulus, congenital abnormalities, intracardiac masses or thrombi, and, less commonly, obstructive vegetations in infective endocarditis. Rheumatic mitral stenosis develops as a consequence of an immune-mediated reaction following infection with group A β -hemolytic streptococci, with cross-reactivity against cardiac tissues. The mitral valve is most commonly affected, often in combination with the aortic valve. Mitral stenosis may remain clinically well tolerated for many years. Maintenance of adequate cardiac output, however, becomes increasingly dependent on preserved sinus rhythm because atrial contraction contributes substantially to left ventricular filling across the narrowed mitral orifice. Progressive pressure overload of the left atrium leads to atrial dilatation and structural remodeling, creating a substrate for atrial fibrillation, which may precipitate hemodynamic decompensation. The resulting elevation in left atrial pressure is transmitted backward to the pulmonary circulation and can be reflected by an increase in pulmonary artery wedge pressure

(PAWP) during right heart catheterization. Persistent pulmonary venous hypertension may subsequently lead to pulmonary vascular remodeling, increased pulmonary vascular resistance (PVR), right ventricular pressure overload, and secondary tricuspid regurgitation. If pulmonary vascular remodeling becomes advanced, elevated PVR and right-sided heart failure may persist even after successful treatment of the mitral stenosis. Timely treatment is therefore important to prevent irreversible pulmonary vascular and right ventricular changes and to optimize long-term outcome.

Clinically, mitral stenosis may remain well tolerated for many years, and patients can be asymptomatic for a prolonged period. When symptoms develop, they most commonly include exertional dyspnea caused by elevated left atrial and pulmonary venous pressures, hemoptysis resulting from rupture of dilated bronchial or pulmonary venous vessels, and systemic thromboembolic events. Thromboembolism may present as ischemic stroke, mesenteric ischemia, acute limb ischemia, or embolization to other systemic vascular territories. Treatment is indicated in symptomatic patients when the mitral valve area is reduced below approximately 1.5 cm² and may also be considered in selected asymptomatic patients with evidence of hemodynamic consequences or a high risk of clinical deterioration. Percutaneous mitral commissurotomy (PMC) is the preferred treatment when valve morphology is suitable. Contraindications include left atrial thrombus, more than mild mitral regurgitation, severe or extensive commissural calcification, absence of commissural fusion, and the presence of another cardiac condition requiring surgery, such as significant concomitant valvular disease or an indication for coronary artery bypass grafting. When PMC is contraindicated or anatomically unsuitable, surgical treatment is indicated. In advanced rheumatic mitral stenosis, durable valve repair is often not feasible, and mitral valve replacement is therefore frequently required.

6.4 Mitral Regurgitation

Mitral regurgitation is defined as the backward flow of blood from the left ventricle into the left atrium during systole. Its causes include ischemic heart disease with secondary functional mitral regurgitation, myxomatous degeneration, rheumatic disease, calcific degeneration of the mitral apparatus, infective endocarditis, congenital abnormalities, endomyocardial fibrosis, myocarditis, and connective tissue disorders. Secondary mitral regurgitation associated with ischemic heart disease results primarily from left ventricular remodeling, with displacement of the papillary muscles, leaflet tethering, and annular dilatation, particularly in the septolateral dimension. Primary mitral regurgitation most commonly results from degenerative disease, including myxomatous degeneration, as Barlow disease, or fibroelastic deficiency. Degenerative mitral valve disease may also occur in patients with hereditary connective tissue disorders such as Marfan syndrome, Ehlers-

Danlos syndrome, and Loeys-Dietz syndrome. These conditions may also be associated with aortic regurgitation and aortic aneurysmal disease.

Mitral regurgitation most commonly presents with reduced exercise tolerance and progressive dyspnea. Patients may remain clinically compensated for a prolonged period, particularly while sinus rhythm and left ventricular function are preserved. Progressive left atrial dilatation and remodeling may lead to the development of atrial fibrillation, which can further worsen symptoms and hemodynamics. The indication for surgical treatment is based primarily on the severity and mechanism of mitral regurgitation, the presence of symptoms, and evidence of cardiac remodeling or dysfunction. Echocardiographic findings supporting severe mitral regurgitation include morphological evidence of flail leaflet, a large holosystolic regurgitant jet, a vena contracta width greater than 7 mm, systolic flow reversal in the pulmonary veins, an E-wave velocity greater than 1.2 m/s, a mitral-to-aortic time-velocity integral ratio greater than 1.4, an effective regurgitant orifice area greater than 40 mm², a regurgitant volume of at least 60 mL, and a regurgitant fraction of at least 50%. Additional findings relevant to the timing of intervention include left ventricular dilatation, an increased left ventricular end-systolic dimension, left atrial enlargement, reduced left ventricular systolic function, pulmonary hypertension, and new-onset atrial fibrillation.

To assess the mechanism of mitral regurgitation from a surgical perspective and to guide operative strategy, the Carpentier classification is most commonly used. More broadly, the Carpentier triad evaluates three components: the etiology of regurgitation, the valve lesion, meaning the structural abnormality responsible for valve dysfunction, and leaflet dysfunction, meaning the resulting abnormality in leaflet motion. This distinction is clinically important because long-term outcomes after mitral valve repair depend largely on the underlying etiology, the overall surgical strategy is guided by the type of leaflet dysfunction, and the specific operative technique is determined by the structural valve lesion. The Carpentier classification itself is based primarily on the pattern of leaflet motion during systole and diastole.

Type I mitral regurgitation in the Carpentier classification is characterized by normal leaflet motion, with regurgitation resulting from annular dilatation or leaflet perforation. Type II is characterized by excessive leaflet motion, with one or both leaflets prolapsing beyond the plane of the mitral annulus into the left atrium during systole. The underlying anatomical lesion is most commonly elongation or rupture of the chordae tendineae or rupture of a papillary muscle head. Acute mitral regurgitation caused by papillary muscle rupture after myocardial infarction therefore also belongs to this category. Type IIIa is characterized by restricted leaflet motion during both systole and diastole and is most commonly caused by thickening, shortening, and fusion of the leaflets and chordae. This mechanism is frequently associated with mitral stenosis. Type IIIb is

characterized by restricted leaflet motion predominantly during systole and usually results from left ventricular remodeling with displacement of the papillary muscles and leaflet tethering (Figure 18).

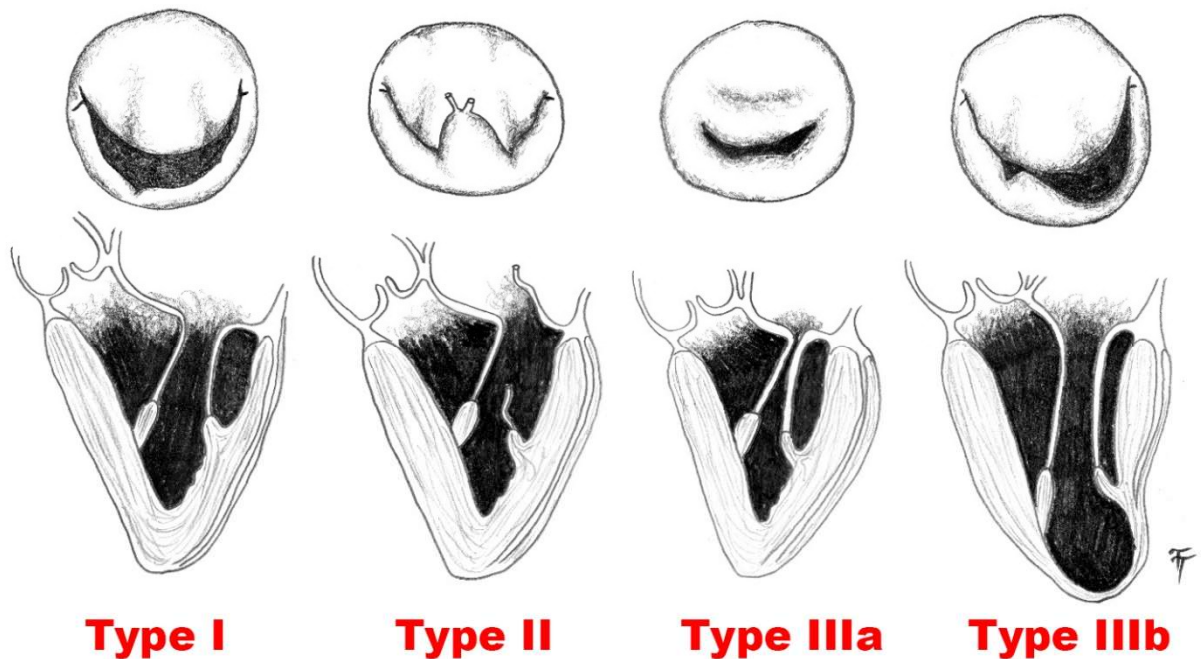


Figure 18 Illustrative depiction of the Carpentier classification.

The earliest surgical attempts at mitral valve repair consisted mainly of plication of the annulus near the commissures, thereby reducing the size of the dilated annulus without precise restoration of its normal geometry. Today, this technique has only limited application and may still be used in selected procedures for atrioventricular septal defects. When used for isolated mitral regurgitation, these early techniques were frequently associated with residual regurgitation, because additional mechanisms responsible for valve incompetence remained untreated, or with the development of mitral stenosis as a consequence of excessive narrowing and subsequent fibrosis at the site of the plication sutures.

With the introduction of mechanical valve prostheses, these relatively unpredictable early repair techniques temporarily lost importance. The concept of mitral annular remodeling in a form resembling contemporary annuloplasty gained broader acceptance in the late 1960s. It is important to distinguish simple reduction of annular size from true annular remodeling. Annuloplasty aims to restore both the size and geometry of the mitral annulus toward its physiological configuration while preserving appropriate leaflet motion. The size of the annuloplasty ring should correspond to the dimensions of the valve leaflets and the intended area of leaflet coaptation, thereby reducing excessive mechanical stress on the repaired valve. An equally important objective is to stabilize the annulus and prevent subsequent redilatation.

In Type I mitral regurgitation, the primary surgical objective is to restore the size and geometry of the mitral annulus toward its physiological dimensions and thereby re-establish an appropriate relationship between annular area and leaflet surface area. Accurate sizing of the annuloplasty ring is therefore essential. This is usually based primarily on the dimensions of the anterior mitral leaflet. Measurement of the intercommissural distance is facilitated by placing commissural sutures, which help define the anatomical limits of the anterior leaflet during sizing. The height of the anterior leaflet should also be taken into account and should correspond appropriately to the anteroposterior dimension of the selected annuloplasty ring. The height of the posterior leaflet is another important parameter, particularly when it is increased, because excessive posterior leaflet height combined with an undersized ring may predispose to postoperative systolic anterior motion (SAM) of the mitral valve. SAM is an important postoperative complication that may result in both recurrent mitral regurgitation and dynamic obstruction of the left ventricular outflow tract.

Anterior leaflet prolapse in Type II mitral regurgitation has historically led to the development of a wide range of reconstructive techniques. No single method is suitable for all patients, and the optimal strategy depends on the specific anatomical lesion, including the width and location of the prolapsing segment, the degree of prolapse, and the underlying mechanism, most commonly chordal elongation or rupture. Careful preoperative and intraoperative assessment of the Carpentier triad therefore remains essential. Based on this assessment, the repair technique is selected. Commonly used methods include limited triangular or resection for small localized areas of prolapse, papillary muscle repositioning or relocation in selected cases of altered subvalvular geometry, chordal transposition using suitable secondary chordae, and implantation of expanded polytetrafluoroethylene (ePTFE; Gore-Tex) neochordae, particularly when native chordae are ruptured or otherwise unsuitable for preservation.

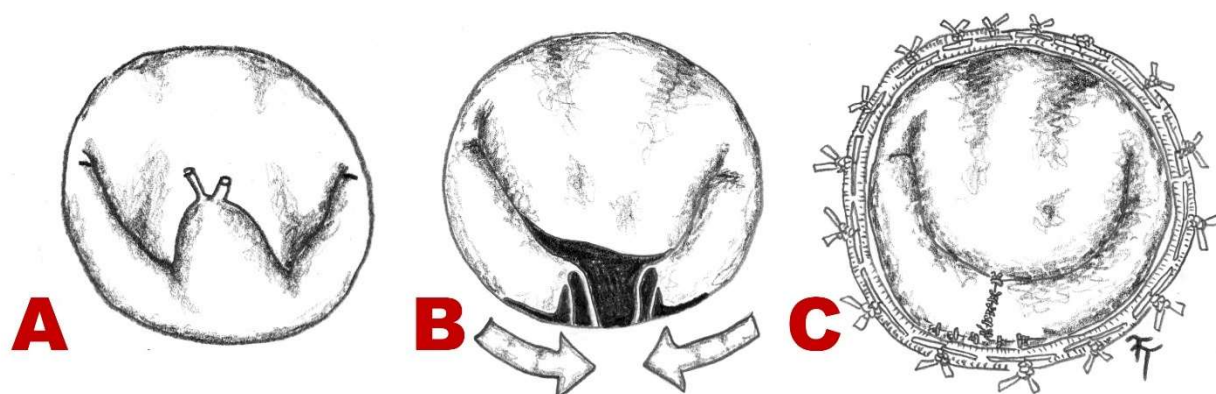


Figure 19 Repair of posterior mitral leaflet prolapse in the P2 segment by quadrangular resection, sliding plasty, and annuloplasty ring implantation (A - prolapsing P2 segment with ruptured primary chordae, B - state after resection of the prolapsing portion of P2, with sufficient chordae visible at the edges of the remaining posterior leaflet, C - state after suturing and annuloplasty ring implantation).

Posterior leaflet prolapse in Type II mitral regurgitation is one of the most common mechanisms of primary mitral regurgitation. A widely used surgical technique is triangular or quadrangular resection of the prolapsing leaflet segment. When a broader portion of the posterior leaflet is involved, quadrangular resection may be combined with a sliding leaflet technique to reduce excess tissue and restore appropriate leaflet geometry (Figure 19). If prolapse involves multiple segments, implantation of ePTFE (Gore-Tex) neochordae is an alternative reconstructive strategy. Commissural prolapse in Type II mitral regurgitation may result from infective endocarditis with chordal destruction or from degenerative leaflet disease. A distinction can be made between limited commissural prolapse, involving tissue close to the commissure, and more extensive paracommissural prolapse. Surgical options include limited leaflet resection, paracommissural leaflet approximation, and neochordal implantation. In selected cases, neochordal reconstruction alone may provide a stable and durable repair.

The most common cause of Type IIIa mitral regurgitation is rheumatic valve disease, characterized by commissural and chordal fusion together with thickening, retraction, and sometimes calcification of the valve leaflets. Commissural fusion can be treated surgically by commissurotomy, while restoration of leaflet mobility may require division, fenestration, or resection of fused and thickened chordae. When mitral stenosis is the predominant lesion, annuloplasty is not always required, particularly in patients with preserved left ventricular function, limited ventricular dilatation, and relatively preserved annular geometry. Because Type IIIa disease usually involves several components of the mitral apparatus, the extent of structural involvement should be carefully considered when deciding between valve repair and replacement. Important determinants of repairability include the mobility and quality of the anterior leaflet and the degree of involvement of the subvalvular apparatus.

In Type IIIb mitral regurgitation, leaflet restriction occurs during systole despite structurally preserved chordae and leaflets. The underlying mechanism is usually left ventricular remodeling with apical and lateral displacement of the papillary muscles, most commonly as a consequence of ischemic cardiomyopathy or dilated cardiomyopathy. The basic reconstructive strategy is implantation of an undersized annuloplasty ring to improve leaflet coaptation. In selected patients, annuloplasty may be combined with additional subvalvular procedures, such as secondary chordal cutting, papillary muscle approximation, or papillary muscle relocation, in order to reduce leaflet tethering and improve the durability of repair.

Systolic anterior motion (SAM) of the mitral valve is characterized by displacement of the anterior leaflet, usually its distal portion, toward the left ventricular outflow tract (LVOT) during systole, resulting in dynamic LVOT obstruction and/or mitral regurgitation. Predisposing factors

for SAM after mitral valve repair include excess leaflet tissue, particularly involving the posterior leaflet, a small left ventricular cavity, a narrow mitral-aortic geometry, and an undersized annuloplasty ring. Increased anterior or posterior leaflet height may also increase the risk. When SAM occurs after repair, management depends on its severity and underlying mechanism. Initial treatment focuses on optimization of loading conditions and reduction of hypercontractility. If surgical revision is required because of persistent hemodynamically significant SAM, several techniques may be used, including reduction of excessive posterior leaflet height, modification of leaflet geometry, neochordal reconstruction, or edge-to-edge repair. The Alfieri technique (edge-to-edge repair) creates a double-orifice mitral valve by placing a suture between the central portions of the anterior and posterior leaflets, usually the A2 and P2 segments.

Contemporary mitral valve surgery generally favors repair over replacement whenever a durable reconstruction can be achieved. The likelihood of successful and lasting repair, however, depends on the underlying valve pathology, the complexity of the lesion, ventricular and annular geometry, patient characteristics, and the experience of the surgeon and treating center. Degenerative primary mitral regurgitation is particularly well suited to repair in experienced centers, whereas the durability of repair is less predictable in complex secondary or rheumatic disease.

In addition to surgical repair or replacement, transcatheter edge-to-edge repair (TEER) has become an established treatment option for selected patients with mitral regurgitation. The technique approximates the free edges of the anterior and posterior mitral leaflets and is conceptually analogous to the surgical Alfieri stitch. In primary mitral regurgitation, TEER is mainly considered in symptomatic patients with severe regurgitation who are judged by the Heart Team to be at high or prohibitive surgical risk, while surgical repair remains the preferred treatment when a durable result can be expected. In secondary mitral regurgitation, TEER is recommended in appropriately selected symptomatic patients who remain severely affected despite optimized guideline-directed medical therapy, including cardiac resynchronization therapy when indicated, provided that ventricular function, dimensions, pulmonary pressures, and valve anatomy are compatible with a favorable outcome. In patients with extensive mitral annular calcification, failed previous repair, or anatomy unsuitable for conventional surgery or TEER, transcatheter mitral valve implantation may be considered in highly selected cases at experienced Heart Valve Centres. The choice between surgical intervention, TEER, transcatheter valve implantation, and continued medical therapy should be individualized by the Heart Team according to the mechanism and severity of regurgitation, ventricular function, comorbidities, procedural risk, and anatomical suitability.

Preoperative assessment of the patient and detailed echocardiographic characterization of mitral valve pathology can help stratify the wide spectrum of mitral valve disease and guide the surgeon toward the most appropriate repair strategy. In general, anterior leaflet involvement is associated with a more technically demanding repair and, in some series, less favorable long-term durability than isolated posterior leaflet disease. Similarly, multisegment posterior leaflet involvement is more complex to reconstruct than a single-segment lesion. Several scoring systems have been proposed to grade the complexity of myxomatous mitral valve disease. Table 6 presents one such system, in which a score of 1 corresponds to a relatively simple repair, a score of 2–4 to a moderately complex procedure, and a score of 5 to a technically demanding reconstruction. The resulting score therefore provides an estimate of repair complexity and of the likelihood that repair may be difficult or unsuccessful, and may help guide both the operative strategy and referral to a surgeon or center with appropriate expertise. These scoring systems, however, have not been universally adopted by professional cardiology or cardiac surgical societies as formal indications or contraindications to mitral valve repair. They should therefore be regarded as adjunctive tools that support, rather than replace, multidisciplinary assessment and discussion between the cardiologist and cardiac surgeon.

Table 6 Risk stratification for mitral valve repair in myxomatous disease.

Anatomical factor	points	Anatomical factor	points
Segmental P1 prolapse	1	Anterolateral commissural prolapse	2
Segmental P2 prolapse	1	Posteromedial commissural prolapse	2
Segmental P3 prolapse	1	Leaflet restriction	2
Segmental A1 prolapse	1	Calcification of a leaflet or papillary muscle without annular involvement	2
Segmental A2 prolapse	1	Annular calcification	3
Segmental A3 prolapse	1	Prior mitral valve repair	3
Complexity of surgical intervention			score
Simple mitral valve repair			1
Moderately demanding surgical intervention			2 - 4
Complex surgical correction of the lesion			≥ 5

Mitral valve replacement remains an established surgical option when durable valve repair is not feasible. From a surgical perspective, preservation of the subvalvular apparatus is one of the most

important principles for maintaining left ventricular geometry and left ventricular function after mitral valve replacement. The extent of subvalvular preservation varies among surgical techniques and published series; in many cases, preservation of the posterior leaflet and its chordal attachments is considered sufficient to maintain continuity between the mitral annulus and the ventricular myocardium. Anticoagulation management after mitral valve replacement depends on the type of prosthesis and may also vary according to national or institutional protocols. Adherence to an appropriate anticoagulation regimen is essential, as inadequate or excessive anticoagulation can substantially influence the incidence of thromboembolic and bleeding complications. Several studies have suggested that when the mitral subvalvular apparatus is extensively preserved during valve replacement, postoperative left ventricular remodeling may approach that observed after successful valve repair.

With the progressive aging of the population, an increasing proportion of elderly patients require surgical or transcatheter treatment for mitral regurgitation. Although referral of these patients for surgery may be influenced by concerns related to age, frailty, and operative risk, numerous studies have demonstrated acceptable short- and long-term outcomes in appropriately selected elderly patients. Mitral valve repair is increasingly performed in this age group whenever a durable reconstruction can be achieved. A meta-analysis published by Tomasso et al. in 2021 reported favorable short- and long-term outcomes after mitral valve surgery in patients older than 75 years and suggested a particular benefit of valve repair compared with replacement, including improved early survival and favorable long-term outcomes. Other contemporary studies have similarly emphasized the importance of timely referral and intervention, as postponing surgery until advanced symptoms, ventricular dysfunction, or frailty develop is associated with less favorable postoperative outcomes. Despite this, a substantial proportion of elderly patients with severe mitral regurgitation are never referred for multidisciplinary Heart Team assessment because of perceived procedural risk or advanced age alone. Increasing institutional experience with mitral valve repair can shorten operative, cardiopulmonary bypass, and aortic cross-clamp times and may result in procedural durations comparable to those of valve replacement. Consequently, concerns regarding prolonged cardiopulmonary bypass or postoperative ventilatory support should not by themselves be considered reasons to favor mitral valve replacement over repair in elderly patients when a durable repair is technically achievable.

Most meta-analyses favor mitral valve repair over replacement across a range of clinically important outcomes, particularly short- and long-term survival, thromboembolic risk, heart failure, and, in many series, the need for reoperation. Other systematic analyses have demonstrated lower mortality after mitral valve repair without a significant difference in reoperation rates. When

preservation of the subvalvular apparatus is consistently achieved during mitral valve replacement, some comparative studies have found no significant differences between repair and replacement in operative mortality, long-term survival, changes in left ventricular ejection fraction (LVEF), or the incidence of valve-related complications. One of the most important predictors of reoperation after mitral valve repair is residual mitral regurgitation present immediately after the primary procedure. The durability of repair is also influenced by the specific annuloplasty technique, the type and size of the annuloplasty ring, and the reconstructive procedures performed on the chordae, leaflets, papillary muscles, and other components of the mitral apparatus. Progressive degenerative changes may contribute to recurrent mitral regurgitation during follow-up. Several factors have been associated with an increased risk of repair failure or reoperation, including younger patient age, larger indexed annuloplasty ring size, omission of an annuloplasty ring, and residual mitral regurgitation of at least grade 1 at the end of the procedure. Accurate preoperative patient stratification and detailed echocardiographic assessment of the severity, extent, and mechanism of mitral regurgitation, together with realistic evaluation of the most appropriate treatment strategy, are therefore essential for achieving durable repair and optimal long-term outcomes.

6.5 Acquired Tricuspid and Pulmonary Valve Disease

The tricuspid valve is affected by regurgitation far more commonly than by stenosis. This is partly related to the complex and highly dynamic anatomy of the tricuspid annulus, whose area changes substantially during the cardiac cycle. These characteristics make the annulus particularly susceptible to dilatation under conditions of chronic volume or pressure overload. Tricuspid regurgitation is most often secondary, resulting from disease of other cardiac structures or from pulmonary hypertension and right ventricular remodeling. Common underlying conditions include mitral valve disease, aortic stenosis, and ischemic heart disease. Increased pulmonary vascular pressure, right ventricular pressure or volume overload, and changes in ventricular geometry are among the principal mechanisms leading to functional tricuspid regurgitation. Echocardiographic findings supporting severe tricuspid regurgitation include a vena contracta width greater than 7 mm, a proximal isovelocity surface area (PISA) radius greater than 9 mm, systolic flow reversal in the hepatic veins, a tricuspid E-wave velocity of at least 1 m/s, an effective regurgitant orifice area of at least 40 mm², and a regurgitant volume of at least 45 mL.

Tricuspid stenosis is diagnosed when the mean transvalvular pressure gradient across the tricuspid valve is at least 5 mmHg. Isolated tricuspid valve surgery is relatively uncommon, mainly because tricuspid valve disease is frequently secondary to other cardiac pathology. The indication depends not only on the severity of the valve lesion but also on right ventricular function and the degree of pulmonary hypertension. When right ventricular function is already severely impaired,

correction of severe tricuspid regurgitation may acutely increase right ventricular afterload and can therefore be associated with significant postoperative hemodynamic compromise.

More commonly, tricuspid valve surgery is performed concomitantly with another cardiac procedure. In this setting, the threshold for intervention is lower and is not restricted to patients with severe tricuspid regurgitation. Dreyfus and colleagues emphasized that tricuspid annular dilatation itself represents an important predictor of subsequent tricuspid regurgitation. Thus, even when significant regurgitation is not yet present at the time of surgery for left-sided heart disease, a markedly dilated tricuspid annulus may continue to enlarge and may lead to progressive regurgitation despite correction of the primary lesion. Clinically, this may later manifest as right-sided heart failure with hepatic congestion, ascites, and peripheral edema. Additional parameters that may help identify patients at increased risk of future tricuspid regurgitation continue to be investigated. At present, concomitant tricuspid valve surgery is generally considered in patients with severe tricuspid regurgitation or significant annular dilatation, commonly defined as a tricuspid annular diameter greater than 40 mm or greater than 21 mm/m².

The anatomy of the tricuspid valve means that annular dilatation occurs predominantly along the portions supporting the anterior and posterior leaflets. In contrast, the septal portion of the annulus lies in close proximity to the cardiac conduction system. For this reason, surgical reduction and stabilization of the tricuspid annulus are usually achieved by implantation of an incomplete annuloplasty ring that reinforces primarily the anterior and posterior annular segments while avoiding the region adjacent to the conduction tissue. In the past, other surgical strategies were also used. These included bicuspidalization of the tricuspid valve, in which the posterior leaflet and the corresponding annular segment were excluded by plication, thereby reducing annular size, and the De Vega annuloplasty, in which the annulus was reduced using running sutures placed along the anterior and posterior portions. Because these techniques are associated with a higher risk of recurrent annular dilatation and repair failure compared with ring annuloplasty, they are now used less frequently, although they may remain useful in selected intraoperative situations.

In recent years, transcatheter treatment of tricuspid regurgitation has become an important option for patients with severe symptomatic disease who are at high or prohibitive surgical risk. The most widely used technique is transcatheter edge-to-edge repair (TEER), adapted from mitral valve treatment. Current guidelines support its use in carefully selected symptomatic patients with persistent severe tricuspid regurgitation despite optimal medical therapy, provided that anatomy is suitable and severe right ventricular dysfunction or advanced pre-capillary pulmonary hypertension is absent. This approach is not intended to replace surgery in operable patients but offers a treatment

option for patients who would otherwise have limited alternatives because of excessive surgical risk.

Infective endocarditis involving the tricuspid valve is encountered particularly in patients with specific risk factors, including intravenous drug use, intracardiac devices, and indwelling venous catheters. Surgical treatment is indicated according to the general criteria for infective endocarditis and the extent of local valve destruction. Tricuspid stenosis is rare and may result from rheumatic disease, congenital abnormalities, carcinoid involvement, or obstruction by an intracardiac mass. The surgical strategy depends on the underlying pathology and the extent of valve involvement.

Because the pulmonary valve is exposed to substantially lower pressure than the left-sided valves, acquired pulmonary valve disease is relatively uncommon. Surgical or transcatheter intervention is indicated according to symptoms, right ventricular size and function, pressure gradients, and the severity of pulmonary regurgitation or stenosis. More commonly, pulmonary valve dysfunction is encountered in patients with previous congenital or reconstructive cardiac surgery, including those who have undergone the Ross procedure. A homograft or conduit in the pulmonary position may gradually develop stenosis, regurgitation, or both. In selected patients, transcatheter pulmonary valve implantation or balloon-based intervention may be feasible. If transcatheter treatment is not suitable, surgical replacement of the dysfunctional conduit or homograft with a new prosthesis or homograft may be required.

7 Selected Congenital Heart Defects in Childhood (Adrián Kolesár)

Congenital heart defects (*vitium cordis congenitum* - CHD) in childhood directly threaten the young patient's life, and early surgical correction is generally required. Congenital malformations of multiple structures are very often combined - the association and coincidence of congenital heart defects is typical, and isolated involvement of a single structure is rather the exception. Congenital malformations can be divided into non-cyanotic and cyanotic. In cyanotic defects, blood mixing occurs through a right-to-left shunt, causing hyposaturation of blood in the systemic circulation. Non-cyanotic congenital malformations, in contrast, involve a left-to-right shunt, or no mixing of blood between the right and left heart chambers occurs at all. The congenital malformations encountered by a cardiac surgeon specializing in pediatric cardiac surgery generally lead, without surgical correction, to early death or rapid progression of heart failure, with a high risk of death in early childhood. Their surgical correction is therefore absolutely necessary. Correction may be single-stage, in which all cardiac defects are corrected during a single operation. This type of intervention requires that all chambers of the heart be sufficiently developed for the heart to be able to tolerate the definitive circulation immediately. More often, therefore, a multi-stage approach is

used, in which the first operation attempts to correct the circulation to a state that the damaged heart is able to tolerate. The goal is to reach a state that is clinically manageable for the patient while placing an appropriate load on the less-developed structures. This correction leads to gradual adaptation of the less-developed cardiac structures toward the target form of circulation. In the second, or possibly third, stage, the circulation is then modified into the best possible form for the patient's future life. The definitive form of circulation need not necessarily replicate a fully physiological circulation, and in some cases the only option is an anatomical arrangement that, while not physiological, is the least symptomatic for the patient. Besides the more severe CHDs described in the following chapters, atrial septal defect and Ebstein's anomaly of the tricuspid valve can also be symptomatic in childhood, even to the point of requiring correction while the patient is still a child.

7.1 Patent Ductus Arteriosus

Patent ductus arteriosus, also known as the ductus of Botallo, is persistence of the open connection between the aorta and the pulmonary trunk. The ductus is necessary for the fetus's intrauterine circulation (it allows oxygenated blood from the placenta to bypass the lungs and delivers nutrients and oxygen directly into the systemic circulation) and should close spontaneously on the second or third day after birth. Its failure to close leads to overload of the pulmonary circulation on one hand, and to systemic hypoperfusion on the other (blood returns from the aorta back into the lungs).

The main risk factors for this congenital malformation include birth before 28 weeks of gestation, excessive fluid therapy, respiratory insufficiency, sepsis, phototherapy, and furosemide administration. In newborns, patent ductus arteriosus leads to bronchopulmonary dysplasia, necrotizing enterocolitis, renal insufficiency, diastolic heart failure, delayed neurological development, and retinopathy. It can be a direct cause of neonatal mortality. On the other hand, in the setting of severe pulmonary hypertension or other complex congenital heart defects, a persistent ductus arteriosus allows systemic circulation to be maintained via a shunt from the pulmonary trunk to the aorta. In its isolated form, however, the shunt runs in the opposite direction, overloading the pulmonary circulation.

Before the era of echocardiography, diagnosis of patent ductus arteriosus was based on the presence of cardiomegaly on chest X-ray, pulmonary edema, and a murmur. The first surgical treatment of this defect was performed in 1938. In 1972, Elliott and Starling first pointed out the role of prostaglandins in the pathophysiology of this condition (a patent ductus is an actively maintained state sustained by the action of prostaglandins, primarily prostaglandin E₂ (PGE₂)).

Since 1976, chloroquine, and since 1977 indomethacin, have therefore been used as prostaglandin antagonists to treat patent ductus arteriosus.

Whether a child is born at term or preterm, closure of the ductus arteriosus may be delayed or may fail entirely. This risk, however, rises significantly with preterm birth. Persistence of the ductus arteriosus beyond the third day after birth can be defined as pathological. Persistence of a patent ductus arteriosus immediately after birth, in the absence of other congenital malformations, causes flow reversal from the aorta into the pulmonary trunk. The pulmonary circulation is thus temporarily volume-overloaded, and the systemic circulation is hypovolemic, a phenomenon called "ductal steal." During intrauterine development, the ductus arteriosus remains open owing to prostaglandin receptors. At this stage of development, both the expression of receptors for these substances is higher, and their systemic circulating levels are also higher. Oxygen acts on the ductus arteriosus as a constrictor. Hemodynamically, the ductus arteriosus is also variable. A shorter length of the ductus arteriosus leads to higher flow through it. Flow is also increased by higher systemic vascular resistance, and, conversely, by a fall in pulmonary vascular resistance. Flow is further increased by prostaglandins and by lower blood viscosity.

Late diagnosis of a persistent ductus arteriosus has significant clinical consequences, so it is essential to correctly diagnose the hemodynamic significance of this shunt. The most common presentation includes cardiovascular, renal, respiratory, and gastrointestinal manifestations. Among the most characteristic cardiovascular symptoms are a widened pulse pressure, hypertension, and an audible murmur. Turbulent blood flow through the ductus arteriosus produces the murmur characteristic of this diagnosis. Volume overload of the pulmonary vasculature further volume-overloads the left heart. A fall in systemic perfusion is most pronounced during diastole, which significantly affects the myocardium, given its dependence on circulation during this phase of the cardiac cycle. Circulatory changes caused by hypoperfusion further manifest in the kidneys as fluid retention, metabolic acidosis, and oliguria. Laboratory findings show hyponatremia and elevated creatinine concentrations. Ultrasound examination may confirm zero, or even reversed, diastolic flow in the renal arteries. Hypoperfusion activates the renin-angiotensin-aldosterone pathway, with resulting reabsorption of sodium and water. Supranormal pulmonary circulation with volume overload leads to pulmonary edema, the need for assisted ventilation, pulmonary hemorrhage, and bronchopulmonary dysplasia. Volume overload reduces pulmonary vascular resistance, which further increases the volume of blood flowing through the ductus arteriosus. Surfactant deficiency in preterm infants further increases the risk of pulmonary edema. Systemic hypotension and diastolic flow reversal subsequently also cause gastrointestinal hypoperfusion.



Figure 20 Patent ductus arteriosus with coarctation of the aorta in the usual location.

Suspicion of patent ductus arteriosus can be raised when several symptoms coincide, including the clinical picture, laboratory parameters, and a specific radiographic finding (Figure 20). Echocardiography is, however, necessary to definitively confirm or rule out the diagnosis itself. The newborn presents with dyspnea, a widened pulse, hypertension, malnutrition, and feeding refusal, and, not least, a holosystolic murmur. Laboratory testing shows acidosis and elevated inflammatory markers, especially C-reactive protein. B-type natriuretic peptide and troponin T then become elevated. Chest X-ray documents cardiomegaly and pulmonary congestion. However, a similar constellation of signs can also be present in newborns with other congenital malformations. Echocardiography is the gold-standard diagnostic method, demonstrating both the anatomy and pathophysiology of a persistent ductus arteriosus. Parameters assessed include the diameter of the ductus arteriosus and its hemodynamics, as well as hemodynamic parameters of the left atrium and ventricle, and of the peripheral circulation, especially in the renal artery. A ductus arteriosus diameter greater than 1.5 mm, measured within the first 31 hours after birth in infants born before 29 weeks, is the best early predictor of a hemodynamically significant patent ductus arteriosus, with a sensitivity of 83% and a specificity of up to 90%. Other parameters assessed include flow through the ductus arteriosus, left atrial size, left atrial filling pressures, and the presence of flow reversal in the middle cerebral or superior mesenteric artery. Pulsatile flow through the ductus arteriosus is an important marker, confirming high flow through the shunt and equalization of aortic and pulmonary trunk pressures at end-diastole. High left atrial filling pressures likewise confirm volume overload

of both the pulmonary circulation and the left heart. Absence of end-diastolic flow, or the presence of reversed flow in the middle cerebral or superior mesenteric artery, already indicates a negative effect on the peripheral circulation.

Treatment of patent ductus arteriosus aims either to close it or to minimize its effects on the circulation. Treatment is divided into conservative, pharmacological, surgical, and endovascular approaches. Conservative treatment aims to reduce left ventricular preload and improve gas exchange in the overloaded pulmonary vasculature. Furosemide, on one hand, optimizes the circulation, but on the other hand has a negative effect on physiological closure of the ductus, and moreover its administration leads to elevated prostaglandin concentrations, with a further undesirable effect. An invasive or non-invasive respiratory regimen with a positive end-expiratory pressure of 5 cm H₂O is appropriate. Optimizing respiration also has a protective effect on the left ventricle of the heart. A further point of treatment is raising the hematocrit above 35-40%, in order to increase blood viscosity and reduce flow through the ductus arteriosus. Since the discovery of the role of prostaglandins in maintaining the ductus arteriosus, pharmacotherapy has been the key element in treating its pathological persistence. Prostaglandin inhibitors lead to natural constriction of the ductus arteriosus. Nonselective inhibitors such as indomethacin, ibuprofen, and acetaminophen are most commonly used. Managing other medications and their interactions is important, especially with gentamicin (nephrotoxicity) and glucocorticoids (risk of gastrointestinal perforation). Failure of pharmacotherapy is an indication for surgical or endovascular intervention. Surgical ligation is the oldest treatment for patent ductus arteriosus. The surgical procedure is performed via a left thoracotomy, with application of a vascular clip. The resulting change in hemodynamics leads to immediate changes in circulatory hemodynamics; nevertheless, it carries a wide range of complications, including bronchopulmonary dysplasia, chylothorax, and cardiopulmonary failure. The literature reports no absolute contraindications to intervention. Transvenous occlusion of the ductus arteriosus is now being introduced into practice. The first results of percutaneous intervention in newborns weighing less than 2 kilograms have already been published. These preliminarily confirm earlier pulmonary recovery after percutaneous intervention compared with open surgical repair. A possible complication of the percutaneous approach that has been reported is device migration into the pulmonary vasculature.

A final question remains: correct timing of treatment and any intervention in these patients. In general, treatment should begin within 12 hours of birth, or at the first signs of hemodynamic instability. Prophylactic indomethacin therapy should be indicated within 12 hours of birth, both for preterm birth and for newborns with very low birth weight. Studies show that early administration of indomethacin reduces the risk of intracerebral hemorrhage and the need for

surgical intervention. On the other hand, indomethacin does not reduce the incidence of chronic lung disease, nor does it improve neurological outcomes. Based on current knowledge, blanket prophylactic administration of indomethacin to all newborns is not indicated. Early screening for patent ductus arteriosus is, however, recommended, so that targeted treatment can begin before the first clinical manifestation of the hemodynamic consequences of the shunt. Early treatment leads both to a reduction in the incidence of complications and to a generally higher success rate of pharmacological treatment itself, and to a lower need for surgical intervention.

7.2 Coarctation of the Aorta

Coarctation of the aorta accounts for 5 to 8% of all congenital heart defects and affects about 0.4% of live-born children. It can occur in isolation, but is often associated with other congenital heart defects. In half of cases it is combined with malformations of the aortic valve, most often a bicuspid aortic valve. Other common comorbidities include aortic arch hypoplasia, subaortic stenosis, mitral valve abnormalities, patent ductus arteriosus, and ventricular or atrial septal defects. Coarctation is defined as a discrete narrowing of the aortic lumen, caused by thickening of the medial layer of the aortic wall. Its occurrence is also associated with arteriopathy of the aorta, particularly in areas close to the coarctation itself. It is most commonly located opposite the site where the ductus arteriosus enters the aorta, but can also be found above or below this level. Its etiopathogenesis has not been definitively clarified, and there are currently three theories of how this congenital malformation arises, pointing to three different time windows for its development. It may involve abnormal embryonic development, or reduced intrauterine flow through this region of the aorta with its subsequent regression; a third possibility is the theory of excess ductal tissue present in the wall of the descending aorta, which causes its narrowing shortly after birth. Coarctation of the aorta is also associated with several genetic mutations and syndromes, for example Turner syndrome, DiGeorge syndrome, and Noonan syndrome.

Prenatal diagnosis reduces early postnatal morbidity and mortality, and, by allowing preparation of the pregnant mother, reduces costly and risky transfers of newborns to cardiac centers. Prenatal ultrasound screening is the mainstay diagnostic method for these conditions. Prenatal cardiac assessment should include four ultrasound views of the heart: the three-vessel view, the four-chamber view, and examination of the left and right ventricular outflow tracts. It must be noted, however, that despite rapid progress in this diagnostic method, prenatal diagnosis of coarctation of the aorta remains difficult. Several scoring systems are therefore used to at least partially objectify this diagnosis, including the aortic arch z-score, the isthmus/ductus score, and the pulmonary artery/aorta ratio score.

Despite prenatal diagnosis, a number of patients are also diagnosed early postnatally. Once the ductus arteriosus closes, sudden aortic obstruction occurs, with hypoperfusion of the lower body, including the kidneys. Hypoperfusion of the abdominal organs leads to metabolic derangement. Moreover, overload of the left heart can progress to its failure. A ductus arteriosus that fails to close can partly compensate for blood flow to the lower half of the body, mitigating symptoms. The classic presentation of coarctation of the aorta includes delayed, weakened, or completely absent femoral pulses, a difference in blood pressure between the upper and lower limbs greater than 20 mmHg, and a systolic murmur. Comorbidities such as an aberrant subclavian artery or aortic arch hypoplasia can cause unequal pressures between the right and left upper limbs, and comparing pressures between a specific upper and lower limb can obscure the diagnosis of coarctation. An aberrant subclavian artery is present in about 3 to 4% of patients with coarctation of the aorta. Newborns with symptomatic coarctation are indicated for prostaglandin E1 administration, aimed at blocking natural closure of the ductus arteriosus, thereby reducing symptoms until surgery. In subclinical presentations, only patient monitoring is indicated. Surgical or interventional treatment should be indicated early, to prevent left ventricular hypertrophy from increased afterload and subsequent heart failure. With late diagnosis, hypertension develops, along with intracranial aneurysms with a risk of hemorrhage, left ventricular hypertrophy with congestive heart failure, and the development of poststenotic aortic dilation.

Although X-ray is not the first-choice diagnostic method, a visible shadow of prestenotic or poststenotic aortic dilation, and shadowing at the level of the fourth rib caused by dilation of the intercostal arteries acting as collateral circulation, can suggest the diagnosis of coarctation. Ultrasonography has much greater diagnostic value, allowing more precise imaging of the narrowed segment of the aorta and quantification of pressure gradients. Given its location, transesophageal examination is even more precise. Ultrasonography can also reveal any comorbidities and the state of the left ventricle. Computed tomography provides an advantageous morphological assessment of both the stenosis itself and the surrounding structures. Radiation exposure, however, considerably limits its use for repeated monitoring of pediatric patients. Further drawbacks include the nephrotoxic effect of the contrast agent and the inability to dynamically assess pressure conditions. Avoiding radiation exposure therefore favors the use of magnetic resonance imaging, especially when repeated examinations are needed for follow-up. The use of gadolinium further improves imaging of both the aorta and the collateral circulation. Analysis of contrast agent flow can further assess the pressure gradients of the stenosis and the collateral circulation. Newer MRI methods also allow quantification of aortic wall stress and estimation of collateral circulation capacity. However, the examination requires the patient to remain still, which is considerably limited in children.

Invasive catheter angiography remains the gold standard, allowing precise imaging of both the stenosis itself and the collateral circulation.

Leaving hemodynamically significant coarctation untreated significantly shortens life through the development of complications, including congestive heart failure, endocarditis, aortic rupture, or stroke. Survival itself is influenced mainly by the severity of the stenosis and the presence of other congenital comorbidities. Even in the absence of a pressure difference between the upper and lower body, due to a well-developed collateral circulation, hemodynamically significant coarctation is indicated for surgical correction. The goal of correction is to reduce the aortic gradient and reduce hypertension. Correct timing of the intervention is critically important. Earlier intervention carries a risk of restenosis requiring reintervention, while later surgery carries a risk of developing hypertension and aneurysm formation.

In newborns and small children, surgical intervention is the method of choice. The child's age and anatomical conditions determine the choice among several applicable surgical techniques. For mild stenosis, resection with an end-to-end anastomosis via a left thoracotomy is the preferred choice. In older children with a longer segment of stenosis, subclavian flap aortoplasty is a popular approach, though it carries a higher risk of restenosis (Figure 21). In adults, where further growth of the individual need not be considered, resection with interposition grafting is the first-choice method. Extra-anatomic bypass is the method of choice in adults with an extensive network of collateral circulation. In the perioperative period, the patient is at risk mainly of paradoxical hypertension, complications from injury to the recurrent laryngeal nerve, and subclavian steal phenomenon. Spinal cord injury with paraplegia is rare but has been reported.

Balloon angioplasty, as a further treatment option, can be performed in infants aged three to six months. Earlier intervention is not possible, because the ductus arteriosus is still active. In general, it is a palliative technique for high-risk patients, capable of delaying definitive treatment until the patient's condition is stabilized and improved. Dilation causes multiple ruptures of the intimal layer of the aorta with subsequent wall remodeling, which on one hand delays the risk of restenosis, but on the other hand increases the risk of dissection and wall rupture. The risk of restenosis after this procedure increases with decreasing age at the time of intervention. Stent implantation after dilation increases the long-term stability of this treatment, reducing the risk of both restenosis and aneurysm formation. The method is advantageous in adults. Its use in children brings the need for redilation as the aorta grows, as well as more frequent restenosis due to intimal hyperplasia. Growth-adaptable and biodegradable stents offer future promise, though long-term results are not yet available for these.

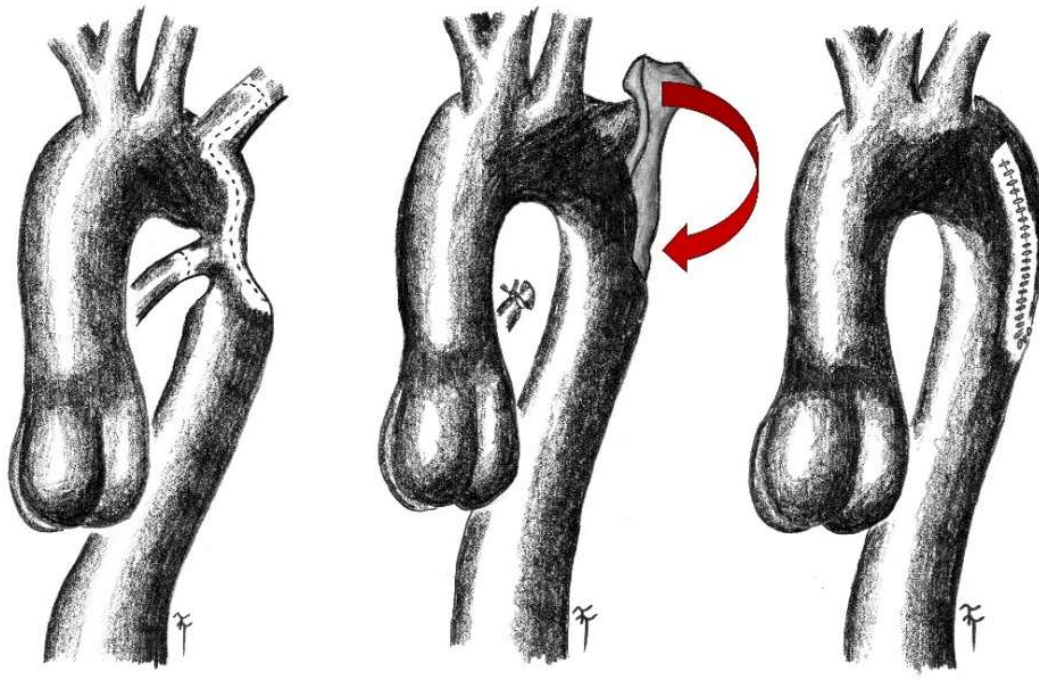


Figure 21 Aortoplasty for coarctation of the aorta in childhood.

Treatment outcomes remain controversial. A quarter of patients undergo cardiovascular intervention. Recoarctation occurs in 4-14% of patients. The incidence is higher in patients younger than one year. Reinterventions are needed more often after balloon angioplasty with or without stent implantation, compared with surgical intervention. Given the high coincidence of coarctation and bicuspid aortic valve, aortic regurgitation or stenosis is a frequent indication for surgery in the setting of hemodynamically borderline recoarctation. The aortic wall proximal to the coarctation also shows a reduced number of smooth muscle cells and an increased proportion of collagen, which affects local hemodynamics and increases the incidence of prestenotic dilation. The incidence of aneurysm formation is 13% in patients after coarctation repair, with a higher proportion among patients who underwent patch repair. Hypertension, as a further complication, has an incidence of 35-68%, with a higher proportion among patients who underwent coarctation repair at a later age. The exact etiology of hypertension after coarctation repair is unclear. Hypotheses point to abnormal baroreceptor function, a specific aortic arch geometry, persistent subclinical stenosis at the coarctation site, and dysfunction of the renin-angiotensin-aldosterone system. Left ventricular hypertrophy during the period the coarctation persists can lead to later myocardial fibrosis with resulting heart failure. Subendocardial fibrosis in particular leads to inadequate coronary blood flow with insufficient diastolic perfusion. The risk of ventricular failure increases with later intervention for coarctation.

Pseudocoarctation is a much rarer diagnosis than coarctation. It is characterized by a kind of kinking of the descending aorta at the site of the ductus arteriosus. Unlike true coarctation, neither a pressure gradient nor collateral circulation is present. Its embryological basis is a pathological fusion of the dorsal aorta and the fourth primitive aortic arch. The result is a pathological elongation of the descending aorta. This entity is generally asymptomatic, without the need for a corrective procedure. Surgery is indicated for the relatively rare cases with symptoms, or for aneurysm formation at this site. Turbulent blood flow in the elongated aorta is considered the cause of aneurysm formation in this diagnosis.

7.3 Aortic Arch Anomalies

A left-sided aortic arch with an aberrant right subclavian artery is the most common congenital malformation of the thoracic aorta, with an incidence of 0.5%-2%. This anomaly arises from regression of the right-sided arch between the right common carotid artery and the right subclavian artery. The right dorsal aorta then forms the definitive retroesophageal segment of the right subclavian artery. The aberrant right subclavian artery is thus the most distal great branch of the thoracic aorta, with a typical retroesophageal course running cranially to the right. The anomaly is typically isolated, but can less commonly be associated with coarctation of the aorta, patent ductus arteriosus, ventricular septal defect, or vertebral artery anomalies. In patients with Down syndrome, it is present in as many as one-third of patients. The malformation is typically asymptomatic, without the need for intervention; in only about 10% of cases can it cause esophageal compression with symptoms of dysphagia lusoria.

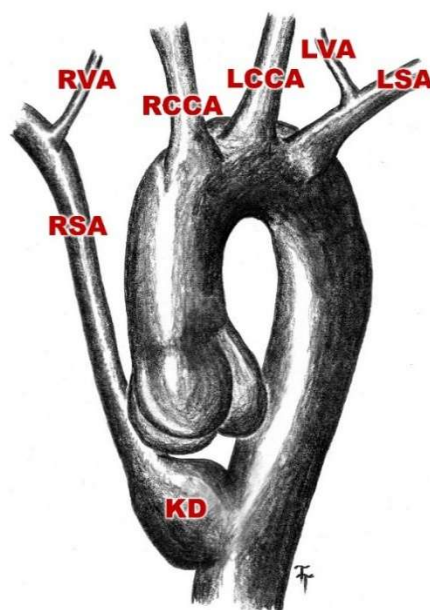


Figure 22 Kommerell's diverticulum with an aberrant right subclavian artery (LCCA - left common carotid artery, LSA - left subclavian artery, LVA - left vertebral artery, KD - Kommerell's diverticulum, RCCA - right common carotid artery, RSA - right subclavian artery, RVA - right vertebral artery)

Kommerell's diverticulum is an aneurysm of the descending aorta at the site where an aberrant subclavian artery arises (Figure 22). It represents a remnant of the right-sided dorsal primitive aorta. This rare diagnosis was first described by Kommerell in 1936, in a case report of a patient with a left-sided aortic arch and a diverticulum in the proximal descending aorta. Despite this specific initial description, the term Kommerell's diverticulum is used today for an aneurysm of the descending aorta from which an aberrant subclavian artery arises, whether with a left- or right-sided aortic arch. Given that an aberrant subclavian artery occurs in 0.8% to 2% of the population, and that Kommerell's diverticulum is present in 20% to 60% of these origins, an overall incidence of more than 0.5% of the population can be assumed.

It remains a fact that only 5% of patients with Kommerell's diverticulum report clinical symptoms, which are caused by compression of the surrounding anatomical structures. These symptoms span a broad spectrum and can manifest at different patient ages. In very early childhood, symptoms are caused by compression of the esophagus and trachea, which results from the vascular ring itself rather than from the diverticulum alone. When symptoms appear in later childhood or adulthood, they are most often caused by dilation of the diverticulum. On the other hand, in 19% to 44% of cases, dissection of the diverticulum wall or spontaneous rupture occurs. According to the literature, degeneration of the media of the diverticulum wall leads to its dilation, and surgical intervention should be indicated once it grows. According to individual studies, surgical correction is therefore recommended in asymptomatic patients once the diverticulum reaches a size of 50 mm. On the other hand, there are currently no unambiguous indication criteria or recommended treatment protocols for this condition.

Several options for surgical indication are currently described. In childhood, many authors use exclusion of the diverticulum without reconstruction of the subclavian artery, which is very well tolerated in children. On the other hand, such a procedure is considered suboptimal in adult patients and is not currently recommended. In adult patients, replacement of a segment of the descending aorta with reimplantation of the aberrant subclavian artery at this site is preferred. Another option is replacement of the descending aorta with bypass grafting of the subclavian artery. Revascularization can thus be performed in situ, or by connecting the subclavian artery to the ipsilateral common carotid artery. In situ reconstruction offers two advantages - a comprehensive single-step solution, and avoidance of a separate bypass operation in the neck with its risk of stroke and injury to the recurrent laryngeal and phrenic nerves. The risk of later bypass thrombosis, however, cannot be overlooked. A drawback of in situ reconstruction is the length of the reconstructed subclavian artery. Nevertheless, it remains the most recommended approach. A further option is replacement of the entire aortic arch together with a segment of the descending

aorta. The trend toward endovascular medicine has also achieved success in treating this anatomical variant. Given the number of alternatives and the lack of clarity in treatment recommendations for Kommerell's diverticulum, it can be said that no ideal treatment approach exists, and every surgical variant carries its own specific risks.

Surgical outcomes are relatively satisfactory, both after in situ reconstruction of the subclavian artery and after extra-anatomic repair. According to the literature, early mortality for acute procedures is around 11%, while for endovascular intervention the 1-year mortality is about 2.6%. Nevertheless, this method is sometimes criticized because of the lack of aneurysm sac volume reduction and the risk of endoleak. This can indeed fail to relieve the symptoms, which are generally caused mainly by compression of the surrounding structures.

Double aortic arch arises from persistence of both the right and left fourth primitive aortic arches, with each side giving rise to its own ipsilateral common carotid artery and subclavian artery. This anomaly most often presents as a vascular ring, in which encirclement of the trachea and esophagus causes their compression, leading to dyspnea and/or dysphagia. It manifests already in early childhood, usually as dyspnea, wheezing, and stridor (worsening when the child cries). The right-sided component is usually more developed, with a larger diameter; in some cases the non-dominant left-sided component is atrophic and present only as a fibrous cord (in which case the condition can be defined as a right-sided aortic arch). A preserved fibrous cord, unlike an isolated right-sided arch, causes symptoms of the vascular ring to appear already in early childhood. When symptoms are caused by a vascular ring, whether from a complete double arch or a right-sided arch with a fibrous left side, ligation and division of one side is indicated, depending on which side is dominant.

A right-sided aortic arch arises from preservation of the right-sided fourth primitive aortic arch, with more or less complete atresia of the left-sided fourth arch. Depending on where the left-sided segment regresses, several anatomical variants of this diagnosis arise (Figure 23).

A right-sided aortic arch with an aberrant left subclavian artery and a retroesophageal diverticulum is the most common variant of this diagnosis. This pathology arises from regression of the left fourth primitive aortic arch between the origins of the common carotid and subclavian arteries. A segment of the left dorsal aorta often persists, forming a retroesophageal diverticulum, from which the left subclavian artery then arises. After double aortic arch, this condition is the second most common malformation causing a vascular ring with symptoms of esophageal and airway compression in early childhood. It may further be accompanied by a persistent left-sided ductus arteriosus or ligamentum arteriosum, located between the pulmonary artery and the retroesophageal diverticulum.

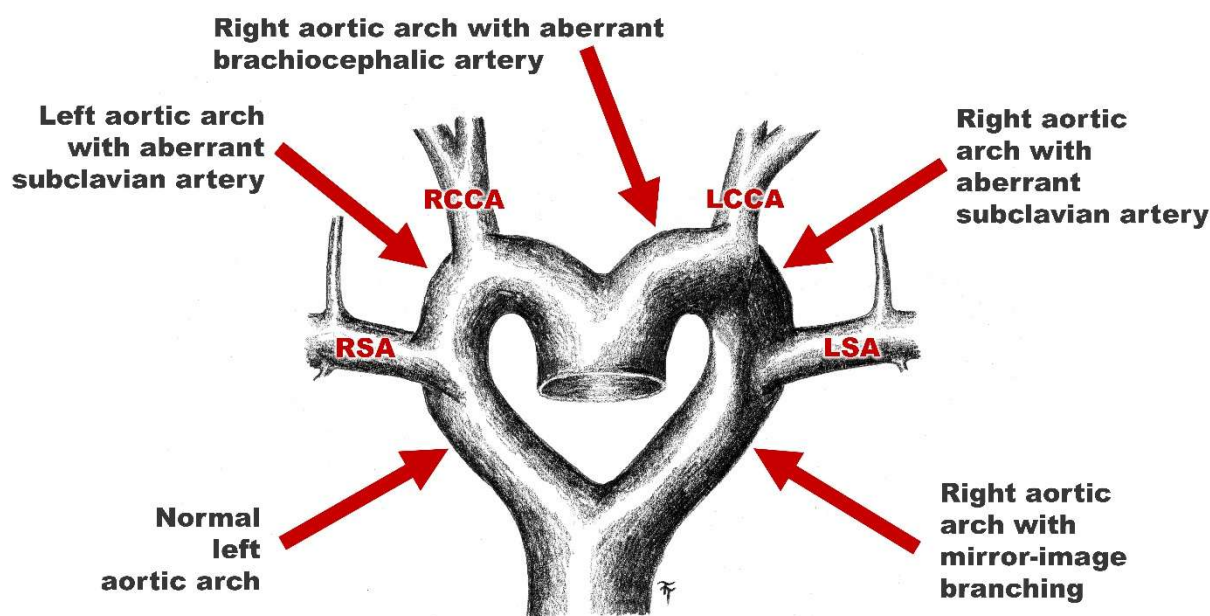


Figure 23 Embryogenesis of the aortic arch and the etiopathogenesis of congenital defects (arrows indicate the site of arch interruption leading to specific congenital anomalies; LCCA - left common carotid artery, LSA - left subclavian artery, RCCA - right common carotid artery, RSA - right subclavian artery)

A second variant of right-sided aortic arch is the presence of an aberrant left subclavian artery without a retroesophageal diverticulum. It arises from regression of both the left fourth and sixth primitive aortic arches, so the left dorsal aorta does not contribute to the definitive descending aorta. An important feature is the absence of a left-sided ductus arteriosus, so the vascular ring is definitively interrupted. Any coexisting coarctation of the aorta can then occur on the right arch at the usual level, corresponding to the left-sided variant. The diagnosis can be asymptomatic on its own, but is often associated with other cardiac anomalies, such as tetralogy of Fallot, and less often coarctation of the aorta.

With a right-sided aortic arch, a left-sided brachiocephalic trunk can also be preserved, with atresia of the left-sided arch occurring beyond the origin of the left common carotid artery. From the right-sided arch, the right common carotid artery, the right subclavian artery, and the left-sided brachiocephalic trunk with a retroesophageal course then arise in sequence. In this case, the ductus arteriosus also arises from the left-sided brachiocephalic trunk, creating a vascular ring.

A right-sided aortic arch with mirror-image branching is the second most common variant of right-sided arch. In this case, the left-sided arch regresses below the origin of the seventh intersegmental artery. This diagnosis is associated with congenital heart defects such as tetralogy of Fallot, truncus arteriosus, tricuspid atresia, and transposition of the great arteries with pulmonary valve stenosis in up to 98% of cases. This configuration typically does not create a vascular ring. The anomaly is often associated with a left-sided ductus arteriosus connecting the left pulmonary

artery and the left subclavian artery, though this too does not create a vascular ring. A right-sided ductus arteriosus is rarer. Even less commonly described is a ductus arteriosus connecting the left pulmonary artery and the descending aorta, as a remnant of the sixth left-sided arterial arch. This rare combination does, in turn, present as a vascular ring.

Isolation of an aortic branch is a rare diagnosis arising from two pathological atresias of the aortic arch, resulting in separation of one branch, which remains connected only to the pulmonary circulation via a persistent ductus arteriosus. An isolated left subclavian artery is the most common form of aortic branch isolation and is associated with other congenital heart defects in about half of cases. It arises from regression of the left fourth aortic arch both above and below the origin of the subclavian artery. The right-sided aortic arch then gives rise, in sequence, to the left common carotid artery, the right common carotid artery, and the right subclavian artery. This anomaly can lead to congenital subclavian steal syndrome with vertebrobasilar insufficiency.

Double-lumen aorta is one of the rare congenital malformations of the aortic arch, with an incidence of 1:330 among all congenital heart defects. This anomaly was first described by Van Praagh in 1969, who hypothesized persistence of the embryonic fifth aortic arch as the caudal component of the double-lumen arch in adulthood. A persistent fifth aortic arch has two forms: a systemic-systemic connection and a systemic-pulmonary connection. The double lumen begins on the arch near the origin of the brachiocephalic trunk. The systemic-systemic form of fifth-arch persistence is represented by its subsequent connection with the fourth arch at the level of the descending aorta, giving rise to a double-lumen aorta. The systemic-pulmonary form involves a distal connection of the fifth arch with the sixth arch, which forms the pulmonary artery (Figure 24). There are three subtypes of a preserved fifth aortic arch: double-lumen aortic arch (both lumens are patent), interruption of the more cranial arch arising from the fourth aortic arch (with preserved flow through the more caudal fifth aortic arch), and systemic-pulmonary fifth aortic arch. A preserved fifth aortic arch is frequently associated with coarctation of the aorta, patent ductus arteriosus, bicuspid aortic valve, atrioventricular septal defect, interrupted aortic arch, pulmonary atresia, tetralogy of Fallot, tricuspid atresia, and ventricular septal defect, although isolated occurrence of this diagnosis has also been described in the literature. When a double-lumen aorta is present as a form of fifth aortic arch persistence, the two aortic lumens are separated by a duplication of the aortic wall; unlike a true double aortic arch, however, no major structures pass through the space between them, and no arterial branches arise from the more caudal lumen. A microdeletion of chromosome 22q11.2 has been proposed in the literature as a possible etiology of fifth aortic arch persistence, whether in its systemic-systemic or systemic-pulmonary form.

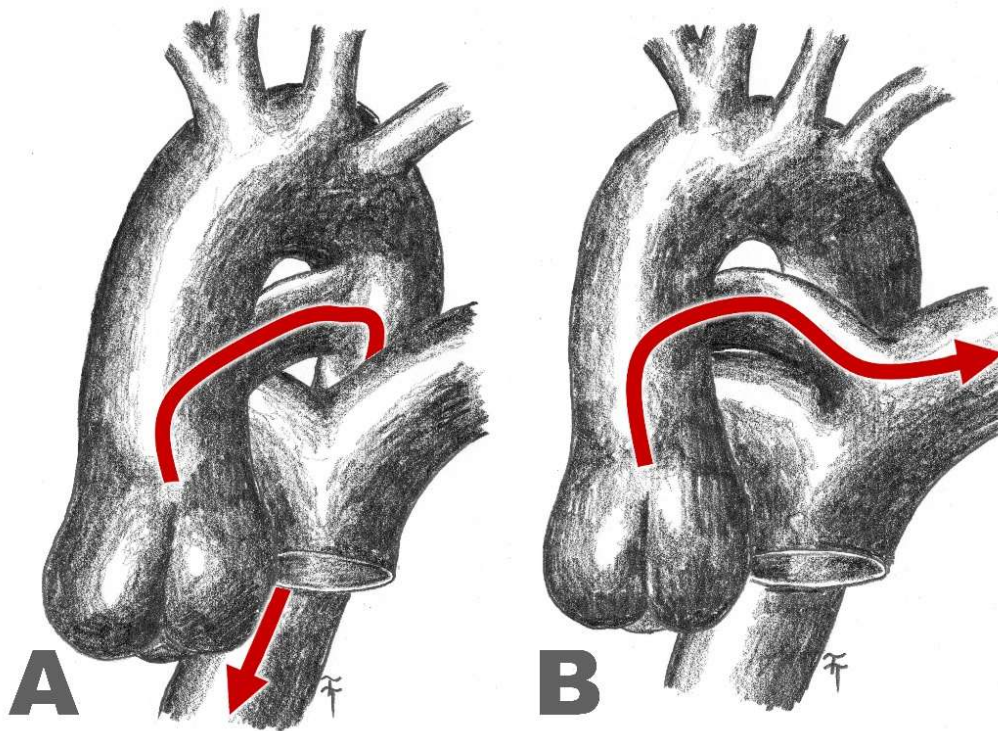


Figure 24 Persistent fifth aortic arch (A - systemic-systemic configuration, B - systemic-pulmonary configuration)

Not all authors accept the theory that a persistent fifth aortic arch is the etiological basis of double-lumen aorta. The fifth aortic arch is present at day eleven of development in only about half of embryos, and it degenerates extremely quickly toward the primitive dorsal aorta. To establish a diagnosis of persistent fifth aortic arch, it must arise proximal to the brachiocephalic trunk and fuse with the fourth aortic arch beyond the origin of the left subclavian artery. According to this theory, double-lumen aorta is thus caused by collateral channels around the fourth aortic arch. Although the etiological origin of double-lumen aorta remains debated, it is in any case a congenital malformation that requires no intervention when asymptomatic, but it can represent a potential complication during certain surgical, and especially endovascular, interventions.

Interrupted aortic arch is a rare malformation, with an incidence of two per 100,000 live births. It is defined as complete interruption of continuity between the ascending and descending aorta. Blood flow is maintained via a patent ductus arteriosus. Its symptoms resemble those of coarctation of the aorta. Symptoms appear during the first two weeks of life and require surgical intervention. Based on the location of the arch interruption, three types of this defect are recognized. In type A interrupted aortic arch, the arch is interrupted distal to the left subclavian artery. Regression occurs at a later embryonic stage, when the subclavian artery has already emerged proximally. It accounts for about 30-40% of all interrupted aortic arches. Type B is characterized by interruption of the arch between the left common carotid artery and the left subclavian artery. It arises from atresia of the fourth aortic arch at an earlier embryonic stage. It is the most common form of interrupted aortic

arch and is often associated with a 22q11.2 chromosomal deletion. It is associated with other anomalies, including bicuspid aortic valve, subaortic stenosis, ventricular septal defect, and subclavian artery anomalies. Clinically, it is associated with minimal left ventricular output, left ventricular outflow tract obstruction, and ventricular septal defect, but with preserved cerebral perfusion. In type C, atresia of the aortic arch occurs at the most proximal location, between the brachiocephalic trunk and the left common carotid artery. It manifests when the most proximal part of the fourth and third aortic arches fails to develop. It is extremely rare, accounting for only 5% of all interrupted aortic arches.

7.4 Atrioventricular Septal Defect

Atrioventricular septal defect is a complex cardiac lesion at the level of the atrioventricular junction. In its partial (incomplete) form, it is an ostium primum atrial septal defect with separate atrioventricular valve annuli, but including a cleft in the anterior leaflet of the mitral valve. The complete form of the defect means the presence of a single atrioventricular valve together with a ventricular septal defect.

Given the pressure gradients involved, this is a left-to-right shunt, i.e., a non-cyanotic congenital heart defect. However, volume overload of the right heart and pulmonary vasculature, if the defect is not corrected, leads to Eisenmenger syndrome. Eisenmenger syndrome is a state in which the originally left-to-right shunt reverses into a right-to-left shunt, due to rising pulmonary resistance and right ventricular pressures.

In the complete form of this congenital defect, surgery is indicated by 6 months of age. In the partial form, with repeated monitoring of pressure gradients (to prevent progression toward Eisenmenger syndrome), a single-stage operation can be delayed until 3 to 5 years of age. A partial atrioventricular defect, if the anatomy is suitable, can even be diagnosed in adulthood.

Surgical correction of the partial form consists of mitral valve repair by suturing the cleft in the anterior leaflet, commissural repair, and closure of the atrial septal defect. Notably, classic annuloplasty is ineffective here and must be modified into commissural plication. If plication does not adequately restore valve competence, replacement is necessary. The complete form of the defect requires repair of the ventricular septal defect, correction of both the mitral and tricuspid valves with their anatomical separation, and subsequent closure of the atrial septal defect. Surgical intervention is performed in a single stage. The close anatomical location of the cardiac conduction system poses a risk.

7.5 Ventricular Septal Defect

Ventricular septal defect is the most common congenital heart defect diagnosed in childhood. A large defect leads to a significant shunt, volume overload of the pulmonary circulation, and, if left uncorrected, to Eisenmenger syndrome. With a small, restrictive defect, the volume of the left-to-right shunt is limited, pressures between the left and right ventricles do not equalize, and the defect can therefore be tolerated long-term. In 25 to 50% of patients, small defects close spontaneously by three years of age. An insignificant ventricular septal defect can also be an incidental finding in an adult patient. The indication for surgery in early childhood depends on the hemodynamics of the defect. Closure can be performed in a single-stage operation. With multiple defects, pulmonary artery banding is performed as a first stage, to prevent a rise in pulmonary resistance and to limit the volume of the left-to-right shunt. Definitive correction is performed as a second step, at a later age. A hemodynamically less significant defect can be operated on at a later age even without early banding.

Anatomically, we distinguish a perimembranous defect, at the site of the membranous septum, which is corrected via a right atrial approach. The second type is a subarterial defect, lying in the left ventricular outflow tract, for which the surgical approach is chosen through the right ventricle. An inlet-type defect lies beneath the septal leaflet of the tricuspid valve and the anterior leaflet of the mitral valve. The last two locations are the trabecular defect and the apical defect, which can be multiple. Correction in adulthood is indicated when pulmonary vascular resistance parameters are still favorable and when complications resulting from the defect's presence are present. These complications include heart failure, infective endocarditis, and aortic regurgitation. The parameter that most significantly determines the need for surgical treatment is a pulmonary-to-systemic flow ratio $Q_p/Q_s > 1.5$, confirming volume overload of the pulmonary circulation.

7.6 Tetralogy of Fallot

Tetralogy of Fallot is a set of congenital heart defects. It comprises a ventricular septal defect, an overriding aorta, infundibular pulmonary stenosis, and right ventricular hypertrophy. In combination with a patent ductus arteriosus, the infant tolerates this anatomical state, but once the ductus closes, the defect begins to become cyanotic. Correction is generally performed in two stages. The first operation must promote dilation of the pulmonary vasculature, increasing its volume capacity, and improve left ventricular ejection fraction. In the first step, therefore, generally only a shunt procedure is performed, to redirect blood flow. Options include a classic subclavian-to-pulmonary (Blalock-Taussig) shunt, or an aorto-pulmonary shunt. In the second step, blood flow is then redirected into a physiological configuration. In single-stage primary treatment of tetralogy,

repair of the right ventricular outflow tract and closure of the ventricular defect are performed, together with enlargement of the pulmonary valve and pulmonary trunk.

7.7 Uncorrected and Corrected Transposition of the Great Arteries

Uncorrected transposition of the great arteries is defined by the aorta arising from the right ventricular outflow tract and the pulmonary trunk arising from the left ventricle. In the absence of a septal defect and a ductus arteriosus, the pulmonary and systemic circulations are thus completely separated, and the condition is incompatible with life. Shortly after birth, with a persistent patent ductus arteriosus and atrial septal defect, temporary mixing of blood between the right and left chambers occurs, resulting in a severe, but short-term tolerable, cyanotic defect. Immediately after diagnosis, balloon atrial septostomy is indicated, to keep the atrial septal defect open and to allow early surgical intervention to be planned. Correction of the defect can be performed anatomically or physiologically. Anatomical correction consists of the arterial switch operation, translocating the aorta and pulmonary trunk, with reimplantation of the coronary arteries at the site of the definitive aorta. Physiological correction changes the flow of blood at the atrial level; in the Senning procedure, blood is redirected from the right atrium to the left ventricle and from the left atrium to the right ventricle. Correction at the ventricular level, according to Rastelli, is also possible, meaning closure of the ventricular septal defect so that the left ventricle diverts blood into the right ventricular outflow tract and then into the aorta, while blood from the right ventricle flows into the left ventricular outflow tract and then into the pulmonary trunk.

Corrected transposition of the great arteries is an anatomical state in which blood from the right atrium flows into the left ventricle and then into the pulmonary trunk, while blood from the left atrium flows into the right ventricle and the aorta. This condition, however, is often combined with a ventricular septal defect, pulmonary stenosis, or Ebstein's anomaly, which requires surgical intervention.

7.8 Pulmonary Artery Stenosis

Pulmonary artery stenosis belongs to the broad spectrum of congenital malformations of the great vessels. Based on the pressure gradient at the site of the stenosis, it is classified as mild (gradient less than 50 Torr), moderate (gradient greater than 50 Torr but not more than 100 Torr), or severe (gradient at the site of stenosis greater than 100 Torr). A rise in right ventricular, and subsequently right atrial, pressure, with a frequently present atrial septal defect, leads to a right-to-left shunt, manifesting as a cyanotic heart defect. Balloon valvuloplasty is the first-choice treatment; if ineffective, surgical intervention follows.

7.9 Glenn Shunt and Fontan Circulation

In certain severe congenital lesions of the right heart, physiological circulation cannot be achieved through surgical intervention. This is mainly due to underdevelopment of the muscle mass of one ventricle, which, after correction, would lead to heart failure. For this reason, some more complex or severe congenital heart defects necessitate redirecting blood flow from the venous system of the systemic circulation directly into the arterial system of the pulmonary circulation. Several palliative procedures have been introduced into practice for this purpose. A commonly used temporary solution is the Glenn shunt. This creates an anastomosis between the superior vena cava and the right pulmonary artery, with disconnection of the pulmonary trunk from the right pulmonary artery. Blood from the upper half of the body thus drains directly into the right side of the pulmonary circulation, while blood from the lower half of the body is pumped by the malfunctioning right heart into the left lung. When the connection between the right pulmonary artery and the pulmonary trunk is preserved, this is called a bidirectional shunt. Fontan circulation then completely bypasses the right heart, in which, at the level of the right atrium, blood is diverted from the inferior vena cava into the superior vena cava and then into the right pulmonary artery. The connection between the right and left pulmonary arteries is left open. The pulmonary trunk is closed above the pulmonary valve. The iatrogenically created partition within the right atrium is fenestrated, to divert blood entering the right atrium via the coronary sinus and other small cardiac veins.

8 Most Common Congenital Heart Defects in Adulthood (Adrián Kolesár)

Congenital heart defects presenting in adulthood generally represent a group of non-cyanotic heart defects with a subclinical presentation. On one hand, non-physiological conditions can lead to gradual progression of symptoms, and the patient becomes symptomatic in adulthood. On the other hand, it may be an incidental finding on imaging performed for another reason. In principle, however, any of the diagnoses listed below, if more severely expressed anatomically, can already present in childhood. Besides the diagnoses listed below, adult patients can also present with coarctation of the aorta, aortic arch anomalies, ventricular septal defect, and partial atrioventricular septal defect, which were discussed in the chapter on congenital heart defects in childhood.

8.1 Atrial Septal Defect and Anomalous Pulmonary Venous Return

Atrial septal defect is the most common incidentally diagnosed congenital heart defect in adulthood. It most often occurs as an ostium secundum defect, i.e., a persistent embryonic shunt at the site of the fossa ovalis. Less commonly, it is diagnosed as an ostium primum defect, located

closer to the tricuspid valve, at the apex of Koch's triangle. An unroofed coronary sinus defect is failure of the septum between the coronary sinus and the left atrium to close. In many cases, it is found together with a persistent left superior vena cava. The other two locations for the defect are the superior sinus venosus type and the inferior sinus venosus type, located at the sites where the superior and inferior venae cavae enter the right atrium (Figure 25). A small atrial septal defect is generally asymptomatic. Larger defects cause a significant left-to-right shunt and lead to volume overload of the pulmonary circulation. A gradual rise in pulmonary resistance leads to increasing right atrial pressure, with the development of Eisenmenger syndrome, in which flow through the defect reverses, and the defect becomes a cyanotic right-to-left shunt. At this stage, closure of the defect is no longer appropriate. The rate at which Eisenmenger syndrome develops depends on the size of the defect and the ratio between pulmonary and systemic blood flow, Q_p/Q_s . Small interatrial defects have a diameter of less than 10 mm and $Q_p/Q_s < 1.5$. For larger defects, progression to an irreversible stage of pulmonary resistance is risky enough that closure of the defect is indicated at an earlier stage.

Catheter-based occluder device implantation is used to treat atrial septal defect. The procedure is less burdensome for the patient, but leaving a foreign body in the heart increases the future risk of infective endocarditis. For large defects (greater than 38 mm), or if the rim of the defect is unsuitable for occluder fixation (a rim of less than 5 mm), surgical closure is indicated. It is also indicated when the defect is diagnosed as an incidental finding and the patient is undergoing cardiac surgery for another reason. Small defects can be closed by simple suture. Larger defects require bridging with a pericardial patch. Both primary and secondary defects can almost always be managed surgically with this technique. An unroofed coronary sinus defect, with normal anatomy of the superior vena cava, can, when the anatomy is unfavorable, be closed while leaving the coronary sinus draining into the left atrium. For a large superior sinus venosus defect, the Warden modification is sometimes chosen, in which blood from the pulmonary veins is diverted into the left atrium via the right atrium and the defect, with this portion of the right atrium separated from the rest of the atrium by a pericardial baffle. The azygos vein, which drains into the right atrium at this site, must then be closed, and the superior vena cava connected to the right atrial appendage. On the other hand, in anomalous pulmonary venous return into the right hepatic vein, blood from the hepatic vein, together with the pulmonary venous blood, can similarly be diverted directly into the left heart, as with the coronary sinus. Whether the coronary sinus or the right hepatic vein is diverted into the left atrium, this volume of deoxygenated blood does not cause a clinically significant right-to-left shunt with clinically manifest cyanosis.

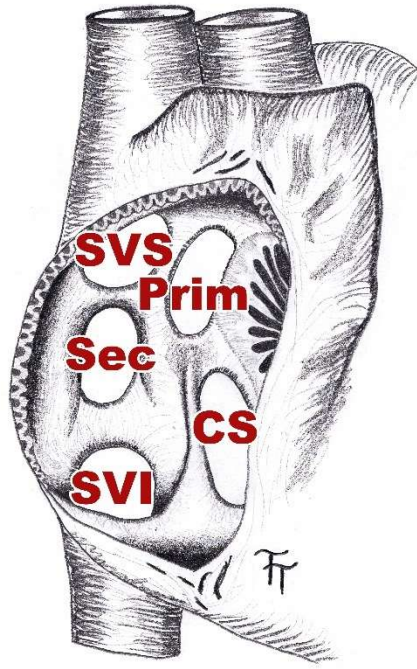


Figure 25 Atrial septal defect and location of individual types (CS - unroofed coronary sinus defect, Prim - primary atrioventricular defect (ostium primum), Sec - secondary atrioventricular defect (ostium secundum), SVI - inferior sinus venosus, SVS - superior sinus venosus).

Atrial septal defect should be treated surgically when there are signs of right heart overload, provided pulmonary resistance has not exceeded 3 Wood units, with the catheter-based technique chosen as the first-line method. In pulmonary hypertension, pulmonary vascular resistance must be quantified by right heart catheterization. When there is diagnostic uncertainty about the effect of closure on hemodynamics and heart failure, a balloon occlusion test of the defect can be performed, quantifying the hemodynamic consequences of closing the shunt. When the pulmonary-to-systemic flow ratio $Q_p/Q_s > 1.5$, closure of the shunt is indicated at a pulmonary resistance below 5 Wood units. If pulmonary resistance is above 5 Wood units, a large symptomatic atrial septal defect can still be closed, but only while leaving a fenestration, to preserve a hemodynamically less significant left-to-right shunt.

8.2 Anomalous Origin of the Coronary Arteries

The origins of the coronary arteries from the aorta do not always follow optimal anatomy. Some variations, for example a separate origin of the left anterior descending branch and the circumflex branch, are usually not clinically manifest, and no treatment is indicated. When a coronary artery arises from a site other than the aorta, however, ischemic heart disease develops to some degree. Coronary collaterals, however, can largely compensate for this pathology, and their dilation results in reversed flow within the abnormally arising coronary branch. Depending on the significance of

the primary pathology and the capacity of the collateral circulation to dilate, the condition can present early, present with physical exertion, or be entirely asymptomatic. Origin of the left coronary artery from the pulmonary trunk is the most severe pathology of this type, and its correction is indicated even in an asymptomatic patient. If the right coronary artery arises from the pulmonary trunk, surgical intervention is indicated for symptoms of angina pectoris or signs of heart failure caused by a decline in right or left ventricular ejection fraction. The goal of surgery in both cases is to redirect blood flow from the aorta into the abnormally arising coronary artery. This can be done by direct transfer, or by creating a tunnel within the pulmonary trunk containing the coronary origin, connected by a bypass graft to the aortic lumen. In adulthood, the last option is occlusion of the coronary origin together with bypass grafting of the entire territory of the affected coronary artery. Symptoms of ischemic heart disease can also occur with an anomalous origin of a coronary artery from the aortic sinus, when its abnormal course causes intermittent occlusion. In symptomatic patients presenting with anginal complaints, surgical correction is indicated for this anatomical anomaly as well.

8.3 Unicuspid and Bicuspid Aortic Valve

Developmental disorders of the aortic valve are among the more common congenital heart defects and are often associated with other congenital malformations of the heart and great vessels. In childhood and early adulthood these malformations are asymptomatic, and both unicuspid and bicuspid aortic valves are competent, with an adequate effective orifice area. Poorer tissue quality and non-ideal blood flow patterns through the valve, which create greater mechanical stress on the tissue, however, lead to much earlier progression of both aortic stenosis and aortic regurgitation, or a combined lesion due to tissue degeneration. In unicuspid valves, hemodynamically significant changes are generally diagnosed in patients in their twenties and thirties, while in bicuspid valves this occurs in patients in their forties and fifties. Moreover, the congenital connective tissue disorder of the aorta associated with this congenital heart defect, together with the pressure conditions in the ascending aorta, leads to an earlier and more frequent incidence of ascending aortic aneurysm. Due to degenerative changes in an otherwise normal valve, so-called bicuspidization of an originally tricuspid valve can also occur, through fusion of one commissure. Anatomically, however, in such a degenerated valve all the commissures extend above the plane where the coronary arteries arise, whereas in the congenital malformation the raphe never reaches this plane (Figure 26).

The indication for surgical intervention follows the same rules as for a tricuspid aortic valve - based on the patient's symptoms and the echocardiographic findings. The indication for a concomitant procedure on the ascending aorta differs, however, in that replacement of the ascending aorta in the affected segment is indicated already at a dilation above 45 mm.

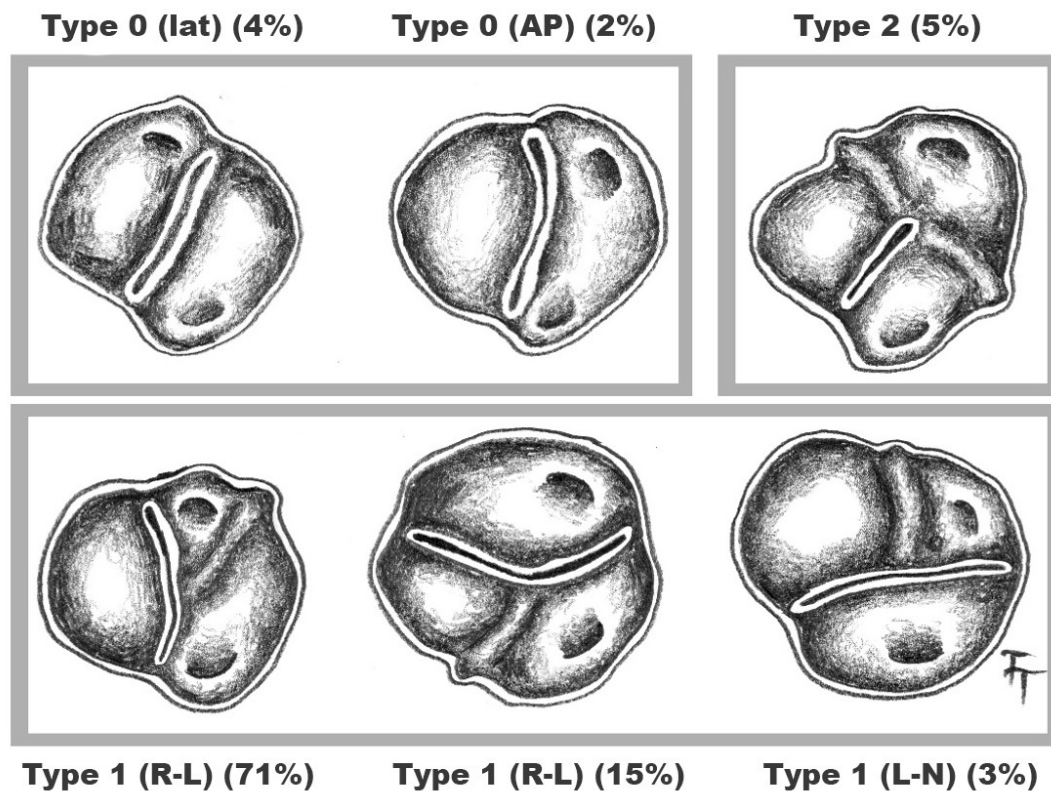


Figure 26 Types of bicuspid aortic valve and their incidence.

8.4 Ebstein's Anomaly of the Tricuspid Valve

Ebstein's anomaly of the tricuspid valve is anatomically defined by the origin of the septal and posterior leaflets of the tricuspid valve below the plane of the tricuspid annulus. The extent of valve involvement ranges from mobile, freely moving leaflets and a competent tricuspid valve, in which case the disease may not be diagnosed until adulthood, through immobile leaflets with short chordae, in which case regurgitation already presents in childhood, to complex right ventricular involvement with its dysfunction, in which case a complex, multi-stage operation is already needed in childhood, and in borderline situations heart transplantation is the only solution. Severe forms are combined with atrial septal defect and patent ductus arteriosus, in which case undiagnosed children reach the stage of Eisenmenger syndrome as teenagers.

Less severe forms, presenting in late childhood or in adults, with progressive signs of tricuspid regurgitation, are an indication for surgical intervention. The Danielson procedure sutures the site of the physiological annulus to the true origin of the posterior leaflet, and annuloplasty is performed at the site of the anterior leaflet. The entire atrialized portion of the right ventricle is thereby excluded from the circulation, and the valve becomes competent. The Carpentier procedure is a more complex surgical technique, requiring intraoperative detachment of the anterior leaflet, removal of the pathologically shortened chordae, exclusion of the atrialized portion of the right ventricle, and subsequent reimplantation of the anterior leaflet at the physiological annulus.

Ebstein's anomaly is very often associated with Wolff-Parkinson-White preexcitation syndrome, and the accessory pathway should be interrupted intraoperatively.

9 Cardiac Tamponade and Pericardial Disease (Adrián Kolesár)

9.1 Pericardial Tamponade

The clinical presentation of pericardial tamponade spans a wide range of symptoms, reflecting both the severity of hemodynamic compromise and the body's physiological reserve. Acute, rapid accumulation of as little as about 100 mL of fluid in the pericardial sac can, given its relatively fixed volume, precipitate critical tamponade. On the other hand, the pericardium can partly adapt to a chronic, long-standing increase in pericardial fluid, and even 1000 mL of accumulated fluid need not lead to hemodynamically significant complications. Chronic, gradual accumulation of pericardial effusion occurs with long-standing inflammatory processes, for example rheumatoid arthritis. A low-pressure chronic pericardial effusion can, however, also decompensate acutely and suddenly become hemodynamically significant, for example in a hypovolemic state due to minimal acute blood loss, or with diuretic therapy.

The etiology of pericardial effusion is diverse. Possible causes include infectious pericarditis due to viruses, bacteria, tuberculosis, and fungi, as well as metabolic consequences of uremia, drug-induced pericarditis, pericarditis associated with rheumatoid arthritis, pericarditis in hypothyroidism, post-radiation pericarditis, and neoplastic pericarditis. A traumatic etiology can also cause acute pericardial tamponade. Frontal injuries are most common, generally injuring the right heart. Because tamponade produces hemostasis, a patient with tamponade has a greater chance of survival compared with a patient with uncontrolled bleeding from a cardiac injury. Blunt trauma is rarely the cause of isolated cardiac injury, but in rare situations can cause trauma to any part of the heart with resulting pericardial tamponade.

Dressler syndrome is post-infarction pericarditis, occurring after transmural myocardial infarction. Inflammation and pain begin 24 to 72 hours after MI and persist for several weeks. Treatment consists of administering non-steroidal anti-inflammatory drugs.

Postoperative tamponade is a delayed occurrence of pericardial effusion, more than 7 days after cardiac surgery. It is a dangerous complication, affecting 0.5 to 6% of patients after heart surgery. It occurs more often in young patients undergoing isolated valve surgery with anticoagulant therapy. In most cases, this condition requires surgical intervention or pericardiocentesis.

Pericardiocentesis is one option for intervention in pericardial tamponade. It is most often performed under ultrasound guidance. Under local anesthesia, in the area between the xiphoid process and the left costal margin, a needle (at a 30-degree angle, directed toward the midpoint of

the right clavicle) connected to an electrocardiogram is inserted into the pericardium. Under ECG and ultrasound guidance, a wire is introduced into the pericardial cavity, followed by a drain. Pericardial fluid is evacuated only slowly, given the possible adverse effect of rapid pressure release on hemodynamic parameters. The most common complication of this method is pneumothorax, so a follow-up chest X-ray is indicated after the procedure. Another option for evacuating pericardial effusion is surgical pericardial fenestration through a small subxiphoid incision, allowing digital exploration of the pericardial cavity and sampling of pericardium for histological examination.

Creating a pericardial window (a connection between the pericardial and peritoneal cavities) is the method of choice for recurrent pericardial tamponade. The procedure is performed via thoracotomy or a subxiphoid incision. The size of the opening should be chosen as a compromise between the risk of tamponade recurrence and the risk of cardiac prolapse. The diaphragmatic pericardium ventral to the phrenic nerve can be removed.

Cardiac surgery for these indications is life-saving, but only palliative, with an important role in diagnosing the underlying process. Short-term results are very good, and the intraoperative risk is minimal; long-term outcomes and the incidence of disease recurrence, however, depend on the etiology of the disease, further medical treatment, patient management, and the body's own response.

9.2 Constrictive Pericarditis

Constrictive pericarditis is a disease with a wide spectrum of etiological factors. Onset of the disease is usually gradual and long-standing. Clinical presentation includes fatigue, edema, hepatic congestion, Kussmaul's sign, and others. Differentiating it from restrictive cardiomyopathy is important.

Surgical pericardiectomy is the only causal treatment for chronic pericardial constriction. Given the frequent occurrence of calcification penetrating into the myocardium, the procedure is technically demanding. Median sternotomy is chosen as the approach, allowing the patient to be placed on cardiopulmonary bypass if complications arise. Some centers place the patient on bypass preventively before the pericardial excision itself, while others use a left anterior thoracotomy as the approach. The procedure is often associated with substantial blood loss. Injury to the coronary arteries running within the epicardium is the most dangerous risk. The most important step is removing the anterior pericardium ventral to both phrenic nerves, as well as the pericardium around both venae cavae and the pulmonary veins. Complete pericardiectomy is often not technically feasible, especially in radiation-induced disease. In such cases, leaving the pericardium in place over the atria and venae cavae is the safer choice for the patient. Intraoperative mortality is reported

to range from 5% to 20%. Even with successful surgical treatment, the risk of disease recurrence is 15%, with 8% of patients requiring reoperation.

Transient pericardial constriction has occasionally been described in the literature, for example after steroid treatment or after cardiac surgery. Cases of pericardial constriction as a consequence of cardiac surgery, or of heart transplantation, have also been described. When the disease is chronic and hemodynamically significant, pericardiectomy is indicated here as well.

9.3 Primary Neoplastic Pericardial Disease

Neoplastic diseases of the heart, and particularly of the pericardium, are extremely rare. Findings can be divided into primary benign and malignant pericardial tumors, and secondary metastasis of malignant tumors from other sites. Both forms are most often asymptomatic. The most common initial symptoms are dyspnea, chest pain, and pericardial effusion. The most common primary pericardial tumors include mesothelial cyst, mesothelioma, or alternatively solitary fibrous tumor, teratoma, bronchogenic cyst, lipoma, lymphangioma, angiosarcoma, hemangioma, schwannoma, and synovial sarcoma.

The most common primary pericardial tumor is mesothelioma, referred to by some authors as solitary fibrous tumor (SFT). In the pleural location, it accounts for only 5% of pleural tumors. By 2002, fewer than 800 cases of this disease at various sites had been published worldwide in the literature. The pleural location is the most typical; it is described more rarely in the pericardium, nasal and oral cavity, pharynx, thyroid gland, breast, kidneys, and spinal cord. The first description of a tumor of this type was published by Wagner in 1870, who used the term localized fibrous mesothelioma. This lesion was later also referred to in the literature as localized mesothelioma, localized fibrous mesothelioma, localized fibrous tumor, solitary fibrous mesothelioma, fibrous mesothelioma, pleural fibroma, submesothelial fibroma, and subserosal fibroma. Today, the term solitary fibrous tumor is preferred. The first pathological characterization of SFT dates from 1931, described by Klemperer and Rabin. Both benign and malignant variants are recognized, including in the pericardial location, with most authors reporting an incidence ratio of 2:1. Solitary fibrous tumor is an uncommon neoplastic disease of submesothelial fibroblastic stem cells of the pleura and pericardium. Given its often asymptomatic course, it is not easy to diagnose. The inability to distinguish the benign from the malignant form of SFT preoperatively is an indication for surgical treatment of every suspicious lesion. Diagnosis is based on histological and immunohistochemical classification of the tumor. Besides the histological nature of the tumor, its macroscopic shape - pedunculated versus sessile - is another important prognostic factor.

Schwannoma is a benign, slow-growing tumor arising from Schwann cells of the epineurium. Intrathoracic schwannoma is most often located in the posterior mediastinum. A primary

schwannoma arising from the pericardium is extremely rare. This tumor's typical anatomy is a fibrous capsule, which allows differential diagnosis already with computed tomography. As with all tumors, the definitive diagnostic method is histological examination after surgical excision. Schwannoma is characterized by the presence of hypercellular Antoni A and hypocellular Antoni B areas. Another key parameter is positivity for S-100 protein. When this type of tumor is suspected, complete surgical excision is indicated, to prevent life-threatening complications of the disease, chief among them cardiac tamponade due to a malignant effusion.

Hemangioma is another rarer form of pericardial tumor. Like other benign tumors, hemangioma is largely asymptomatic. As one of the few tumors, spontaneous regression is also possible with pericardial hemangioma. On the other hand, dangerous complications from this tumor are also possible, such as hemopericardium, heart failure, syncope, and sudden death.

Angiosarcoma accounts for 36% of all cardiac sarcomas. It is also the most common malignant tumor of the heart. Its typical location is the right atrium, but pericardial involvement with this tumor is also not rare. Angiosarcoma shows endothelial cell differentiation, no different from angiosarcomas at extracardiac sites. It is characterized by the presence of vascular channels lined by endothelium, and expression of the endothelial antigens CD34 and CD31. Macroscopically, it most often has a cauliflower-like appearance. In the pericardial location, hemopericardium is a common complication. This tumor carries an unfavorable prognosis for the patient, given its generally early dissemination, with survival of only a few months. Metastases most often occur in the lungs and liver.

Synovial sarcoma has a biphasic structure, comprising spindle-cell and epithelioid components; these patients more often carry the balanced translocation $t(X;18)(p11.2;q11.2)$ genetically. The most common location for this tumor in the heart is the endocardium of the left atrium and the pericardium. Despite the histological characteristic of a biphasic structure, the monophasic variant is more common for tumors located in the heart.

9.4 Secondary Neoplastic Pericardial Disease

In general, any tumor can metastasize to the heart and to the pericardium. Mainly because a diagnosis of either a primary or a secondary pericardial tumor is rare, the incidence of tumor metastases at this site is considerably more common than the incidence of primary tumors. Given the longer survival of patients with oncological disease, as well as the rising incidence of oncological disease in the population, the incidence of secondary pericardial tumors is also increasing. The tumors that most often metastasize to the pericardium are malignant melanoma, lymphoma, various carcinomas, and leukemias. The frequent occurrence of metastases from breast

and lung cancer simply reflects their high incidence in the population and their anatomical proximity.

Metastatic cells can reach the heart and pericardium by four routes: lymphatic, hematogenous, direct extension, or transvenous via the superior and inferior venae cavae. The lymphatic route most often leads to pericardial metastases, and the hematogenous route to myocardial metastases. Direct extension is common with tumors of the lung, esophagus, and breast, given their anatomical proximity, and more often affects the pericardium. The transvenous route is typical for renal cell carcinoma (Grawitz tumor). Of all cardiac metastases, the pericardium is involved in 62% to 81% of cases.

Most cardiac and pericardial metastases are asymptomatic. When they do present, the symptoms are highly variable, reflecting their location, size, and degree of tissue fixation. Symptoms include dyspnea or tachypnea, a systolic murmur, peripheral edema, pleural or pericardial effusion/tamponade, systemic hypotension, peripheral cyanosis, pulsus paradoxus, signs of congestion, and others. Pericardial metastases can lead to pericarditis with effusion, or even to pericardial tamponade. Anginal symptoms, acute myocardial infarction, syncope, and sudden death are also possible. On the other hand, the presence of a pericardial effusion in a patient with cancer need not necessarily be associated with local metastases, but may simply be a reaction to oncological treatment.

Treatment consists mainly of treating the primary tumor. For solitary intracardiac metastases, surgical resection is possible. Pericardial effusions causing symptoms of tamponade require immediate evacuation of the fluid. Radiotherapy is generally possible but often leads to pulmonary or myocardial fibrosis. Doxorubicin, daunorubicin, and cyclophosphamide, used in chemotherapy, also have cardiotoxic side effects.

10 Cardiac Arrhythmias - Surgical Treatment (Tomáš Toporcer)

The treatment of cardiac arrhythmias today encompasses a broad spectrum of catheter-based and device-based procedures performed primarily by electrophysiologists, while the role of the cardiac surgeon is more limited. Nevertheless, several clinical situations remain in which catheter-based therapy alone does not provide satisfactory results and a combined approach involving both electrophysiology and cardiac surgery may offer additional benefit.

Cardiac rhythm disorders are broadly divided into tachyarrhythmias and bradyarrhythmias. Among tachyarrhythmias, atrial fibrillation and atrial flutter are the most important conditions in which cardiac surgery may have a therapeutic role. In these patients, management of the left atrial appendage should also be considered because it is the predominant site of thrombus formation in

atrial fibrillation and therefore an important source of thromboembolic complications. Among bradyarrhythmias, various forms of atrioventricular block are clinically most important and are generally treated by permanent pacemaker implantation. Other invasive treatment modalities for rhythm disorders include cardiac resynchronization therapy in selected patients with heart failure and implantation of an implantable cardioverter-defibrillator for the prevention or treatment of life-threatening ventricular arrhythmias, particularly ventricular tachycardia and ventricular fibrillation.

10.1 Surgical Treatment of Atrial Fibrillation and Flutter

Atrial fibrillation is a supraventricular arrhythmia characterized by rapid, disorganized electrical activation of the atria, resulting in ineffective and uncoordinated atrial contraction. Conduction of impulses through the atrioventricular node is irregular, producing an irregular ventricular rhythm with a variable ventricular rate. According to its temporal pattern, atrial fibrillation is commonly classified into several categories. First-diagnosed atrial fibrillation refers to the first documented episode in a given patient, irrespective of its duration or symptoms. Paroxysmal atrial fibrillation terminates spontaneously or with intervention within 7 days of onset, although most episodes terminate within 48 hours. Persistent atrial fibrillation lasts longer than 7 days and includes episodes requiring cardioversion after this period. Long-standing persistent atrial fibrillation is continuous atrial fibrillation lasting for more than 12 months when a rhythm-control strategy is still being pursued. Permanent atrial fibrillation refers not primarily to its duration, but to a joint decision by the patient and physician to accept the arrhythmia and discontinue further attempts to restore sinus rhythm.

Conversion to sinus rhythm may occur spontaneously or may be achieved pharmacologically or by electrical cardioversion. From a pathophysiological perspective, atrial fibrillation results from a complex interaction between electrical triggers and an arrhythmogenic atrial substrate. In patients with predominantly trigger-mediated, particularly paroxysmal, atrial fibrillation, ectopic electrical activity most commonly originates in or around the pulmonary veins. Electrical isolation of the pulmonary veins therefore represents the cornerstone of catheter ablation and can achieve favorable long-term results in appropriately selected patients. With progression of the disease, structural and electrical remodeling, particularly atrial fibrosis and dilatation, increasingly contribute to maintenance of the arrhythmia. This substrate-dependent component is especially important in persistent and long-standing persistent atrial fibrillation and makes both catheter-based and surgical treatment more complex.

Atrial flutter is a macroreentrant atrial tachyarrhythmia characterized by rapid and regular atrial activation, typically at a rate of approximately 250–350 beats per minute. Because the atrioventricular node usually conducts only a proportion of these impulses to the ventricles,

ventricular conduction commonly occurs in ratios such as 2:1, 3:1, or 4:1. Typical atrial flutter is caused by a macroreentrant circuit in the right atrium that is dependent on conduction through the cavotricuspid isthmus, located between the tricuspid annulus and the inferior vena cava. Because this anatomical pathway is well defined, catheter ablation of the cavotricuspid isthmus achieves a high rate of long-term success. Atypical atrial flutter refers to macroreentrant atrial tachycardias that are not dependent on the cavotricuspid isthmus and may arise in either atrium, frequently in association with atrial scarring or previous surgical or catheter-based ablation procedures. Their mechanisms are more variable, and treatment is therefore generally more complex and less predictable than in typical atrial flutter.

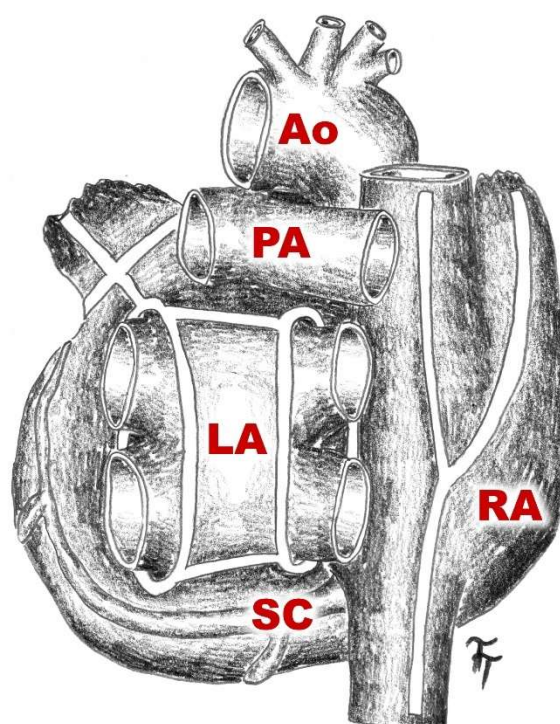


Figure 27 Lesion lines of cryoablation or radiofrequency ablation in the Cox-Maze IV procedure (for completeness, the lines must still be connected to the mitral valve annulus and the cavotricuspid isthmus, which could not be shown in the illustration) (Ao - aorta; LA - left atrium; PA - pulmonary trunk; RA - right atrium; SC - coronary sinus).

Both catheter-based and surgical treatment of atrial fibrillation and atrial flutter aim to interrupt the abnormal electrical circuits or electrically isolate the sites responsible for initiating and maintaining the arrhythmia, thereby allowing sinus rhythm to be restored and maintained. The principal surgical treatment of atrial fibrillation is the Cox-Maze procedure, in which a predefined pattern of electrically nonconductive lesions is created within the atria to prevent the propagation of pathological reentrant electrical activity while preserving conduction from the sinus node to the atrioventricular node (Figure 27). In the original Cox-Maze procedure, these lesions were created using a “cut-and-sew” technique, in which the atrial wall was surgically incised along predetermined lines and subsequently resutured. Healing resulted in fibrous scars that acted as

barriers to electrical conduction. The lesion pattern was progressively modified to improve efficacy and simplify the procedure. Contemporary surgical ablation most commonly uses the Cox-Maze IV lesion set, in which most of the traditional surgical incisions have been replaced by lesions created using radiofrequency energy and/or cryoablation. These energy sources produce controlled transmural myocardial injury, resulting in electrically nonconductive scar tissue while preserving the structural integrity of the atrial wall and eliminating the need for extensive atrial incision and resuturing. These procedures are commonly referred to as radiofrequency or cryoablation-based Maze procedures.

The first components of the Maze lesion set are the circumferential lesions used to isolate the pulmonary veins. Additional connecting lesions are then created across the posterior wall of the left atrium, forming a so-called posterior box lesion that electrically isolates the posterior left atrial wall together with the pulmonary vein ostia. The left atrial appendage is excluded or occluded, and a further lesion connects the left atrial lesion set to the mitral annulus. In the right atrium, lesions are created toward the superior and inferior venae cavae and toward the tricuspid annulus, including a line across the cavotricuspid isthmus to prevent typical atrial flutter. In selected patients with paroxysmal atrial fibrillation, pulmonary vein isolation alone may be sufficient, whereas more extensive lesion sets are generally required in persistent and long-standing persistent atrial fibrillation. The resulting lines of electrically nonconductive scar tissue create a controlled pathway for atrial activation and prevent maintenance of pathological reentrant circuits.

A Maze procedure may be performed as a stand-alone operation using minimally invasive thoracoscopic techniques or as part of a hybrid approach combining surgical epicardial ablation with catheter-based endocardial electrophysiological treatment. Such strategies are considered particularly in symptomatic patients in whom antiarrhythmic therapy and previous catheter ablation have failed or are unsuitable. Much more commonly, however, surgical ablation is performed concomitantly in patients already undergoing cardiac surgery for another indication. It is particularly well established in patients with atrial fibrillation undergoing mitral valve surgery, while it may also be considered during other cardiac procedures after taking into account the likelihood of maintaining sinus rhythm and factors associated with recurrence, including marked left atrial enlargement, long-standing atrial fibrillation, advanced age, renal dysfunction, and other cardiovascular comorbidities.

Surgical exclusion or occlusion of the left atrial appendage is an important adjunct in patients with atrial fibrillation undergoing cardiac surgery because it reduces the risk of thromboembolic complications. It should generally be regarded as complementary to, rather than a replacement for, oral anticoagulation when anticoagulation is otherwise indicated. The left atrial appendage may be

managed surgically by excision, internal closure, or external clipping, and may also be occluded percutaneously using a dedicated device. Thoracoscopic epicardial exclusion represents another minimally invasive option in selected patients.

10.2 Pacemaker and Defibrillator Implantation and Resynchronization Therapy

Implantation of a permanent pacemaker, implantable cardioverter-defibrillator (ICD), or cardiac resynchronization therapy (CRT) device is indicated according to the underlying electrophysiological disorder and the patient's clinical condition. In conventional transvenous pacing, leads are introduced through the venous system, most commonly via the subclavian or axillary vein, and advanced through the superior vena cava into the right heart. A ventricular pacing lead is usually positioned in the right ventricle, while an additional atrial lead may be placed in the right atrium when atrioventricular synchrony is required. Conventional right ventricular pacing produces ventricular activation that resembles left bundle branch block and may therefore be less physiologic from a hemodynamic perspective. In patients requiring biventricular pacing as part of cardiac resynchronization therapy for heart failure, an additional lead is advanced through the coronary sinus into a suitable cardiac vein to stimulate the left ventricular myocardium.

For ICD implantation, the transvenous lead system follows a similar route, while the pulse generator is usually implanted in a subcutaneous or subpectoral pocket in the upper chest. Transvenous leads may interfere with intracardiac structures, particularly the tricuspid valve, and are also associated with risks of venous thrombosis, lead-related infection, and infective endocarditis. In patients undergoing open cardiac surgery in whom permanent pacing or, more commonly, cardiac resynchronization therapy is expected to be required, an epicardial lead may be placed directly on the left ventricular surface at a site selected to achieve effective ventricular activation and, where possible, QRS narrowing. Epicardial left ventricular leads can also be implanted as a stand-alone procedure through a limited thoracotomy or thoracoscopic approach when transvenous placement through the coronary sinus is unsuccessful or anatomically unsuitable. Because epicardial leads avoid passage through the central veins and right-sided cardiac structures, they eliminate several complications related specifically to transvenous lead systems, although they remain associated with their own surgical and device-related risks.

11 Injuries of the Heart and Great Vessels (Tomáš Toporcer)

The most common mechanisms of traumatic cardiac injury are blunt cardiac injury and penetrating trauma to the chest. Depending on the mechanism and severity of injury, hemopericardium, hemothorax, and cardiac tamponade may develop. Penetrating injuries, including stab wounds, most commonly involve the right ventricle because of its anterior

anatomical position, although the left ventricle and other cardiac structures may also be affected. Traumatic injury of the thoracic aorta, particularly after high-energy deceleration trauma, represents another potentially life-threatening condition. Severe injuries frequently require urgent surgical or endovascular treatment, including exploration of the pericardial or pleural cavities, evacuation of hemopericardium or hemothorax, control of active bleeding, and direct repair of myocardial lacerations. Traumatic aortic injury may be treated by open surgical repair or replacement of the affected aortic segment, although thoracic endovascular aortic repair (TEVAR) has become an important treatment option for suitable lesions. In complex injuries involving the aorta or great vessels, hybrid surgical and endovascular approaches may also be required.

Cardiac and great-vessel injuries encompass a very broad spectrum of severity. At one end are minor injuries associated with limited symptoms, favorable prognosis, and no permanent sequelae; at the other are catastrophic injuries associated with high prehospital mortality and a substantial risk of long-term complications among survivors. Cardiac trauma is frequently accompanied by injuries to the chest wall, lungs, pleural cavities, and other intrathoracic structures. According to the mechanism of injury, thoracic cardiovascular trauma can broadly be divided into blunt cardiac injury, penetrating cardiac injury, blunt traumatic injury of the aorta and great vessels, and penetrating injury of the great vessels. Blunt cardiac injury most commonly results from direct compression or impact to the chest, whereas traumatic aortic injury is characteristically associated with rapid deceleration. Penetrating cardiac and great-vessel injuries are less common but are particularly dangerous because they may cause rapid hemorrhage, cardiac tamponade, circulatory collapse, and death.

11.1 Blunt Cardiac Injury

Blunt cardiac injury is one of the most common forms of cardiac trauma and usually occurs as part of polytrauma, particularly after road traffic accidents, although it may also occur as an isolated injury. The mechanism typically involves compression of the heart between the sternum and the vertebral column, often combined with rapid deceleration. Because the mechanism is frequently complex, myocardial contusion may coexist with injuries to the valves, coronary arteries, conduction system, pericardium, or great vessels. An apparently unilateral chest wall injury does not exclude contralateral intrathoracic injury. Owing to its anterior position, the right ventricle is particularly vulnerable and may also be injured by displaced rib or sternal fragments.

Clinical presentation may be delayed or initially obscured by more apparent injuries to other organs. Blunt cardiac injury should therefore be considered particularly after high-energy mechanisms such as high-speed motor vehicle collisions, pedestrian impact, or falls from height. Physical examination may reveal chest wall deformity, crepitus from rib fractures, reduced breath

sounds associated with pneumothorax or hemothorax, and, in the case of traumatic valvular injury, a newly developed cardiac murmur. In patients with significant hemopericardium and tamponade, findings may include hypotension, elevated jugular venous pressure, and muffled heart sounds, although the complete Beck triad is not always present. Bedside echocardiography is an important initial imaging method for detecting pericardial effusion, tamponade, ventricular dysfunction, and major valvular injury, while focused thoracic ultrasonography may also identify hemothorax or pneumothorax. Further diagnostic assessment may include comprehensive echocardiography, chest radiography, and computed tomography, depending on the patient's hemodynamic status and associated injuries. Electrocardiography may reveal ischemic changes, arrhythmias, or conduction disturbances resulting from myocardial, coronary, or conduction-system injury. Cardiac troponin is the most useful biochemical marker of myocardial injury, whereas CK and CK-MB are less specific and are of limited additional diagnostic value in contemporary practice.

Treatment is primarily directed toward stabilization of the circulation and management of life-threatening associated injuries. Hemodynamically significant pericardial effusion or tamponade requires urgent decompression, most often by pericardiocentesis or surgical drainage depending on the clinical situation and suspected mechanism of injury. Persistent or high-volume bleeding after drainage should raise suspicion of ongoing cardiac or great-vessel injury and may require surgical exploration. If active bleeding from the cardiac chambers, ascending aorta, or aortic arch is suspected, rapid transfer to a center with immediate cardiac surgical capability is essential. Repair of such injuries may require cardiopulmonary bypass and specialized experience in complex thoracic cardiovascular trauma.

11.2 Closed Cardiac Injury

Closed cardiac injuries can be divided into five principal groups according to the type and severity of tissue damage: myocardial concussion, myocardial contusion, myocardial rupture, valvular injury, and trauma-induced acute coronary syndrome.

Myocardial concussion represents the mildest form of myocardial injury and is usually caused by blunt impact or a deceleration mechanism, often during sports or falls. It is not associated with structural myocardial damage and generally does not cause elevation of cardiac-specific biomarkers or persistent electrocardiographic abnormalities. Nevertheless, transient rhythm disturbances may occur, ranging from supraventricular tachyarrhythmias to potentially life-threatening ventricular arrhythmias.

Myocardial contusion involves structural injury to the myocardium with associated microvascular damage and is most frequently pronounced in the right ventricle because of its anterior position. Electrocardiographic abnormalities may include ST-segment elevation or

depression, conduction disturbances, and atrioventricular block of varying degree. The risk of arrhythmias may be further increased by concomitant factors such as alcohol or other substances. Myocardial contusion may also be associated with pericardial effusion of varying extent. In hemodynamically stable patients, further assessment in a center with appropriate cardiology and cardiac surgical facilities should be considered when clinically significant myocardial injury or pericardial effusion is present. The extent of the effusion and its hemodynamic effect determine the need for pericardial drainage or surgical exploration, particularly when tamponade develops or persistent bleeding is suspected.

The most severe form of closed myocardial injury is cardiac rupture, which may involve different anatomical structures. Rupture of the ventricular free wall can rapidly result in hemopericardium and acute cardiac tamponade, with immediate hemodynamic collapse. Atrial rupture is less common. Early recognition by echocardiography or other appropriate imaging and immediate surgical treatment are therefore critical. A similar mechanism of blunt or deceleration injury may damage the cardiac valves and produce acute regurgitation. Injury to the valve leaflets may affect the aortic, mitral, or tricuspid valve, whereas rupture of papillary muscles or chordae tendineae produces acute atrioventricular valve regurgitation. Traumatic injury may also involve the coronary arteries through dissection, thrombosis, rupture, or external compression, leading to post-traumatic acute coronary syndrome (ACS). Its clinical presentation may resemble ACS of non-traumatic origin and, depending on the extent of myocardial injury, may subsequently be complicated by the mechanical complications of myocardial infarction described in the corresponding chapter.

11.3 Closed Injury of the Aorta and Great Vessels

Cranio-cerebral injury is the leading cause of death after road traffic accidents, while blunt traumatic injury of the aorta and great vessels represents another major cause of early mortality. Traumatic aortic injury is responsible for a substantial proportion of deaths following high-energy deceleration trauma, and most patients with complete aortic disruption die at the scene or shortly after the accident. The risk is increased in high-speed collisions, particularly when appropriate restraint systems such as seatbelts and airbags are not used or fail to deploy. According to the extent of aortic wall injury, as assessed primarily by computed tomography angiography (CTA), blunt traumatic aortic injury is commonly divided into four grades.

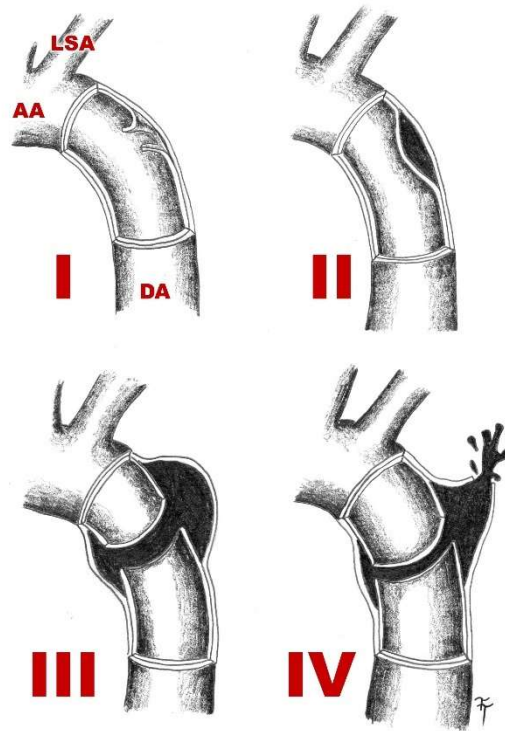


Figure 28 Stages of closed injury of the aorta and great vessels (AA - aortic arch, DA - descending aorta).

Grade I represents an intimal tear without formation of an intramural hematoma. On computed tomography angiography, a small intimal flap or irregularity may be visible, without pseudoaneurysm formation or extravasation of contrast beyond the aortic wall. Grade II is characterized by an intramural hematoma, resulting from hemorrhage within the aortic wall, usually involving the media. Imaging demonstrates localized or circumferential thickening of the aortic wall, sometimes protruding into the lumen, without contrast extravasation. Grade III represents a traumatic pseudoaneurysm, in which disruption extends through the intima and media while the adventitia and surrounding tissues contain the injury. Imaging demonstrates focal bulging beyond the normal contour of the aorta, but without free extravasation of contrast. Grade IV is complete aortic rupture, with disruption of all layers of the aortic wall and active extravasation of blood and contrast outside the vessel. Rapid exsanguination and death may occur when bleeding progresses freely into a large body cavity, most commonly the left pleural cavity. If the rupture is temporarily contained by surrounding mediastinal tissues, however, short-term survival is possible, allowing emergency definitive treatment (Figure 28).

Blunt traumatic aortic injury most commonly results from rapid deceleration, with the pattern of injury influenced by differences in mobility and fixation of individual aortic segments. The ligamentum arteriosum and the origins of the supra-aortic branches contribute to fixation of the proximal descending thoracic aorta, creating substantial shear stress at the aortic isthmus during sudden deceleration. Consequently, the aortic isthmus, immediately distal to the left subclavian

artery, is by far the most common site of injury. Less frequently, the ascending aorta, aortic arch, or more distal descending thoracic aorta may be involved. Injury to the ascending aorta may result in bleeding into the pericardial cavity with acute cardiac tamponade, whereas rupture of the descending thoracic aorta more commonly causes massive left-sided hemothorax. The anatomical location and extent of injury, together with whether bleeding is contained or free, largely determine the immediate clinical presentation and prognosis.

Blunt traumatic aortic injury should be suspected on the basis of the mechanism of trauma and associated clinical findings. High-risk mechanisms include high-speed motor vehicle collisions, particularly impact with a fixed object or an oncoming vehicle, absence of seatbelt restraint, steering-wheel deformation, motorcycle accidents, pedestrian impact, falls from height, crush injuries, and trauma associated with loss of consciousness. Clinical findings that may raise suspicion include shock, fracture of the sternum or first rib, extensive anterior chest-wall bruising, new cardiac murmurs, dyspnea, back or interscapular pain, hemothorax, differences in arterial pressure between the upper limbs or between the upper and lower extremities, and neurological deficits such as paraplegia. Diagnosis relies primarily on imaging. Chest radiography may demonstrate a widened mediastinum or other indirect signs, while computed tomography angiography is the principal diagnostic modality in hemodynamically stable patients. Aortography and echocardiography are used selectively according to the clinical situation and anatomical location of the suspected injury. Initial conservative management is based on anti-impulse therapy aimed at reducing aortic wall stress by controlling heart rate and blood pressure. In hemodynamically stable patients without contraindications, β -blockade is generally used to reduce heart rate and systolic pressure. Lower-grade injuries, particularly grade I and selected grade II lesions, may be managed non-operatively with close imaging surveillance, whereas higher-grade injuries usually require definitive repair. For suitable injuries of the descending thoracic aorta, thoracic endovascular aortic repair (TEVAR) is generally preferred because it excludes the injured segment from the circulation with a stent graft and avoids open thoracotomy. Adequate proximal and distal landing zones are required, and intentional coverage of the left subclavian artery may sometimes be necessary, with revascularization considered according to the individual anatomy and clinical situation. Injuries involving the ascending aorta or aortic arch are substantially more complex because endovascular exclusion may compromise coronary or supra-aortic branch perfusion. These lesions are therefore treated using principles similar to those applied in proximal aortic surgery, most commonly by open replacement of the injured aortic segment or, in selected cases, by hybrid repair. Hybrid strategies may include surgical debranching or transposition of the supra-aortic vessels followed by endovascular stent-graft implantation, replacement of part of the arch with reimplantation of the

head-and-neck vessels followed by endovascular completion, or complex arch reconstruction using elephant trunk (ET) or frozen elephant trunk (FET) techniques. These procedures are discussed in greater detail in the chapter Aortic Aneurysm and Dissection.

11.4 Penetrating Injuries

In peacetime, penetrating cardiac injuries account for only a small proportion of all traumatic injuries but are associated with high mortality. Gunshot wounds generally carry a worse prognosis than stab wounds, with death most commonly resulting from exsanguination or acute pericardial tamponade. The structures most frequently involved are the right ventricle, right atrium, left ventricle, great vessels, and, less commonly, the coronary arteries, particularly the left anterior descending coronary artery. With the increasing use of invasive and transcatheter cardiovascular procedures, iatrogenic penetrating cardiac injuries are also encountered more frequently. These may occur as complications of coronary angiography and percutaneous coronary intervention, pacemaker or implantable cardioverter-defibrillator implantation, endomyocardial biopsy, and transcatheter valve procedures. Cardiac injury caused by fractured ribs or the sternum during cardiopulmonary resuscitation may also be considered an iatrogenic traumatic injury.

Penetrating cardiac injuries most commonly present with cardiac tamponade, hemorrhagic shock, cardiogenic shock, hemothorax, or signs of acute coronary syndrome. Prehospital management should focus on maintaining vital functions, controlling external bleeding when possible, and treating shock. If a penetrating object remains embedded in the chest, it should generally not be removed before definitive surgical control is available, because it may partially tamponade the wound and limit further hemorrhage. A similar principle applies to suspected iatrogenic cardiac injuries. When a needle, catheter, or drain is suspected of having penetrated a cardiac chamber during pericardial or pleural intervention, leaving it in place until surgical assessment may help reduce bleeding and facilitate intraoperative localization of the injury.

Diagnostic imaging may include focused echocardiography and computed tomography, depending on the patient's hemodynamic condition. In patients with severe hemodynamic instability or impending cardiac arrest, emergency thoracotomy may be required to relieve tamponade, control bleeding, perform open cardiac massage, and temporarily repair the cardiac defect. In hemodynamically stable patients, rapid transfer to a cardiac center with immediate cardiac surgical capability is indicated for definitive treatment. Median sternotomy is commonly used because it provides wide exposure of the heart and great vessels and allows rapid institution of cardiopulmonary bypass when necessary. The extent of repair depends on the anatomical site and severity of the injury and on the structures involved (Figure 29).

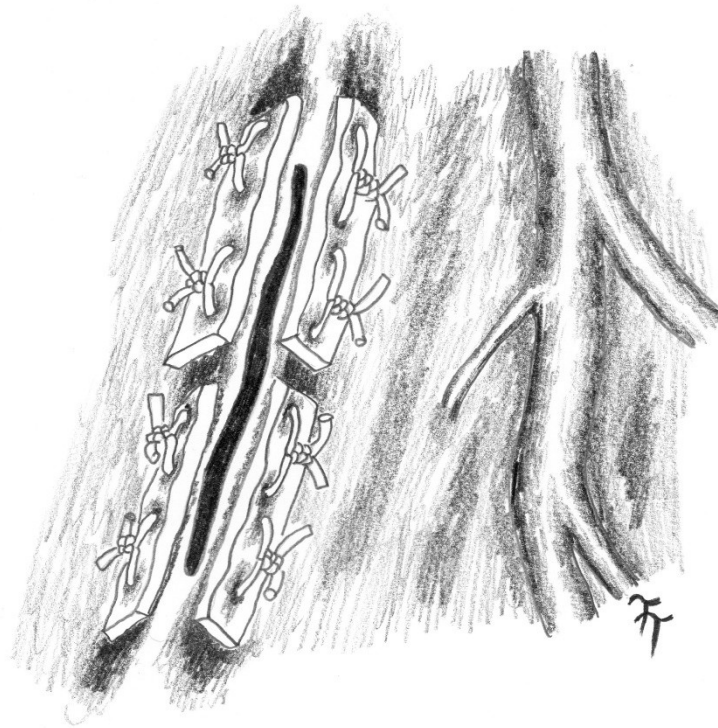


Figure 29 Suture repair of a penetrating right ventricular injury with pledgeted sutures

12 Aortic Aneurysm and Dissection (Adrián Kolesár)

Aortic aneurysm means an increase in the diameter of the aorta beyond the upper limit of its physiological size, with the aneurysm wall containing all the histological layers of a normal aortic wall. For the thoracic aorta and aortic arch, a diameter of 5.5 cm is considered the critical threshold. Other indication criteria for intervention on the ascending aorta include an increase in aortic diameter of more than 1 cm per year, or more than 0.5 cm in six months. In connective tissue disease, especially Marfan syndrome, the indication threshold is lowered to 4.5 cm, and in congenital aortic valve malformation - that is, a bicuspid or unicuspid aortic valve - to 5 cm. When aortic valve surgery is indicated, a procedure on the ascending aorta is also indicated once its diameter exceeds 4.5 cm. Some endovascular guidelines lower the threshold for intervention to as little as 3.8 cm. Aortic dissection means a tear (rupture) in the inner layer of the aortic wall, with bleeding and separation of the individual histological layers of the aortic wall. Because of the high risk of rupture, dissection requires immediate, emergency intervention on a vital indication.

12.1 Etiology of Aortic Aneurysm

Hypertension is the most important risk factor for diseases of the thoracic aorta. All factors that increase pulsatile stress on the aortic wall likewise act as risk factors for these diseases. Genetic predisposition is the second most important risk factor. In 20% of patients diagnosed with thoracic

aortic aneurysm, a genetic mutation associated with the disease has been demonstrated. More than 15 genes have been described in the literature whose mutation can influence the risk of aneurysmal changes in the aortic wall. Among congenital heart abnormalities, bicuspid aortic valve is most strongly associated with these diseases. An association with older age and a higher incidence in men are also well known. On the other hand, some studies document diabetes mellitus as a protective risk factor.

A large proportion of patients with a positive family history and confirmed disease have no identified genetic predisposition. Marfan syndrome, on the other hand, is one of the thoroughly studied etiological bases of the disease, based on a mutation in Fas Binding Factor 1 (FBN1), leading to an imbalance of fibrillin-1 and subsequent aneurysm, or direct dissection, of the aorta. Marfan syndrome is so strongly associated with aortic aneurysm that preventive stabilization of the ascending aorta is indicated in these patients even before it dilates to the size at which replacement would otherwise be indicated. Aneurysmal changes in the aorta result from changes in the medial layer of its wall, which contains more than fifty interconnected microlayers of elastic fibers and smooth muscle fibers. Aneurysm formation is thought to be associated with impaired smooth muscle function and a reduction in the total number of smooth muscle cells, causing insufficient resistance to pressure loading. A change in the extracellular matrix has also been described, in the sense of a reduced proportion of elastic fibers with accumulation of proteoglycans and collagen. Similar changes, though to a lesser degree, can also be observed in healthy individuals depending on age.

12.2 Aneurysm of the Ascending Aorta

Aortic dissection means rupture of the intimal layer, with blood passing between the intima and media, creating a false lumen. Although the first references to this diagnosis are quite historical, the first anatomical description dates from the seventeenth and eighteenth centuries. Maunoir's naming of "dissection" also dates from this same period. The first therapeutic intervention then dates from the early twentieth century, when fenestration was created to treat hypoperfusion. In the mid-twentieth century, Abbott and Poppe developed a method of wrapping the aorta in cellophane to prevent rupture. As the use of cardiopulmonary bypass developed, surgical techniques for treating this condition also improved.

In December 1948, Rudolph Nissen, at Brooklyn Jewish Hospital in New York, operated on Albert Einstein for a thoracic aortic aneurysm. Given the extent of the finding and the possibilities of thoracic aortic medicine at the time, the surgeon only wrapped the ventral two-thirds of the aneurysm in yellow cellophane. Albert Einstein then lived for another 6 years, until the aortic wall

ruptured. Today's medicine offers a range of advanced solutions for this diagnosis, with relatively encouraging postoperative results.

A positive family history of this disease is relatively common and can help confirm the surgeon's indication for surgery in borderline situations. Computed tomography and magnetic resonance imaging, both with an appropriate contrast agent, are the recommended imaging methods. Given that this disease occurs at older ages, coronary angiography and cardiac ultrasound are also indicated, to consider a concomitant cardiac surgical procedure. In a patient indicated for elective surgery, thorough examination of the supra-aortic branches is necessary, confirming the patency of both common and internal carotid arteries as well as the vertebral arteries. If there is a positive personal history of stroke, computed tomography of the brain is further indicated, to establish the extent of damage and to allow postoperative comparative imaging if neurological complications become clinically apparent.

Anesthesia for aortic arch surgery is similar to that for other cardiac surgical interventions. Standard monitoring should include invasive pressure measurement at the radial and femoral arteries, a Swan-Ganz catheter, and a central venous catheter. Intraoperative transesophageal echocardiography is recommended to monitor ejection fraction and adequate flow through the aortic arch. Some centers also use intraoperative electroencephalography. Cerebral oximetry can be used to monitor correct selective antegrade cerebral perfusion. This allows consideration of converting from unilateral to bilateral selective antegrade cerebral perfusion, if the anatomy proves inadequate, as well as early detection of a possible complication, including catheter occlusion or migration. Another option is transcranial Doppler examination, which brings a greater dependence on the examining physician's expertise.

Median sternotomy, the gold standard surgical approach in cardiac surgery, provides adequate visualization of the ascending aorta, the aortic arch, and the proximal part of the descending aorta about 5 cm beyond the origin of the left subclavian artery. If needed, the incision can be extended further along the border of the left sternocleidomastoid muscle. Visualization is further limited by the course of the left brachiocephalic vein, which can be divided if necessary and reconstructed at the end of the operation. If involvement of the aorta requires resection of a larger segment, thoracosternotomy or bilateral anterior thoracotomy can be used as the surgical approach, though this carries a higher risk of postoperative respiratory insufficiency and phrenic nerve injury.

Despite the rapid development of endovascular techniques, including in the region of the aortic arch, open surgical repair remains a widely used strategy. Today's available literature reports very encouraging results for open aortic arch surgery, with a 30-day mortality of 5% to 9.7% and a stroke incidence of 2.8% to 6.2%.

12.3 Aortic Arch Dissection

Aortic dissection is a condition in which a tear in the intimal layer of the aortic wall redirects blood flow into the aortic wall itself. A false lumen for blood flow is thus created between the intima and the media. Acute forms of every type of dissection carry a high mortality. In the Stanford classification, type A dissection is recognized when the intimal tear occurs in the ascending aorta, and type B when the tear is located in the descending aorta (Figure 30). Cardiac surgical intervention is required for type A dissection, and, as an alternative now being introduced, for the form described as non-A non-B dissection. Non-A non-B aortic dissection is a retrogradely propagating aneurysm involving the aortic arch but not the ascending aorta, or one in which the primary tear lies directly within the aortic arch.

Acute type A dissection is a disease with high mortality. Half of patients die within 2 days of onset. Some studies document that mortality rises by 1% for every hour the diagnosis is not established. The high mortality with delayed diagnosis confirms that this disease must be considered with any suspicious presentation. In hemodynamically unstable patients with suspected pericardial tamponade, or with hemorrhagic shock, transport to the operating room is indicated. Tests and procedures such as transesophageal echocardiography or placement of a central venous catheter are, because of the risk of hypertension from a painful procedure, indicated only once in the operating room, or even only under general anesthesia. The first step is to ensure the conditions are met for immediate pericardiotomy and placing the patient on cardiopulmonary bypass.

In hemodynamically stable patients, measuring the pressure in both upper limbs, and titrating treatment to the higher measured value, is important. Stabilizing arterial pressure is necessary to reduce transmural stress on the aortic wall and to reduce the risk of rupture, as well as to prevent further propagation of the dissection by lowering pressure in the false lumen. Systolic pressure must therefore be stabilized to between 90 and 110 mmHg, with a heart rate below 60 beats per minute. Pain management is equally important, in order to reduce catecholamine release. Conservative treatment in stable patients thus includes opioids, beta-blockers, and vasodilators (particularly nitrates). If beta-blockers are contraindicated, calcium channel blockers are the drugs of choice.

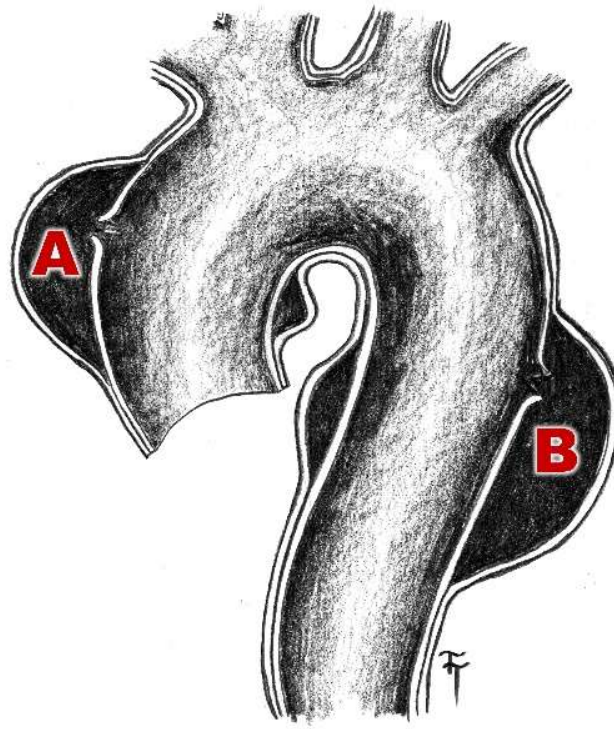


Figure 30 Stanford type A and type B classification of aortic dissection according to entry-tear location.

The goal of urgent surgery is to prevent a state incompatible with life, by redirecting blood flow into the true lumen. Complications of dissection that lead to the patient's death include rupture of the aortic wall into the pericardial or pleural cavity, dissection or occlusion of the coronary artery origins, and severe acute aortic regurgitation. Neither age nor the patient's current neurological status is a contraindication to acute surgery, given the possibility of improvement once blood flow is redirected and hemodynamic parameters are stabilized. Likewise, the time elapsed since the first symptoms, or signs of thrombosis of the false lumen, are no guarantee that further complications will not develop, and are therefore not a reason to withhold surgery. Some studies prefer, in these situations, an elective procedure without unnecessary delay rather than an emergency procedure. The goal of surgery is thus to replace the aorta to the extent it is affected, to manage any resulting complications, and to thoroughly redirect blood flow into the true lumen.

12.4 Surgical Interventions on the Thoracic Aorta and Aortic Arch

The ascending aorta and the aortic valve form a single functional unit, so when assessing an aneurysm or dissection of the ascending aorta, thorough assessment of aortic valve function is necessary. The surgical procedure must then include repair or replacement of all affected parts of the aortic valve, the ascending aorta, and the aortic arch, either in their entirety, or to the extent needed to allow a subsequent endovascular intervention on the descending thoracic aorta or the aortic arch.

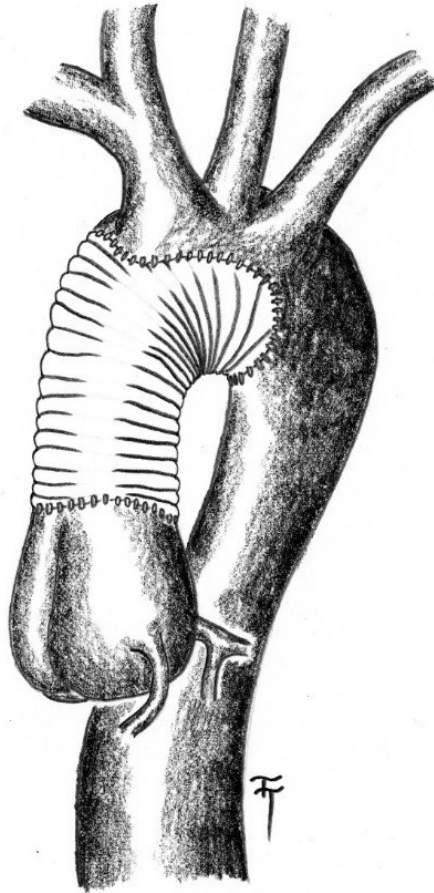


Figure 31 Supracoronary replacement with replacement of the aortic arch concavity (hemiarch).

When the aortic valve and aortic root are involved, the Bentall procedure is the gold standard. The Bentall procedure replaces the aortic valve and aortic root with a conduit combining a valve prosthesis and a linear aortic graft. The conduit is implanted with individual sutures directly into the aortic valve annulus after its excision; buttons of the coronary artery origins are then implanted into it, and the distal end is sutured with an end-to-end anastomosis to a healthy segment of the aorta, or to another prosthesis used to replace the aortic arch. If replacement of the aortic root is not needed, because its size does not meet the indication criteria, but the aortic valve is diseased, the Wheat procedure can be performed. This combines replacement of the aortic valve with a prosthesis and replacement of the ascending aorta from the sinotubular junction distally. Reimplantation of the coronary arteries is not needed, which considerably shortens the duration of cardiopulmonary bypass and the risk of postoperative complications. When the aortic valve and root are normal, only supracoronary replacement of the ascending aorta, from the sinotubular junction distally to the required extent, can be performed (Figure 31).

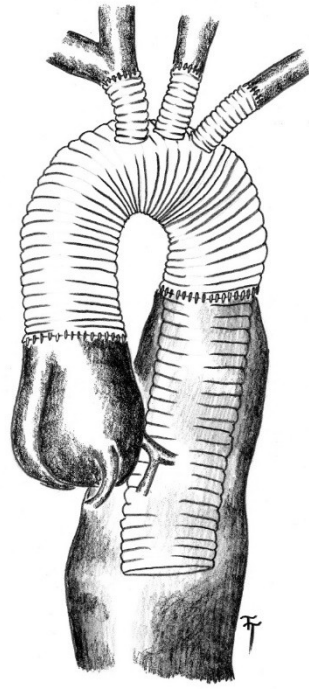


Figure 32 32 Supracoronary replacement of the ascending aorta with frozen elephant trunk.

Distally, the aortic replacement must end at a point where the aortic diameter has already reached physiological values, or where no further distal tear of the aortic intima is present. If such a point cannot be surgically reached from a sternotomy approach, the elephant trunk or frozen elephant trunk technique is chosen (Figure 32). If the aortic arch is not affected, the distal suture line can be placed at the junction of the ascending aorta and the aortic arch with a simple end-to-end anastomosis. If replacement of the concavity of the aortic arch is needed, a so-called hemiarch is performed, in which the excision line is made proximal to the origins of the aorta's head-and-neck branches, but the concavity of the arch is excised. The distal suture line can also be placed beyond the origins of the head-and-neck branches, but these must then be reimplanted into the prosthesis, most often on a single common button. When an aneurysm or dissection extends into the descending aorta, the elephant trunk technique can be used, in which the distal anastomosis with the aorta is created at its non-physiological segment, in the middle of the linear prosthesis. Half of the prosthesis is left hanging freely within the lumen of the descending aorta, and the head-and-neck branches of the aorta are implanted into the proximal portion, either on a common button or individually. The proximal anastomosis is sutured to the healthy part of the ascending aorta, or to the prosthesis that replaced it. The last option is the frozen elephant trunk, in which the linear prosthesis extending into the descending aorta is replaced with a stent graft. A prefabricated device is most often used, combining a stent graft continuing into a linear prosthesis, with prefabricated side branches for suturing the head-and-neck branches of the aorta and the proximal anastomosis. The prosthesis

often also includes one more branch for inserting the arterial cannula of the cardiopulmonary bypass circuit. Both the elephant trunk and the frozen elephant trunk are the first stage of a type III hybrid procedure, in which the descending aorta is later reinforced, if needed, with a further stent graft over the required length.

12.5 Aneurysm of the Descending Thoracic Aorta

Aneurysm of the descending aorta involves the aorta distal to the origin of the left subclavian artery. When the disease is limited to above the diaphragm, it is called descending thoracic aortic aneurysm (DTAA). When the disease crosses the diaphragm and also involves the abdominal aorta, it is called thoracoabdominal aortic aneurysm (TAAA). The view of this diagnosis's etiological cause has changed over time. At the start of the twentieth century, the disease was most often a consequence of tertiary syphilis. Later, and still today, degeneration of the media of the aortic wall is the most important etiological factor in aneurysm formation.

At the time of diagnosis, a large number of patients are asymptomatic, and the diagnosis is made as an incidental finding on imaging. Nearly 50% of patients with this diagnosis may be asymptomatic, with symptoms typically only triggered by aortic wall rupture or dissection. Of the 57% of patients who are symptomatic, rupture itself is the presenting symptom in as many as 9%. The most typical symptom is back pain between the shoulder blades. If the segment of the aorta crossing the diaphragm is dilated, the disease can present as epigastric abdominal pain. Less typical symptoms, arising from compression of surrounding structures, include hoarseness, cough, hemoptysis, dysphagia, and gastrointestinal obstruction or bleeding. More severe symptoms arise from compression of the vagus and recurrent laryngeal nerves. The mycotic form is often associated with erosion of adjacent vertebrae. Computed tomography and magnetic resonance imaging are the recommended first-choice imaging methods when this diagnosis is suspected. Adding a contrast agent to the examination can also reveal an intraluminal thrombus, dissection, intramural hematoma, retroperitoneal hematoma, rupture, and periaortic fibrosis, suggesting an inflammatory etiology. Aortography, now rarely used, remains an available method of choice.

In asymptomatic patients, the anatomical dimensions of the aorta are the indication criteria for surgical treatment. An aortic diameter greater than 5.5 cm, or growth of more than 1 cm per year, represents a risk of rupture and an indication for elective surgery. If the anatomical criteria for surgery are not met, strict conservative treatment of hypertension is indicated, with follow-up at least once a year. In symptomatic patients, meeting the anatomical indication is not strictly necessary. Both endovascular percutaneous intervention and open surgery are treatment options.

When choosing between endovascular and surgical treatment, the most important considerations are the anatomical characteristics of the aneurysm and the patient's physiological reserve. The open

surgical method shows good results and benefits over the long term, but is considerably more burdensome for the patient. Endovascular treatment is an alternative especially for patients with cardiac and renal comorbidities, for patients who have had prior surgery at this site, or for very elderly patients. Compared with open surgery, the endovascular method is characterized by lower periprocedural morbidity and mortality. On the other hand, a narrow landing zone, excessive angulation of the aneurysm, a significant intraluminal thrombus, or marked calcification of the aneurysm wall considerably disadvantage and jeopardize a successful endovascular outcome. For high-risk patients with a narrow landing zone, a hybrid procedure is an option, in which debranching of the aortic arch creates sufficient space for a subsequent endovascular intervention.

12.6 Dissection of the Descending Aorta

Classification of aortic dissection is based on the site where it begins and the location of its propagation. The subtype is then further subclassified based on the time elapsed since its onset. Dissection is considered acute if it arose less than 2 weeks ago. The term chronic is reserved for dissection arising more than 2 months ago. Subacute dissection is an intermediate stage, arising more than 2 weeks but less than 2 months ago. For anatomical classification, two systems are most commonly used today. The DeBakey classification divides aortic dissections based on the location and propagation of the disease (Figure 33). This classification more precisely defines the anatomical extent of disease and is better suited for comparing different treatment approaches in vascular surgery. The Stanford classification is strictly functional, based on the location of the intimal tear.

Aortic dissection is the most common lethal diagnosis affecting the thoracic aorta. Its occurrence is three times more common compared with rupture of the abdominal aorta. Worldwide, the incidence of this disease ranges from 5 to 30 cases per million population per year. Lethal cases that are never diagnosed in time must also be added to this incidence. Several hypotheses exist for the etiological cause of the intimal tear, several of which are based on biochemical abnormalities in the medial layer of the aortic wall. Nevertheless, a direct relationship between intimal tearing and medial necrosis has not been explained. Moreover, medial degeneration is described in only a minority of patients with dissection, mainly in children. Another theory describes bleeding into the aortic wall from the vasa vasorum, weakening the intima and precipitating its rupture. An intramural hematoma, however, is found in fewer than a quarter of patients with dissection. Another group of authors points to penetration of an atherosclerotic ulcer, with subsequent spread into the surrounding tissue, and on this basis argue for the same indication criteria for treating a penetrating atherosclerotic ulcer as for aortic dissection. Despite the lack of clarity about the etiopathogenesis of dissection, several risk factors for its development have been identified, including arterial hypertension, Ehlers-Danlos syndrome, Marfan syndrome, Turner syndrome, Sheehan syndrome,

Cushing syndrome, cystic medial disease of the aorta, aortitis, iatrogenic damage to the aortic wall, bicuspid aortic valve, trauma, coarctation of the aorta, congenital aortic stenosis, polycystic kidney disease, pheochromocytoma, and others. In general, these are factors that increase mechanical loading on the aortic wall, or that reduce its mechanical strength. Hypertension is found in two-thirds of patients with dissection.

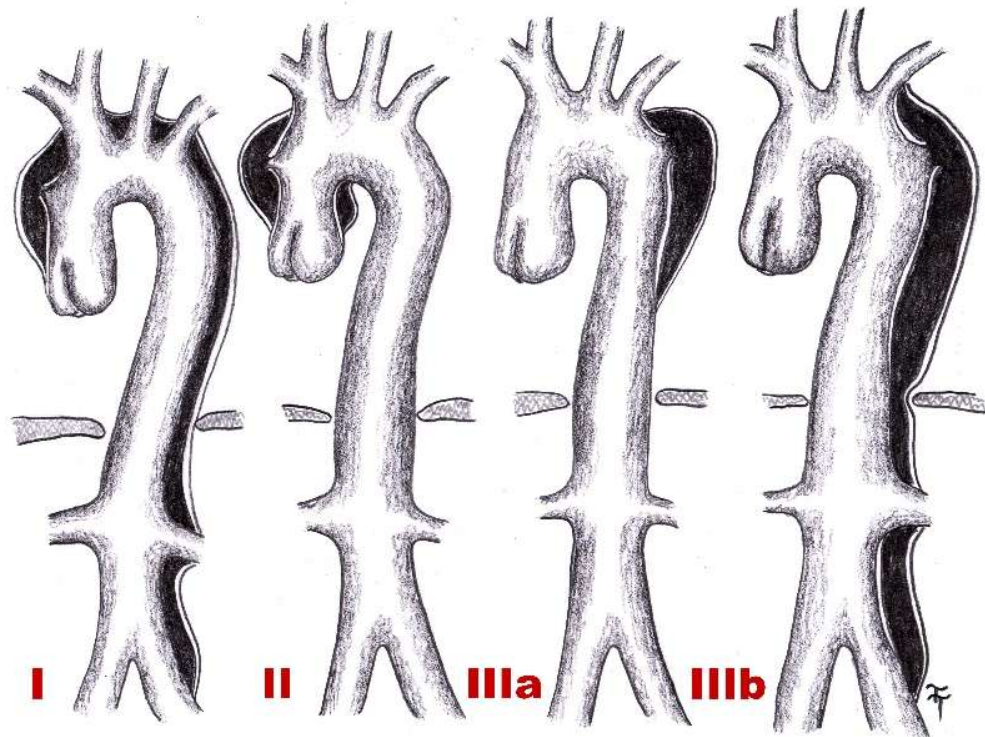


Figure 33 DeBakey classification of thoracic aortic dissection.

Type B aortic dissection accounts for about 40% of all aortic dissections, and its course is less destructive compared with type A. More than half of patients are able to heal the defect with conservative treatment, and only about 20-30% of patients undergo an emergency surgical or interventional procedure. Situations requiring intervention include aortic rupture, aortic expansion, hemodynamic instability, persistent pain, hypertension refractory to treatment, and especially hypoperfusion of peripheral tissues. Aortic rupture and visceral organ hypoperfusion are the most common causes of death in this condition. When conservative treatment is sufficient and the disease does not progress, in-hospital mortality can be expected to be only about 10%. Survival falls to 85% during the first year, and overall mortality at 5-year follow-up can be expected to be around 29%. On the other hand, in patients indicated for surgery, 30-day mortality reaches 30%. These figures have led to intensive adoption of endovascular methods, which show a mortality of around 5% in the same patient group. Given the outcomes of invasive versus conservative treatment, the indication for emergency interventional or surgical treatment of acute type B aortic dissection has traditionally been driven exclusively by the risk of life-threatening complications developing.

Indications for emergency intervention thus include persistent, uncontrollable pain, hypertension that cannot be controlled medically, a rapidly enlarging aortic diameter, progression of the extent of the dissection despite maximized conservative treatment, signs of impending rupture, or hypoperfusion in the territory of branches arising from the affected segment of the aorta. In patients whose acute dissection remains uncomplicated by these criteria, current guidelines also support a pre-emptive strategy: TEVAR performed in the subacute phase, between 14 and 90 days after onset, should be considered in selected patients with high-risk anatomical features, in order to prevent later aortic complications such as false lumen expansion or aneurysm formation, rather than waiting for a complication to develop.

The goal of endovascular intervention, now the first-choice treatment for type B aortic dissection, is to redirect blood flow into the true lumen and to ensure perfusion of tissues supplied by branches arising within the treated segment. Obliteration of the false lumen is desirable, though not mandatory; its thrombosis, however, ensures long-term stabilization of the aortic wall and limits growth of the aneurysm. The stent graft should cover and seal the primary intimal tear, leading to thrombosis of the false lumen and reperfusion of the branch vessels. If reperfusion of the branch vessels is not achieved, fenestration of the intima at the site of origin is an option, allowing blood to pass from the false lumen into the true lumen of the aortic branch. The site can then be covered with a stent. If needed, a further stent can be placed directly into the branch vessel. When the intimal tear is located high, stent graft implantation may also cover the origin of the left subclavian artery, except in situations where the left internal thoracic artery was used for myocardial revascularization, where the circle of Willis is incomplete, where the left vertebral artery is dominant, or where the right vertebral artery is occluded. In these cases, the procedure can only be performed after revascularizing it. When the stent graft must be placed higher, debranching of the aortic arch is required.

Open surgical intervention for dissection of the descending aorta is performed under general anesthesia with selective lung ventilation. Pressure monitoring via both the radial and femoral arteries is necessary. Spinal cord injury leading to paraplegia is a feared complication, occurring in a third of patients. Several protective measures are used to protect the spinal cord, including cerebrospinal fluid drainage, an intraoperative bypass between the left atrium and the femoral artery, and reimplantation of intercostal arteries. A left posterolateral thoracotomy is chosen as the surgical approach, with the patient positioned on their right side. When the abdominal aorta must also be addressed, the incision is extended, and a transperitoneal or retroperitoneal approach is used. Strategies for emergency surgery differ among centers. Some centers recommend minimizing the surgical procedure to the segment of aorta most affected by the dissection, rarely exceeding one-

third of the descending thoracic aorta. Other centers, in contrast, extend the procedure to the entire aneurysmally dilated aorta.

13 Minimally Invasive and Hybrid Methods in Cardiac Surgery (Adrián Kolesár)

Since the early 1960s, cardiac surgery has written its history mainly through surgical interventions performed via median sternotomy. However, following the example of other surgical specialties, cardiac surgery has also sought ways to minimize its invasiveness. Minimizing the invasiveness of the surgical approach does not only have a positive cosmetic effect, which is preferred by younger patients. For a multimorbid, borderline patient, often undergoing an extensive cardiac surgical intervention, infection during sternotomy healing is often a serious, life-threatening complication that significantly worsens the postoperative outcome of an otherwise successful surgical procedure. Prolonged rehabilitation further worsens the results of surgery. Minimizing invasiveness thus finds its most important place particularly in the group of elderly and frail patients.

13.1 Minimally Invasive Procedures

Current guidelines for myocardial revascularization in single-vessel coronary disease with significant involvement of the proximal segment of the left anterior descending branch place percutaneous and surgical intervention on an equal indication footing. This reflects equivalent patient survival when comparing the two approaches. Although surgical intervention provides a significantly lower risk of disease recurrence, with a lower incidence of needing repeat intervention, many patients, even after adequate counseling, choose the less invasive method, because of lower post-intervention morbidity and a faster return to an active life (Table 7). Minimally invasive approaches offer the benefit of rapid rehabilitation and a return to an active life while preserving the positive effects of a fully surgical intervention. The anatomical proximity of the course of the left internal thoracic artery and the left anterior descending branch led to a modification of the surgical approach. Median sternotomy can be replaced with a left anterior thoracotomy in the fourth or fifth intercostal space, which allows visualization of both the internal thoracic artery and the left anterior descending branch. This procedure is called MIDCAB. Without the need for median sternotomy, and without the need for cardiopulmonary bypass with its negative consequences, revascularization of the most important coronary artery is thus possible, using the best available graft, with the best long-term results. Some centers push this approach even further, using thoracoscopic or robotic telemanipulation instruments to harvest both internal thoracic arteries,

allowing more extensive revascularization of more significant coronary disease, without the need for sternotomy.

Table 7 Relative contraindications for minimally invasive mitral valve surgery.

	Patient characteristic
Cardiac function	Left ventricular ejection fraction < 30% Severe right ventricular dysfunction
Comorbidities	Liver dysfunction Renal insufficiency Severe cerebrovascular disease
Patient habitus	History of breast implants Chest wall deformities Body mass index > 40
Related to the procedure	Concomitant revascularization History of right thoracotomy
Respiratory system	Pulmonary hypertension > 45 mmHg Pulmonary adhesions
Valvular disease	Aortic regurgitation Mitral annular calcification Endocarditis
Vascular complications	Calcification of the ascending aorta Dilation of the ascending aorta Aortic aneurysm or dissection Grade 4 aortic disease Severe peripheral vascular disease An implanted inferior vena cava filter

The anatomical location of the mitral valve in a patient with a dilated left ventricle makes it considerably difficult to visualize via a median sternotomy approach. Right thoracotomy is therefore becoming widely used as an alternative surgical approach to the mitral and tricuspid valves and the interatrial septum. With this approach, cardiopulmonary bypass can be established centrally or via peripheral cannulation, which allows the skin incision to be minimized further. The surgical procedure itself can be performed via open thoracotomy, video-assisted, fully thoracoscopically, or with robotic telemanipulation assistance. This approach offers the patient a better cosmetic result and earlier rehabilitation, and often gives the surgeon better visualization of the valve structures. Specific instrumentation is required, however (Table 8).

The location of the aortic valve allows the surgical approach to be minimized to an upper partial J-sternotomy. The incision runs in a cranio-caudal direction along the midline of the sternum, and is directed into the intercostal space on the right at the level of the third or fourth rib. This surgical approach allows cannulation of the aorta as well as the right atrial appendage. A vent can also be placed via the right superior pulmonary vein. This approach thus allows every type of surgical procedure to be performed on the aortic valve, aortic root, and ascending aorta. A further, even less

invasive approach to the aortic valve is a right anterior thoracotomy in the second intercostal space. With this approach, however, cardiopulmonary bypass is established peripherally, to allow better visualization of the aortic valve.

A minimally invasive approach via left thoracotomy or thoracoscopy is also chosen for patients indicated for a Maze procedure or surgical occlusion of the left atrial appendage. This approach can also be used when epicardial pacing lead implantation is needed. A further benefit of the minimally invasive approach is a lower incidence of postoperative pericardial adhesions. This benefit is particularly valuable for multi-stage surgical procedures. Left thoracotomy combined with a partial upper sternotomy can be advantageous for a patient indicated for long-term cardiac support implantation as a bridge to transplantation. Combining these surgical approaches allows full visualization of the cardiac apex and the ascending aorta, while protecting the rest of the pericardial cavity and the retrosternal connective tissue, making dissection of the heart easier at the time of transplantation.

Table 8 Overview of the most important results comparing two surgical approaches to the mitral valve.

	Minimally invasive approach	Sternotomy	<i>p</i>
In-hospital mortality	1%	1.3%	0.6
Reoperation for bleeding	4%	2.3%	0.09
In-hospital stroke	1.1%	2.1%	0.5
Length of hospital stay (days)	7.5	7.0	0.71
Long-term mortality	0.7%	1%	0.46
Stroke at long-term follow-up	0.2%	0.7%	0.2
Cardiopulmonary bypass time (min)	129.2	97	0.01
Ischemic cardiac arrest time (min)	85.6	63.4	0.01
Need for valve replacement with a prosthesis	1.6%	3%	0.66

Minimally invasive approaches also find their place in harvesting venous grafts for coronary artery bypass grafting, when, with the help of specialized instruments, a 60 cm segment of the great saphenous vein - needed to construct 3 coronary bypasses - can be dissected and harvested through just two or three 4 cm incisions.

The results of minimally invasive techniques at centers that favor this surgical approach reflect their benefits for the patient, achieving better outcomes than conventional sternotomy. It must be emphasized, however, that the approach still needs to be carefully individualized for each patient. A minimally invasive approach does not allow immediate management of every unexpected intraoperative complication, and median sternotomy remains the gold standard, allowing the full spectrum of cardiac surgical procedures to be performed from a single, universal approach.

13.2 Hybrid Procedures

Both surgical operation and percutaneous intervention carry certain benefits, which led to the idea of combining these approaches in a single patient. This combination offers the patient the benefits of both techniques and, when appropriately indicated, better postoperative outcomes with lower postoperative morbidity and mortality. A hybrid approach is increasingly chosen in the treatment of ischemic heart disease, in which bypass surgery with an arterial graft is chosen to revascularize the left anterior descending branch, while a percutaneous interventional approach is used to revascularize the remaining coronary arteries. Revascularization of the left anterior descending branch can also be performed using the MIDCAB technique. A hybrid procedure is also the choice for a patient in whom neither full interventional nor full surgical revascularization is possible. This occurs, for example, with specific involvement of the left main coronary artery, where dilation and stent placement are very risky. On the other hand, it may be a patient at an advanced stage of heart failure, or one in whom atherosclerotic disease of the ascending aorta is so severe that using cardiopulmonary bypass would be very risky. Surgical intervention on the left anterior descending branch thus revascularizes this coronary artery without the need to clamp the ascending aorta, and once it is adequately revascularized, a safe percutaneous intervention becomes possible. The revascularized left anterior descending branch then ensures adequate perfusion of the left ventricular myocardium during the demanding and risky invasive procedure.

The second important area where hybrid procedures find their indispensable place is aortic arch surgery. Aneurysm, and especially dissection, of the aortic arch is a diagnosis that poses a very intense threat to the patient's life. While a purely surgical approach necessitates invasively extending the site of intervention, prolonging the time needed to restore circulation during the operation leads to irreversible changes in peripheral tissues, which significantly affects postoperative morbidity and mortality. On the other hand, a very fast and effective endovascular solution does not allow intervention at the site where important head-and-neck branches of the aorta arise. A hybrid procedure in this case means a surgical intervention that creates a safe zone for fixing the stent graft. Hybrid procedures at this site are divided into three types. Type I is feasible when the ascending aorta is of good quality and unaffected. The origins of the head-and-neck branches can then be relocated, using a prosthesis, more proximally onto the ascending aorta, allowing the stent graft to be fixed at the level of the aortic arch and to cover the entire affected segment of the aorta. If the ascending aorta does not offer sufficiently healthy, unaffected tissue, a type II hybrid procedure must be performed. The ascending aorta is replaced with a linear prosthesis or conduit to the required extent, with the distal anastomosis sutured at a site where aneurysmal dilation is still present. The origins of the head-and-neck branches are then again relocated, using a

prosthesis, into the safe material of the graft. In a second step, a stent graft can then be implanted, with its proximal fixation into the distal part of the linear prosthesis placed by the cardiac surgeon. If the site of the peripheral anastomosis does not offer tissue adequate for a safe anastomosis, a type III hybrid procedure must be performed, representing the elephant trunk or frozen elephant trunk technique. These techniques are described in more detail in the chapter Surgical Interventions on the Thoracic Aorta and Aortic Arch (Figure 34).

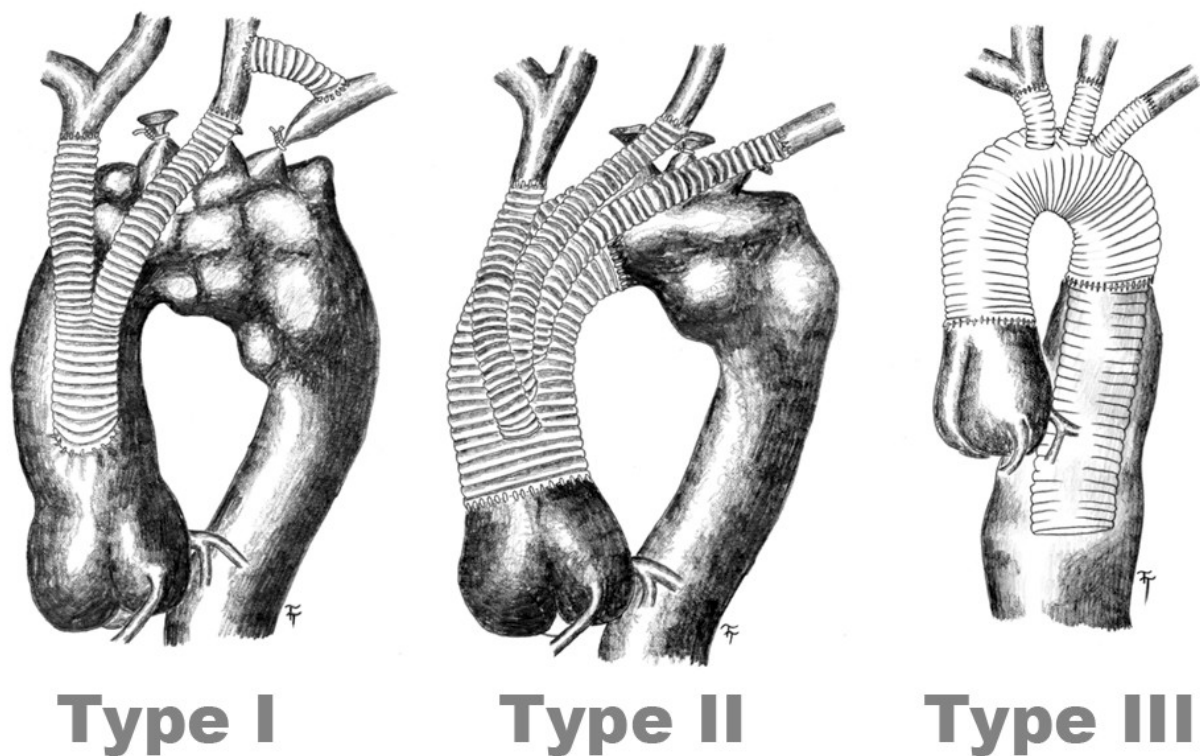


Figure 34 Types of hybrid procedures on the aortic arch.

Hybrid procedures thus combine the benefits of two interventional strategies in a single patient, offering lower periprocedural morbidity and mortality, with excellent postoperative results.

14 References

- Baumgartner H et al.: 2020 ESC Guidelines for the management of adult congenital heart disease: The Task Force for the management of adult congenital heart disease of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC), International Society for Adult Congenital Heart Disease (ISACHD). *European Heart Journal*, 2021, 42(6), 563-645
- Byrne R.A. et al.: 2023 ESC Guidelines for the management of acute coronary syndromes. *European Heart Journal*, 2023, 44(38), 3720-3826
- Chrysakis N. et al.: Heart Transplantation. *Journal of Clinical Medicine*, 2024, 13(2), 558
- Cohn L.A. et al.: *Cardiac Surgery in the Adult* Fifth Edition. New York: McGraw-Hill, 2017. ISBN 007163312X.
- Cohn W.E. et al.: Total artificial hearts: past, present, and future. *Nature Reviews Cardiology*, 12 609–617, 2015.
- Cook J.A. et al.: The total artificial heart. *Journal of Thoracic Disease*, 7(12) 2172–2180, 2015.
- Gaudino M. et al.: Expert systematic review on the choice of conduits for coronary artery bypass grafting: endorsed by the European Association for Cardio-Thoracic Surgery (EACTS) and The Society of Thoracic Surgeons (STS). *European Journal of Cardio-Thoracic Surgery*, 2023, 64(2), ezad163
- Gega A. et al.: Straight deep hypothermic arrest: experience in 394 patients supports its effectiveness as a sole means of brain preservation. *The Annals of Thoracic Surgery*, 84(3) 759–766, 2007
- Hulman M.: *Mechanické podporné systémy a totálna náhrada srdca v kardiochirurgii dospelého veku* [Mechanical support systems and total heart replacement in adult cardiac surgery]. Bratislava: Berlina s.r.o, 2020. ISBN 978-80-972674-9-0.
- Jakub P. et al.: Modified Del Nido Cardioplegia with a 1:4 Crystalloid-to-Blood Ratio Versus Blood-Based St. Thomas Cardioplegia in Isolated Aortic Valve Replacement. *Journal of Cardiovascular Development and Disease*, 13(6) 263, 2026.
- Joglar J.A. et al.: 2023 ACC/AHA/ACCP/HRS Guideline for the Diagnosis and Management of Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*, 2024, 149(1), e1-e156
- Kakoudaki T. et al.: Heart Transplantation: Is It Still the Gold Standard? *British Journal of Hospital Medicine*, 2025, 86(12), 1-18
- Lee J. et al.: Comparative analysis of anatomic changes and feasibility of valve-in-valve transcatheter aortic valve replacement between Konno and Y-incision aortic root enlargement. *JTCVS Tech*. 2025. 30, 32–42.
- Mazzolai L. et al.: 2024 ESC Guidelines for the management of peripheral arterial and aortic diseases. *European Heart Journal*, 2024, 45(36), 3538-3700
- McCullough J.N. et al.: Cerebral metabolic suppression during hypothermic circulatory arrest in humans. *The Annals of Thoracic Surgery*, 67(6) 1895–1899, 1999.
- Mills N.L. et al.: Fifth Universal Definition of Myocardial Infarction (2026): On Behalf of the Joint European Society of Cardiology/American College of Cardiology/American Heart Association/World Heart Federation Task Force. *Circulation*, 2026, 154, e00-e00.
- Moscarelli M. et al.: Minimal Access Versus Sternotomy for Complex Mitral Valve Repair: A Meta-Analysis. *Ann Thorac Surg*. 2020. 109(3),737-744
- Muri J., Grussmannová A.: *Klinická anatomie hrudníku* [Clinical anatomy of the thorax]. Ostrava: Ostravská univerzita, 2019. ISBN 9788075990754.
- Neumann F.J. et al.: 2018 ESC/EACTS Guidelines on myocardial revascularization. *European Heart Journal*, 40(2) 87–165
- Pirk J. et al.: *Kardiochirurgie* [Cardiac Surgery]. Praha: Maxdorf, 2019. ISBN 9788073455682.

- Praz F. et al.: 2025 ESC/EACTS Guidelines for the management of valvular heart disease: Developed by the task force for the management of valvular heart disease of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). *European Heart Journal*, 2025, 46(44), 4635-4736
- Rao S.V. et al.: 2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Journal of the American College of Cardiology*, 2025, 85(22), 2135-2237.
- Sabol F.: Aortic arch and supraaortic branches, In. Sihotský V.: *Vascular surgery*. New York. Nova science publishers, 2020. ISBN: 978-1-53616-454-1.
- Samak M. et al.: Past and Present of Total Artificial Heart Therapy: A Success Story. *Medical Science Monitor Basic Research*, 21 183–190, 2015.
- Srivastava P.K., Kittleson M.M.: Modern advances in heart transplantation. *Progress in Cardiovascular Diseases*, 2024, 82, 147-156
- Teuteberg J.J. et al.: Implant strategies change over time and impact outcomes: insights from the INTERMACS (Interagency Registry for Mechanically Assisted Circulatory Support). *JACC: Heart Failure*, 1(5) 369–378, 2013
- Thygesen K. et al.: Fourth universal definition of myocardial infarction (2018). *European Heart Journal*, 40(3), 237–269
- Toporcer T., Kolesár A.: *Chirurgia srdca pre medikov [Cardiac Surgery for Medical Students]*. Košice: Univerzita Pavla Jozefa Šafárika v Košiciach, 2023.
- Toporcer, T.: Descendent thoracoabdominal aorta, In. Sihotský V.: *Vascular surgery*. New York. Nova science publishers, 2020. ISBN: 978-1-53616-454-1.
- Toporcer T. et al.: Isolated Unroofed Coronary Sinus without Persistent Left Superior Vena Cava. *Cor et Vasa*. 2020, 62(5). 513-516
- Toporcer T., Kolesár A.: Odporúčanie pre liečbu chlopňových chýb [Recommendation for the treatment of valvular heart disease] [electronic resource] *Standard Procedures in Healthcare*. (available at: <https://www.health.gov.sk/?Standardne-Postupy-V-Zdravotnictve>.)
- Toporcer T., Kolesár A.: Odporúčanie pre revaskularizáciu myokardu [Recommendation for myocardial revascularization] [electronic resource] *Standard Procedures in Healthcare*. (available at: <https://www.health.gov.sk/?Standardne-Postupy-V-Zdravotnictve>.)
- Torre D.E., Pirri C.: Harlequin Syndrome in Venoarterial ECMO and ECPPELLA: When ECMO and Native or Impella Circulations Collide—A Comprehensive Review. *Reviews in Cardiovascular Medicine*, 26(8) 39992, 2025.
- Vahanian A. et al.: 2021 ESC/EACTS Guidelines for the management of valvular heart disease: Developed by the Task Force for the management of valvular heart disease of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). *European Heart Journal*, 2022, 43(7), 561-632
- Van Gelder I.C. et al.: 2024 ESC Guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). *European Heart Journal*, 2024, 45(36), 3314-3414
- Vrints C. et al.: 2024 ESC Guidelines for the management of chronic coronary syndromes. *European Heart Journal*, 2024, 45(36), 3415-3537

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Lecture Notes

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