Complex open and endovascular reconstructions of aorta and its branches

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COMPLEX OPEN AND ENDOVASCULAR RECONSTRUCTIONS OF AORTA AND ITS BRANCHES

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Abbreviations

AAA - Abdominal Aortic Aneurysm

AI - Augmentation Index

CAD - Coronary Artery Disease

CAVI - Cardioankle Vascular Index

CHF - Congestive Heart Failure

CSF - Cerebrospinal Fluid

CVD - Cardiovascular Disease

DAP - Distal Aortic Perfusion

DHCA - Deep Hypothermic Circulatory Arrest

EVAR - Endovascular Aortic Repair

GFR - Glomerular Filtration Rate

HTN - Hypertension

MMPs - Metalloproteinases

MSCT - Multi-Slice Computed Tomography

PET - Positron Emission Tomography

PGS - Perigraft Seroma

PWV - Pulse Wave Velocity

Re - Reynolds Number

SPECT - Single Photon Emission Computed Tomography

SVS - Society of Vascular Surgery

TAA Thoracic Aortic Aneurysm

TAAA - Thoracoabdominal Aortic Aneurysms

TEVAR - Thoracic Endovascular Aortic Repair

VSMC - Vascular Smooth Muscle Cell

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1. SUMMARY

Title: Complex open and endovascular reconstructions of aorta and its branches

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The aorta is the largest blood vessel in the human body, responsible for supplying blood for the systemic circulation and enabling the blood supply to the organs via its branches. Aortic diseases are of major global concern since the history of humankind, though, their significance is even more pronounced nowadays in developed countries, due to the additive and synergistic effects of natural physiologic factors combined with various risk factors that emerged with the transition to the current sedentary western lifestyle (smoking, western diet, less physical activity, etc.) worldwide. The nature of the aging process together with those behaviors might, in turn, cause significant pathologic changes in the aortic anatomy and histology, facilitating the development of various pathological processes, which emphasizes the need for research in the field of the aorta and aortic branches reconstruction.

The pathophysiological processes causing aortic disease can be interrupted or decelerated via the use of medical treatment modalities and lifestyle changes, though, research and practical experience in the field teach us that, past a certain point, surgical or endovascular reconstruction of the aorta will warrant better results in managing such patients.

Aorta and its branches can be reconstructed surgically via the established open surgical approach, or via the relatively modern endovascular and hybrid approaches. The open surgical approach allows for excellent durability and a large field of view compared to the endovascular approach which has the advantages of decreased trauma, faster recovery with lower early complication rate, and shorter postoperative hospital stay.

Keywords: open, endovascular, aorta, reconstruction, surgical.

2. SAŽETAK

Naslov: Složene otvorene i endovaskularne rekonstrukcije aorte i njezinih ogranaka

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Aorta je najveća krvna žila u ljudskom tijelu, odgovorna za opskrbu sistemske cirkulacije, koja svojim ograncima omogućuje opskrbu tjelesnih organa krvlju. Bolesti aorte predstavljaju veliku globalni problem od povijesti ljudske vrste, no njihov je značaj još izraženiji u razvijenim zemljama današnjice zbog aditivnih i sinergijskih učinaka prirodnih fizioloških čimbenika u kombinaciji s različitim čimbenicima rizika koji su se pojavili s prijelazom na sadašnji sjedilački zapadnjački stil života (pušenje, zapadnjačka prehrana, nedostatak tjelesne aktivnosti itd.) diljem svijeta. Priroda procesa starenja zajedno sa suvremenim stilom života može zauzvrat uzrokovati značajne patološke promjene u anatomiji i histologiji aorte i prouzročiti razvoj različitih patoloških procesa, što uzrokuje potrebu za istraživanjima u području rekonstrukcije aorte i njenih ogranaka.

Patofiziološki procesi koji uzrokuju bolesti aorte mogu se prekinuti ili usporiti primjenom medicinskih modaliteta liječenja i modifikacijom stila života, međutim, istraživanja i iskustvo u praksi pokazuje da će, progresijom bolesti, nakon određene točke, kirurška ili endovaskularna rekonstrukcija aorte jamčiti bolje rezultate u liječenju takvih bolesnika.

Aorta i njezine grane mogu se kirurški rekonstruirati klasičnim otvorenim kirurškim pristupom ili relativno modernim endovaskularnim i hibridnim pristupima. Otvoreni kirurški pristup omogućuje izvrsnu izdržljivost i veliko vidno polje u usporedbi s endovaskularnim pristupom, koji ima za prednost manju traumu, manje ranih poslijeoperacijskih komplikacija, brži oporavak i kraće trajanje hospitalizacije nakon operacije.

Ključne riječi: otvorena, endovaskularna, aorta, rekonstrukcija, kirurška.

3. INTRODUCTION

Aortic diseases are relatively common in the general population, for which, there aren’t many conservative medical solutions (unlike other diseases). Surgical aortic and aortic branch repair and reconstruction is the solution for many diseases as such.

This review paper will be discussing most of the essential anatomy, major histological concepts, relevant physiology (hemodynamics, endothelial factors, and regulation), and the common pathologies and diseases for which aortic repair is indicated allowing for a basic understanding of the concepts reviewed.

The methods of aortic reconstruction, including the open surgical approach, the complete endovascular approach, and the combined “hybrid” approach will be discussed and compared to each other, considering their advantages, disadvantages, indications, contraindications, complications, and particular scenarios suitable for each disease.

This paper is made in an attempt to solidify and back up the common knowledge regarding the topic of aortic repair and upgrade it with the most updated articles and guidelines.

Limitations: Specific devices, instruments, and subtypes of the aforementioned procedural approaches are beyond the scope of this review paper and won't be discussed here.

4. AORTA – BASIC ANATOMY, HISTOLOGY, AND PHYSIOLOGY

4.1. BASIC ANATOMY AND HISTOLOGY

4.1.1 Basic Anatomy

The aorta is the largest blood vessel in the human body and originates at the left ventricle of the heart, from which it transports oxygenated blood throughout the body to the systemic circulation. Aortic size is proportional to an individual's height and weight and is typically the largest as it leaves the heart and the smallest at its end. It is the first artery by chronological order and is responsible for transporting nutrient-rich blood to the systemic circulation following ejection from the heart's left ventricle. The aorta extends from the aortic valve, allowing pressure to build within the left ventricle during ventricular systole and terminates at the proximal iliac bifurcation at the L4 vertebral level.

The vessel can be divided into various segments depending on course and location. The thoracic aorta consists of ascending, aortic arch, and descending aorta. The descending thoracic aorta passes through the diaphragm's aortic hiatus at the T12 vertebral level, at which point it continues as the abdominal aorta. The abdominal aorta terminates as it bifurcates into common iliac arteries, providing arterial supply to the pelvis and lower limbs.

Once the pressure in the ventricle exceeds the pressure in the aorta, the aortic valve opens, allowing blood to flow into the ascending aorta. At the end of systole, the pressure within the ventricle drops below the pressure within the aorta resulting in the closure of the aortic valve. The aortic valve typically processes three leaflets; however, it is congenital, with two leaflets in 1% of the population (1,2).

The ascending aorta is approximately 5 cm in length and 2.5 cm in diameter, originating from the left ventricle and ending at the beginning of the aortic arch. The coronary arteries branch from the ascending aorta and supply oxygenated blood to the cardiac tissue.

The aortic arch begins at the level of the upper border of the 2nd sternocostal articulation, at the sternal angle, where the body of the sternum articulates with the manubrium. From there, the aortic arch runs upward, anterior to the trachea, and then curves inferiorly and to the left, running along the left side of the body of the fourth thoracic vertebra. At this point, it becomes the descending aorta. Branches from the aortic arch supply oxygenated blood to the head, neck, and arms.

Within the thoracic cavity is the upper portion of the descending aorta, referred to as the thoracic aorta, which begins on the left side of the inferior border of the body of the T4 vertebrae. Branches of the thoracic aorta supply blood to the ribs and other structures of the chest.

The abdominal aorta is the next section of the descending aorta, which begins at the aortic hiatus in the diaphragm at the level of the T12 vertebra and is approximately 13 cm in length. Branches from the abdominal aorta provide blood flow to most major abdominal organs. The descending aorta ends as a bifurcation into the common iliac arteries at the level of the L4 vertebra, supplying oxygenated blood to the pelvis and lower extremities (3–5).

4.1.2 Basic Histology

The aortic wall is comprised of three distinct layers at a microscopic level: the tunica intima, the tunica media, and the tunica adventitia. The innermost layer of the aortic wall, known as the "tunica intima," is the surface of the aortic wall, which faces the arterial lumen. It is composed of an endothelial cell monolayer on a basement membrane and supported by an internal elastic lamina at the border to the tunica media. The primary role of the tunica intima is to provide a smooth, non-thrombogenic surface for blood flow.

The middle layer of the aortic wall – the "tunica media," is made primarily of smooth muscle cells and elastic tissue. Transverse sections of the aorta display a lamellar structure of the tunica media with layers of smooth muscle separated by a considerable quantity of layered elastic tissue with elastic fibers (elastic lamellae) and some connective tissue connecting the lamellae to the inner membrane. This elastic, muscular organization allows the aorta to expand and contract with the pulsatile blood flow generated by the heart.

The outermost layer of the aortic wall – the "tunica adventitia," consists of collagen fibers, mast cells, and fibroblasts which provide additional support and structure to the vessel (6). The luminal half of the wall receives its blood supply via diffusion from the blood within the lumen. The outer half of the aortic wall receives its blood supply via small penetrating vessels called the vasa vasorum (3–5).

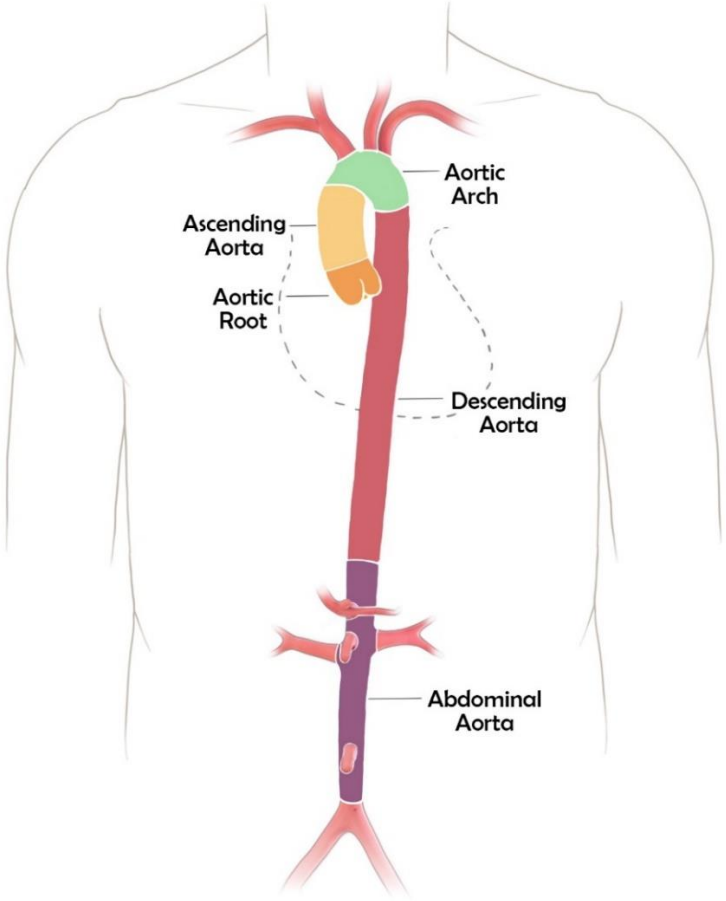

Figure 1. Basic anatomy of the aorta.

(Original artwork)

4.2. HEMODYNAMICS

The risk for cardiovascular disease (CVD) progression is highly associated with changes in hemodynamics. Genetic studies have demonstrated the existence of mechanosensitive genes causing CVD. Turbulent blood flow changes the vessel wall shear stress, activating mechanoreceptors, ultimately inducing the progression of degenerative processes, leading to CVD. The likelihood of turbulent blood flow increases with the Reynolds number (Re). It is a dimensionless quantity in fluid mechanics which is proportional to blood density, blood flow velocity, and vessel diameter; and inversely proportional to blood viscosity. Reynolds number greater than 2000 is critical for the occurrence of turbulent blood flow.

Since abnormal blood flow may increase CVD risk, there were studies investigating the phenomenon of CVD development due to hemodynamic changes using data from cardiac magnetic resonance imaging of the ascending aorta. The studies showed that hemodynamic

factors, including the Re number, are well correlated to CVD and have the potential for future novel intervention strategies in patients with chaotic blood flow and vascular abnormalities (7).

It has been established that hypercholesterolemia and oxidative stress, combined with low wall shear stress (WSS) and nonlaminar blood flow (turbulent blood flow), produce the right environment for creating focally distributed atherosclerotic lesions in post stenotic segments of blood vessels. In the presence of hypercholesterolemia and oxidative stress, in a normotensive segment with laminar blood flow and normal WSS, only diffuse fatty streaks could be identified with no sign of atherosclerotic lesions (8).

5. PATHOLOGICAL PROCESSES AND RISK FACTORS

5.1. AGING

Aging is the gradual accumulation of changes over time that are linked to or cause the increased susceptibility to disease and death (9).

Age-associated anatomical and pathological alterations of the aorta impact hemodynamics. These changes have a remarkable effect on the cardiovascular system, allowing for an increase in CVD, including atherosclerosis, coronary artery disease (CAD), hypertension (HTN), congestive heart failure (CHF), aortic aneurysms, and stroke (5,10).

The diameter of the ascending aorta enlarges steadily with increasing age. Also, the difference in dilation between systole and diastole decreases with age. Finally, the length of the aorta increases with age. The dilation and stiffening of the aortic wall and the elongation of the aorta result in increased blood pressure and heart workload, which gives rise to many pathologies. Age-related physiological changes in the aorta are associated with a progressive decline in the elastic properties of the aortic wall. The vascular aging process begins in childhood but is not noticeable until middle age. The mechanical principle of "fatigue" predicts that the elastic lamella within the tunica media of the vessel will break after approximately 1 billion cycles, with about an 8% change in vessel diameter per billion cycles, which occurs at around 40 years of age. The tunica media stiffens gradually due to elastin fiber fracturing and collagenous remodeling. Following the loss of functional interlaminar elastic fibers, the elastic lamellae become more separated and filled with proteoglycans and altered collagen, which changes from thin, wavy fibers to thicker and more linear in arrangement with age. As a result, stress is transmitted to collagen in the vessel wall.

Additionally, as people get older, there are fewer smooth muscle cells in the tunica media, which are responsible for elastin synthesis in the aorta. Furthermore, while tropoelastin (a molecule responsible for forming the protein elastin) is expressed throughout life, its expression decreases by 50% every decade, indicating a decrease in elastin regenerative potential. The stiffening of the vessel wall raises aortic impedance and pulse pressure. Increasing wall stiffness and peripheral wave reflection velocity causes an increase in systolic and pulse pressure by 40 mmHg while decreasing diastolic pressure. Pulse wave velocity (PWV), cardioankle vascular index (CAVI), and augmentation index (AI) measurements have all shown a positive correlation between increasing age and arterial stiffness (5,11,12).

The relative ratio of collagen and elastin within the vascular wall determines aortic compliance. The ratio of the two components in normal tissue is kept relatively constant by a gradual production and degradation process. The collagenolytic and elastolytic properties of catabolic matrix metalloproteinases (MMPs) regulate collagen and elastin. Multiple factors influence the activity of these MMPs, including increased gene expression, post-translational activation via cleavage of pro-MMP protein, MMP–MMP interactions, and plasmin, thrombin, and reactive oxygen species (ROS). A change in the regulation of this process causes an increase in collagen production within the artery and a decrease in function. This change results in a considerably thickened intima and media, smooth muscle cell proliferation, and hypertrophy (5,12–14).

The concept of Calcification Age has previously been identified as a nonmodifiable risk factor for atherosclerosis, but the impact of age on disease progression is unknown. Although coronary calcium is linked to aortic calcium levels, and levels are found to rise with age, calcium levels may not affect atherosclerotic wall burden on their own. An MRI study of healthy older adults found no link between atherosclerotic wall burden and aortic or coronary calcium levels or progression of coronary calcium. This study suggests that age may be a risk factor for atherosclerosis progression by its pathophysiologic pathway (5,15,16).

Other aging-related changes, such as intimal thickening, have been observed in the aortic valve. According to studies, the aortic valve leaflet thickness increases with age, as does the tunica intima of the aorta. The thickness of the tunica media remains constant with age, implying that increasing aortic wall diameter is caused by tunica intima thickening. The regular endothelial monolayer lining the aortic lumen shows irregularly shaped cells as it ages (5,17–19).

Another process worth mentioning is the migration of smooth muscle cells from the tunica media, known as “vascular smooth muscle cell (VSMC) migration,” which causes endothelial thickening. VSMC migration occurs in response to vascular injury and during atherosclerosis development. Intimal thickening also decreases vessel elasticity and lumen diameter. These changes are associated with the general rise in systolic blood pressure that comes with age (5,20,21).

5.2. ATHEROSCLEROSIS

Atherosclerosis is a pathophysiologic process involving endothelial cell dysfunction, and it develops most rapidly in areas with bends and branch points that encourage turbulent blood flow. The lower abdominal aorta and the coronary arteries are the vascular beds most susceptible to atherosclerosis. Atherosclerotic lesions (e.g., intimal thickening, fatty streaks) occur in these vessels the earliest (as early as the second decade of life), with the highest overall atherosclerotic burden. Atherosclerosis is also a major contributing factor in developing aortic pathologies such as aortic aneurysms (8,22–24).

5.3. AORTIC ANEURYSM

The term “Aneurysm” (derived from Greek) is defined as a permanent, irreversible, localized widening or dilatation of a vessel. Aneurysmal change of the aorta is termed “aortic aneurysm,” defined as the widening and dilatation of the aorta to a diameter of 3 cm or more, which can occur throughout the body. An aortic aneurysm can present multiple risks, and the most common are rupture, embolism, thrombosis, and more. Generally speaking, aneurysmal dilatation of the aorta can be classified anatomically into abdominal aortic or thoracic aortic aneurysms. Aneurysms could be further classified as true aneurysms, affecting all three layers of the blood vessel; and false aneurysms (pseudoaneurysms), which are not enveloped by all three vessel wall layers. Moreover, pseudoaneurysms usually represent a defect of all three layers of the vessel wall with extravasation and hematoma, walled off by surrounding tissues (25,26).

The critical size of 6 cm is accepted as a mechanical and clinical threshold for complications. The aorta loses its natural elasticity at this diameter and effectively becomes a stiff tube. The stress created in the wall of a 6 cm aorta can reach or exceed the maximum tensile strength of aortic tissue at a blood pressure of 200 mmHg, which can be easily achieved through strenuous physical activity or emotional distress (26–28).

Medical therapy usually focuses on controlling blood pressure (beta-blockers, ACE inhibitors/ARBs) and dyslipidemia (statins); other experimental drugs for this indication are unproven and beyond the scope of this paper (27).

Surgical therapy is indicated in patients with symptomatic aortic aneurysms or patients with an aortic diameter of > 5.5 cm.

5.3.1. Abdominal Aortic Aneurysm (AAA).

An abdominal aortic aneurysm (AAA) is defined as a localized dilation of an abdominal aorta with a diameter of 3 cm or more. AAAs can be further classified into infrarenal, juxtarenal, pararenal, and suprarenal aortic aneurysms, depending on the relationship to renal arteries. The aneurysms extending upwards to the level of the diaphragm are, however, classified as Crawford Type IV thoracoabdominal aortic aneurysms (TAAA), due to a similar surgical approach and management as for the other types of TAAA's (25,29).

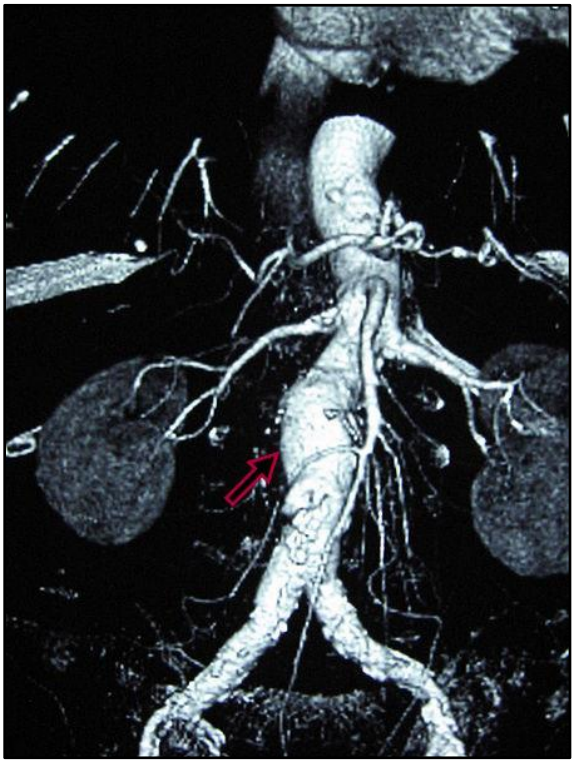

Figure 2. AAA, shown on the Multi-Slice Computed Tomography (MSCT), surface rendering (the patent lumen is filled with contrast, while the real aneurysm size can be deduced by noticing the wall calcifications producing an egg-shell semilunar appearance, to the left of the visible lumen).

5.3.2. Thoracic Aortic Aneurysm.

A thoracic aortic aneurysm is defined as a localized dilation of the thoracic aorta with a diameter greater than 150% of its normal diameter for a given segment. For the thoracic aorta, a diameter greater than 4.5 cm or more is considered an aneurysmal dilatation (30). The thoracic aorta grows slowly at approximately 0.12 cm/year, and the descending aorta grows faster than the ascending aorta (27).

To avoid the morbidity and mortality associated with aneurysm rupture/dissection, prophylactic repair of thoracic aortic aneurysm (TAA) with suitable diameter/expansion criteria is advised. The 5-year survival rate after elective open surgical repair of a TAA in contemporary series is approximately 85%. Emergency surgery for TAA complications has much worse outcomes, with a 5-year survival of 37%. When discussing aortic aneurysms, it is essential to elaborate on aortic dissections and distinguish between acute and chronic dissections and the relevant etiologies. Aortic dissections are more common in younger people and have a higher rate of early rupture than arteriosclerotic aneurysms. Aortic dissections are more commonly found in the ascending aorta and cause symptoms (31).

In contrast, atherosclerotic aneurysms are more commonly found in the descending aorta and are often asymptomatic unless they are relatively large. Acute dissection in the ascending aorta necessitates rapid surgical intervention. Because of the established risk of late rupture, patients with chronic aortic dissection should be considered for surgical repair. Furthermore, due to the high risk of rupture, atherosclerotic aneurysms should be evaluated for elective surgery. The operative mortality rate for elective resection of atherosclerotic aneurysms is lower than the estimated rate of rupture (32).

5.4. AORTIC DISSECTION

Aortic dissection describes a spontaneous tear of the intima of the aorta and the subsequent propagation of that tear along the anatomic plane within its media. Without further propagation, a penetrating aortic ulcer or intramural hematoma may form (26).

Aortic dissection is one of the most common catastrophic aortic complications, occurring 2-3 times more frequently than abdominal aortic rupture. When left untreated, around 33% of patients with ascending aortic dissection die within the first 24 hours and 50% within the first 48 hours, while the 2-week death rate surpasses 75% (33).

5.4.1 Classification of Aortic Dissection

Aortic dissection and other aortic syndromes are classified based on the anatomic site of the intimal tear, the duration of time from its occurrence, and clinical criteria such as the presence or lack of symptoms and whether the condition progresses. The Stanford classification and the DeBakey classification are two commonly used anatomical classification systems (Figure 3); However, different anatomical classification systems have been created by the Society for Vascular Surgery (SVS) and the Society for Thoracic Surgery (STS) due to the limited ability

of the abovementioned classification systems to denote the specifics regarding the precise extent of the dissection and the ambiguity regarding the involvement of the aortic arch.

In the Stanford classification system (Figure 3, upper part), aortic dissections involving the ascending aorta and/or the aortic arch (between the heart and the origin of the left subclavian artery) are classified as "Stanford Type A," regardless of the location of the main intimal tear, and all other dissections (peripheral to the origin of the left subclavian artery) are classified as "Stanford Type B".

The DeBakey classification system (Figure 3, lower part) specifies the type of dissection based not only on the origin of the tear but also on the extent of the dissection. DeBakey Type I involves the ascending aorta, arch, and descending thoracic aorta and may progress to involve the abdominal aorta. DeBakey Type II is confined to the ascending aorta. DeBakey Type IIIa involves the descending thoracic aorta distal to the left subclavian artery and proximal to the celiac artery. DeBakey Type IIIb dissection involves the thoracic and abdominal aorta distal to the left subclavian artery.

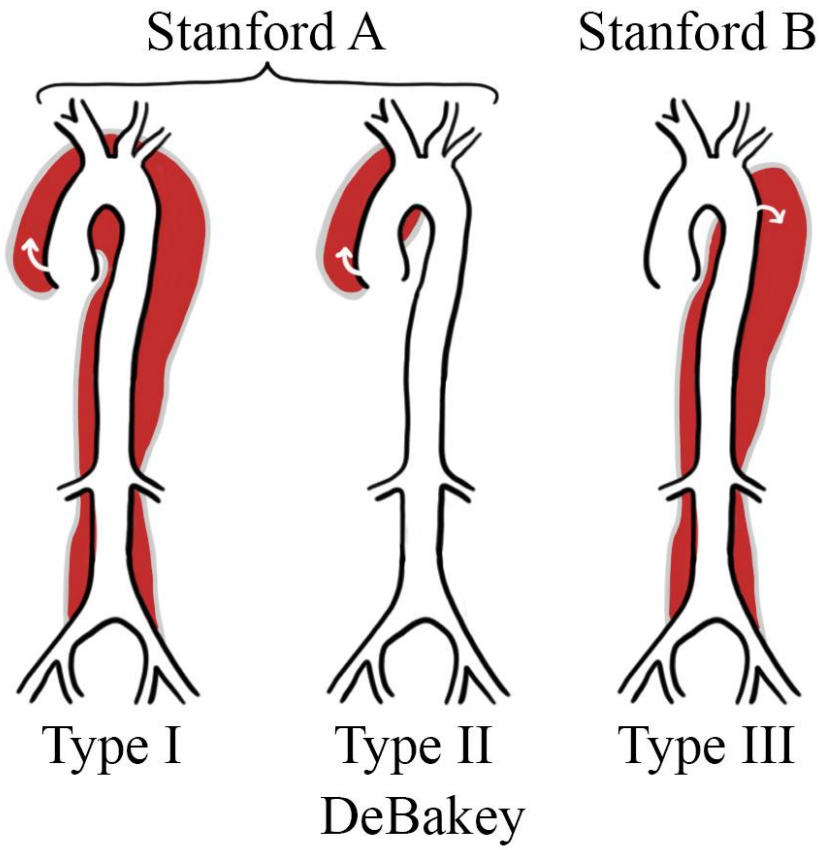

Figure 3. Aortic dissection, "Stanford" and "DeBakey" classification. (Original Artwork).

6. AORTIC RECONSTRUCTION

Surgical interventions of aortic repair can generally be categorized as the more conservative "endovascular techniques" and the classic "open surgical techniques." Each intervention category has its indications, advantages, disadvantages, and complications. Those two categories are suitable for different pathologies and have specific indications based on up-to-date and evidence-based data.

To choose the right approach to the repair, we should consider the etiology (genetically mediated, degenerative), the location, the extent of the pathology, and the age and comorbidities of the patient.

For example, ascending TAA is treated with an open surgical procedure involving cardiopulmonary bypass, and aortic root replacement with coronary artery reimplantation is sometimes required. For the descending TAA, we can use the open surgical approach, endovascular approach, or the combined approach (hybrid repair) (31).

The choice of approach (open vs. endovascular) for descending TAA should consider the etiology (degenerative, genetically mediated, infectious), location in the descending aorta, the extent of the aneurysm, and the patient's expected survival (short-term and long-term), which depends upon the patient's age and medical comorbidities.

6.1. OPEN SURGICAL PROCEDURES

The open surgical approach for aortic reconstruction was described in the 1950s and continued to evolve in Houston, Texas, by Michael Ellis DeBakey. Other significant advances were made with the establishment of modern principles in the treatment of thoracoabdominal aortic aneurysms (TAAA) by Ernest Stanley Crawford and with the use of pulsatile and non-pulsatile flow techniques by Connoly and associates at the University of California (34). The open surgery in the case of AAA is performed via a retroperitoneal or transperitoneal approach, usually via median laparotomy. Surgical approaches to TAA's and TAAA's include thoracotomy, thoracophrenolaparotomy, and thoracoretroperitoneal approach. After gaining access to the relevant aortic section, exclusion of the aneurysmal part is performed by implantation of a graft (straight or bifurcated in the case of AAA) (35), followed by reimplantation of important intercostal, lumbar and visceral arteries.

6.1.1. Advantages, Disadvantages, and Complications

Open surgical aortic repair procedures have excellent durability and a large field of view (compared to the endovascular approach), making them the go-to approach for primary management of ascending thoracic aortic aneurysm and acute ascending thoracic aortic dissection. Open surgery is also frequently employed to address complications of previous endovascular procedures (of the descending aorta predominantly).

Multiorgan protection is believed to be the major challenge in reconstructing the aorta due to the risk of damaging multiple organs, especially the spinal cord, the brain, and the kidneys (34). Techniques such as deep hypothermic circulatory arrest (DHCA), retrograde cerebral perfusion, distal aortic perfusion (DAP), mild passive hypothermia, CSF drainage, and selective antegrade cerebral perfusion are used to overcome such challenges (26,34,36). Tanaka *et al.* provided that the combination of DAP with CSF drainage and mild passive hypothermia can reduce the risk for spinal cord ischemia in Extent I TAAA from 15% to less than 2% and in Extent II TAAA from 33% or 50% (without or with clamp time exceeding 40 minutes, respectively) to less than 4% (34).

Additional possible complications of the open surgical approach are the accumulation of periaortic fluid, known as perigraft seroma (PGS), which can become infected, compress mediastinal structures, and form drainage tracts with the overlying thoracic soft tissue and skin (37).

There are new advances used today allowing better outcomes for the open surgical approach, such as alternative cannulation techniques (femoral, direct ascending, and axillary) and improved surgical techniques (38). Other advances have been made based on the physiologic properties of the body, one example of which is the use of an omental flap to treat PGS occurring after graft placement. This procedure can be done with a 1-stage approach or with a 2-stage approach (39,40). Recently, Yugo *et al.* described the use of fibrin glue to seal the graft and treat the PGS (41). To use the omental flap procedure or the fibrin glue sealing procedure, it is important to rule out infection, since infection usually requires graft replacement with biological material, or major vessel ligation and extraanatomical bypasses. For the exclusion of graft/perigraft infection, one can use a radionuclide scan with labeled leucocytes and MSCT aortography, or PET(SPECT)/CT in a hemodynamically stable patient without sepsis. To be noted, if the PGS is a consequence of technically insufficient vascular anastomosis, the fibrin glue sealing procedure alone is inappropriate and insufficient.

Published results of open surgical repair for ascending aortic aneurysms, aortic arch aneurysms, and descending aortic aneurysms (thoracic and thoracoabdominal) show mortality rates as low as 8% and even lower (5%) in those with normal glomerular filtration rate (GFR). The stroke rate is 3%, and paraplegia/paraparesis rates are 1%-5%, depending on the extent of the procedure (31,42).

In general, elective patients undergoing thoracic aortic replacement stay in the hospital for 5 to 6 days, whereas thoracoabdominal replacement patients stay for 7 to 10 days, assuming no complications.

6.1.2. Indications and techniques

Thoracic Aortic Pathologies:

In ascending TAA, the nature of open surgery differs based on the location and size of the aneurysm. Ascending TAA is repaired via an open surgical procedure, using a median sternotomy and cardiopulmonary bypass with cardioplegia. Aortic root replacement or coronary artery reimplantation are frequently required. A composite graft (i.e., the aortic valve is replaced) or a valve-sparing procedure is usually used to replace the damaged aortic section (43). If there is a bicuspid aortic valve, it can be taken care of while performing the aortic surgery (44). Hypothermic circulatory arrest, usually with antegrade or retrograde cerebral perfusion, is required when the aortic arch is involved. In case of aortic arch involvement, a hemi-arch technique or total arch replacement can be used to repair the aortic arch.

In aortic arch pathologies, open surgery is the gold standard for treating patients with limited comorbidities (45). The mortality rate of open surgery in aortic arch pathologies is 5-20% (46). As mentioned above, there is high morbidity associated with those procedures, such as neurological impairment, renal impairment, and cannulation site injuries.

The open repair of descending TAA is done with a left thoracotomy and does not always require full cardiopulmonary bypass or cardioplegia; however, it is crucial to take measures to protect the spinal cord. Native arterial reimplantation with or without endarterectomy, or bypass grafting, may be required for end-organ revascularization (visceral, renal) (31). Due to a lower rate of complications and better results, thoracic endovascular aortic reconstruction is nowadays the preferred method of treatment for the pathologies involving only descending thoracic aorta, including TAAs.

Abdominal Aortic Pathologies:

The open surgical approach for AAA repair is a safe, widely available technique, effective in preventing aneurysm rupture, and extremely durable (47). This procedure is done under general anesthesia with the proper monitoring of volume status, allowing proper administration of fluids and transfusions. The procedure is performed in a systemic manner, requiring clamping of the aorta, opening the aneurysm, removing thrombus and debris from the aorta, and suturing the synthetic graft to replace the affected section of the aorta.

The open approach is indicated for symptomatic AAA of any size, including symptoms such as abdominal, back, or flank pain, evidence of embolization, and frank rupture. Other indications are aneurysm diameter $\geq$ 5.5 cm in men and $\geq$ 5.0 cm in women, rapidly expanding AAA, AAA associated with another arterial disease (i.e. iliac aneurysm), infected AAA, and complications following endovascular repair necessitating early or late conversion to an open AAA repair (48,49).

Contraindications for open elective repair of AAA exist when the estimated risk of the procedure exceeds the risk of rupture, usually in extremely old and frail patients, with severe cardiac, pulmonary, liver, or kidney disease (26,50,51). Relative contraindications to open surgical repair of AAA include the following: hostile abdomen, obesity, major cardiac or pulmonary comorbidities, and limited life expectancy (26).

6.2. ENDOVASCULAR PROCEDURES

The development of technology that had steadily been accumulating ultimately led to an endovascular revolution in the 1990s. It started with endovascular aneurysm repair (EVAR) and later expanded to thoracic endovascular aneurysm repair (TEVAR). Surgeons have now broadened the indications for treating ascending aortic arch aneurysms and more extensive aneurysms.

6.2.1. Advantages, Disadvantages, and Complications

The endovascular approach allows for minimally invasive procedures, associated with lower morbidity due to the lack of need for hypothermic circulatory arrest and rerouting of aortic

blood flow. Endovascular repair has been linked to lower perioperative mortality as well, though, late complications have been described, including graft migration and aortic rupture.

Worth mentioning in the context of endovascular techniques is the benefit of using intravascular ultrasound (IVUS) in the endovascular treatment of thoracic aortic dissections due to its ability to identify the true and false lumens, allowing for characterization of the dissection and its entry tear and fenestrations (26).

6.2.2. Indications

Thoracic Aortic Pathologies:

A thoracic aortic aneurysm can be repaired endovascularly referred to as thoracic endovascular aortic repair (TEVAR), in which the placement of a modular graft component delivered via the iliac or femoral arteries can be done. The graft placement allows for isolation of the aneurysm and its exclusion from circulation. TEVAR was initially used to provide treatment to patients who were not considered to be surgical candidates, but it is now the preferred technique for treatment due to improved outcomes compared with open thoracic aortic surgery. There are some anatomic criteria required for the placement of endovascular repair procedures. Evidence shows that the most suitable pathology treated with an endovascular approach is a descending TAA which is not involving the abdominal segment (31).

Using endovascular procedures to treat aortic arch aneurysms could be a low-risk alternative to high-risk treatments such as open total aortic arch replacement or hybrid arch repair. The use of custom stent-grafts is possible, including scalloped, fenestrated, and branched grafts. Particularly engineered for use in the aortic arch, both single-branch and multi-branch grafts, as well as fenestrated grafts, are currently being tested in clinical trials for arch pathologies (52–54).

Due to the reduction in perioperative morbidity and mortality compared with the open surgical approach, it is recommended to apply the endovascular approach also to other descending aortic pathologies such as blunt thoracic aortic injury, and aortic dissection (mainly Type B with malperfusion), aortic intramural hematoma, penetrating aortic ulcer and aortoesophageal fistula (55).

Abdominal and Thoracoabdominal Aortic Pathologies:

Although widely used, using endovascular aortic repair (EVAR) for treating TAAA is a challenging process, mainly due to the need to revascularize the visceral branches of the abdominal aorta (56).

Some randomized clinical trials comparing the open approach of repairing AAA with EVAR have found significantly improved short-term (30-day) morbidity and mortality for EVAR. Comparing long-term outcomes of up to 10 years, this advantage over open surgery was lost (56–62). Also, complications specific to endovascular technique were noted, such as endoleaks and graft migrations, and even late aortic ruptures. Also, there are anatomical contraindications for EVAR, summarized in Table 1.

The role of stent-grafting in the treatment of significant thoracoabdominal aortic disease, which requires debranching procedures or specialized grafts (fenestrated, branched endograft), is now being studied (56).

Endovascular techniques can be successfully used in the treatment of ruptured AAA and have some advantages over the open surgical approach. Though this approach is not universally available and the selection of technique is determined by the surgical team and their preference (63–67).

Table 1. Contraindications for EVAR (68).

Absolute contraindications for EVAR	Relative contraindications for EVAR
Proximal neck < 15 mm	Iliac tortuosity
Infrarenal aortic diameter > 26 mm	Proximal neck angulation > 60 °
Thoracoabdominal aortic aneurysm	Bilateral common and internal iliac aneurysms
External iliac diameter < 9 mm or > 16 mm	Iliac artery occlusive disease
Bilateral internal and external iliac aneurysms	
Prosthetic graft material in both groins	
Evidence of retroperitoneal leak on CT	

6.3 THE COMBINED APPROACH

The combined approach for aortic repair, known as the “hybrid repair” involves an open approach, typically to manage the ascending aorta or aortic arch, with an endovascular approach for the descending thoracic aorta (i.e., frozen elephant trunk) (31).

The aortic arch is still the proximal limiting factor for endovascular treatment, though, recent developments of lesser invasive techniques have been presented, one of which is the hybrid repair and debranching technique (45).

A report was published by Kent and colleagues (69) on using a hybrid approach of open surgical and endovascular repair for aneurysms involving the ascending aorta, arch, descending aorta, and thoracoabdominal aorta. This report emphasizes both excision (which reduces the complications associated with patency of the diseased aorta, such as false aneurysm, aneurysmal dilatation, and retrograde dissection) and replacement of the ascending aorta as essential components of this combined approach. A new device invented by Kent and colleagues (69), called the Bavaria graft, had been built to fit this need. Discussing morbidity and mortality in the context of the hybrid approach, the results of the aforementioned study series show a combined hospital and 40-day mortality of 10%, stroke rate which is 5%, and temporary paraplegia or paraparesis of 20%, all of which are concerning findings. Furthermore, the prevalence of endoleak in type I (15%), type II (5%), and graft enfolding is the weakness of any endovascular treatment (5%).

Because of the tendency of the remaining aorta to expand, there are drawbacks to placing a graft or stent graft in the descending thoracic aorta, particularly in patients with aortic dissection or connective tissue disorder. As a result, these findings should be compared to the published results of open surgical repair for ascending aorta, arch, and descending thoracic and thoracoabdominal aortic aneurysms (42).

In the ascending aorta and the aortic arch, significant progress is being made with using the hybrid repair for repairing aneurysmal disease. From the time of the introduction of the endovascular aortic stent graft to treat abdominal aortic pathologies, technological advances extended their use in treating thoracic aortic pathologies. Type B aortic dissection and descending thoracic aneurysms are largely managed using a complete endovascular approach, although sometimes an extra-anatomic bypass or surgical fenestration might be warranted after TEVAR, usually for the treatment of remaining or newly developed malperfusion in type B aortic dissection (36).

One important disadvantage of the hybrid approach involving aortic arch reconstruction is the fact that in most cases cardiopulmonary bypass and deep hypothermia are necessary, while in the total endovascular approach it is possible to avoid such extreme hypothermia and circulatory arrest and the risks associated with it (42).

7. CONCLUSION

Many factors have to be taken into account upon deciding on the right approach for treating complex aortic pathologies. There are a few categories of factors that are still open for discussion.

The patient's factors are factors that can be attributed to the patient, either in the family history (genetics) or in the patient's history (current and previous health conditions). Some of the most important factors are the aortic disease that the patients are suffering from, their age, their comorbidities, their anatomy, their past medical history, past procedures, and also their expectations and will. Patient-centered evidence-based medicine is the future we are looking toward and willing to achieve; as such, it is very important to take into account the abovementioned patient characteristics and tailor the procedure individually.

Another group of factors that need to be considered belongs to the professional/technical part. Under this term, one must take into account the surgeon's capabilities, the institutional and organizational abilities, the adherence to the relevant current guidelines, research data, and medical advances in the field of aortic reconstruction, always applying common sense as well.

After considering all the aforementioned factors, a decision on the best approach for given pathology is made, always weighing risks against benefits. The endovascular approach is preferred in patients with reasonable or limited life expectancy if the anatomy is suitable, especially for isolated reconstruction of descending thoracic aorta or infrarenal abdominal aorta, since it can minimize unwanted morbidity and mortality associated with the procedure of medically induced hypothermia and circulatory arrest, additionally, the hospitalization (recovery) times are shorter and the long-term results are comparable to, and in some cases even better than (70–74) the open surgical approach. This recommendation is in agreement with guidelines from the Society for Vascular Surgery (SVS) guidelines for the care of a patient with an abdominal aortic aneurysm (AAA) (74). However, according to the European Society for Vascular Surgery guidelines (75) in fit patients with long life expectancy, open abdominal aortic aneurysm repair should be considered the preferred treatment modality, as it is more durable in the long term, with significantly fewer late complications.

The open surgical approach is made via a transperitoneal or retroperitoneal approach, in most cases via median laparotomy. After gaining access to the relevant aortic section,

exclusion of the aneurysmal part is performed by implantation of a graft (straight or bifurcated in the case of AAA). This procedure should be used when there are anatomical, instrumental, or any other contraindications which are not allowing us to continue with the endovascular approach. The surgical approach is preferred in many cases of repairing complications of a previously made endovascular procedure or other primary indications discussed in the "methods" section of this review paper. A major drawback of the open approach and the hybrid approach is the risk of ischemic injury to the vital body organs (spinal cord, brain, etc..). the open approach has varying morbidity and mortality rates, depending on the indication, and other parameters (35).

Hybrid approaches are mostly used for the pathology of the aortic arch, or the suprarenal aorta since the obliteration of the supra-aortic trunks or visceral aortic branches by stent grafts necessitates their revascularization with extra-anatomic bypasses (a procedure called "debranching"). Open surgery, frequently done in deep hypothermic circulatory arrest with extracorporeal circulation or by utilizing distal aortic perfusion, is still the "golden" standard for the most complex thoracoabdominal aortic aneurysms. However, recent advancements in endovascular techniques and materials (fenestrated and branched endografts, sometimes even combined with mechanical aortic valves), as well as the accumulation of experience in the hands of the interventionists, might soon make the total endovascular aortic repair more widely used globally.

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9. REFERENCES

1. KOLETSKY S. CONGENITAL BICUSPID AORTIC VALVES. Arch Intern Med. 1941 Jan 1;67(1):129–56.

2. Hoffman M, MD. The Aorta (Human Anatomy): Picture, Function, Location, and Conditions [Internet]. WebMD. [cited 2022 May 10]. Available from: https://www.webmd.com/heart/picture-of-the-aorta

3. White HJ, Bordes S, Borger J. Anatomy, Abdomen and Pelvis, Aorta. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 [cited 2022 Apr 3]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK537319/

4. Kelley JD, Kerndt CC, Ashurst JV. Anatomy, Thorax, Aortic Arch. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 [cited 2022 May 10]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK499911/

5. Collins JA, Munoz JV, Patel TR, Loukas M, Tubbs RS. The anatomy of the aging aorta. Clin Anat N Y N. 2014 Apr;27(3):463–6.

6. Fritze O, Romero B, Schleicher M, Jacob MP, Oh DY, Starcher B, et al. Age-related changes in the elastic tissue of the human aorta. J Vasc Res. 2012;49(1):77–86.

7. McGurk KA, Owen B, Watson WD, Nethononda RM, Cordell HJ, Farrall M, et al. Heritability of haemodynamics in the ascending aorta. Sci Rep. 2020 Sep 1;10(1):14356.

8. Prado CM, Ramos SG, Elias J, Rossi MA. Turbulent blood flow plays an essential localizing role in the development of atherosclerotic lesions in experimentally induced hypercholesterolaemia in rats. Int J Exp Pathol. 2008 Feb;89(1):72–80.

9. Harman D. The aging process. Proc Natl Acad Sci U S A. 1981 Nov;78(11):7124–8.

10. North BJ, Sinclair DA. The Intersection Between Aging and Cardiovascular Disease. Circ Res. 2012 Apr 13;110(8):1097–108.

11. Cikach FS, Koch CD, Mead TJ, Galatioto J, Willard BB, Emerton KB, et al. Massive aggrecan and versican accumulation in thoracic aortic aneurysm and dissection. JCI Insight. 3(5):e97167.

12. Extracellular matrix, regional heterogeneity of the aorta, and aortic aneurysm | Experimental & Molecular Medicine [Internet]. [cited 2022 May 11]. Available from: https://www.nature.com/articles/s12276-019-0286-3

13. Jabłońska-Trypuć A, Matejczyk M, Rosochacki S. Matrix metalloproteinases (MMPs), the main extracellular matrix (ECM) enzymes in collagen degradation, as a target for anticancer drugs. J Enzyme Inhib Med Chem. 2016;31(sup1):177–83.

14. Frantz C, Stewart KM, Weaver VM. The extracellular matrix at a glance. J Cell Sci. 2010 Dec 15;123(24):4195–200.

15. Kälsch H, Lehmann N, Moebus S, Hoffmann B, Stang A, Jöckel K, et al. Aortic Calcification Onset and Progression: Association With the Development of Coronary Atherosclerosis. J Am Heart Assoc. 6(4):e005093.

16. Leopold JA. Vascular Calcification: An Age-Old Problem of Old Age. Circulation. 2013 Jun 18;127(24):2380–2.

17. Kozakova M, Schmidt-Truksäss A. Chapter 14 - Age-Induced Endothelial Dysfunction and Intima–Media Thickening. In: Nilsson PM, Olsen MH, Laurent S, editors. Early Vascular Aging (EVA) [Internet]. Boston: Academic Press; 2015 [cited 2022 May 1]. p. 137–45. Available from: https://www.sciencedirect.com/science/article/pii/B9780128013878000144

18. Martinsson A, Östling G, Persson M, Sundquist K, Andersson C, Melander O, et al. Carotid Plaque, Intima-Media Thickness, and Incident Aortic Stenosis. Arterioscler Thromb Vasc Biol. 2014 Oct;34(10):2343–8.

19. Petrini J, Yousry M, Eriksson P, Björk HM, Rickenlund A, Franco-Cereceda A, et al. Intima-media thickness of the descending aorta in patients with bicuspid aortic valve. Int J Cardiol Heart Vasc. 2016 Apr 2;11:74–9.

20. Gerthoffer WT. Mechanisms of Vascular Smooth Muscle Cell Migration. Circ Res. 2007 Mar 16;100(5):607–21.

21. Louis SF, Zahradka P. Vascular smooth muscle cell motility: From migration to invasion. Exp Clin Cardiol. 2010;15(4):e75–85.

22. VanderLaan PA, Reardon CA, Getz GS. Site specificity of atherosclerosis: site-selective responses to atherosclerotic modulators. Arterioscler Thromb Vasc Biol. 2004 Jan;24(1):12–22.

23. Tuzcu EM, Kapadia SR, Tutar E, Ziada KM, Hobbs RE, McCarthy PM, et al. High prevalence of coronary atherosclerosis in asymptomatic teenagers and young adults: evidence from intravascular ultrasound. Circulation. 2001 Jun 5;103(22):2705–10.

24. Boussel L, Rayz V, McCulloch C, Martin A, Acevedo-Bolton G, Lawton M, et al. Aneurysm Growth Occurs at Region of Low Wall Shear Stress. Stroke. 2008 Nov;39(11):2997–3002.

25. Sakalihasan N, Limet R, Defawe O. Abdominal aortic aneurysm. The Lancet. 2005 Apr 30;365(9470):1577–89.

26. Rutherford's Vascular Surgery and Endovascular T - 9780323427913 [Internet]. US Elsevier Health. [cited 2022 Apr 12]. Available from: https://www.us.elsevierhealth.com/rutherfords-vascular-surgery-and-endovascular-therapy-2-volume-set-9780323427913.html

27. Elefteriades JA, Farkas EA. Thoracic Aortic Aneurysm. J Am Coll Cardiol. 2010 Mar 2;55(9):841–57.

28. Koullias G, Modak R, Tranquilli M, Korkolis DP, Barash P, Elefteriades JA. Mechanical deterioration underlies malignant behavior of aneurysmal human ascending aorta. J Thorac Cardiovasc Surg. 2005 Sep;130(3):677–83.

29. Gerhard-Herman M, Beckman JA, Creager MA. Chapter 12 - Vascular Laboratory Testing. In: Creager MA, Beckman JA, Loscalzo J, editors. Vascular Medicine: A Companion to Braunwald's Heart Disease (Second Edition) [Internet]. Philadelphia: W.B. Saunders; 2013 [cited 2022 May 10]. p. 148–65. Available from: https://www.sciencedirect.com/science/article/pii/B9781437729306000124

30. Thoracic Aneurysm: Background, Pathophysiology, Etiology. 2022 Mar 8 [cited 2022 Jun 18]; Available from: https://emedicine.medscape.com/article/761627-overview

31. Management of thoracic aortic aneurysm in adults - UpToDate [Internet]. [cited 2022 May 11]. Available from: https://www.uptodate.com/contents/management-of-thoracic-aortic-aneurysm-in-adults#H8

32. Pressler V, Judson McNamara J. Thoracic aortic aneurysm Natural history and treatment. J Thorac Cardiovasc Surg. 1980 Apr 1;79(4):489–98.

33. Levy D, Goyal A, Grigorova Y, Farci F, Le JK. Aortic Dissection. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 [cited 2022 Jun 18]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK441963/

34. Tanaka A, Estrera AL, Safi HJ. Open thoracoabdominal aortic aneurysm surgery technique: how we do it. J Cardiovasc Surg (Torino). 2021 Aug;62(4):295–301.

35. Teufelsbauer H, Polterauer P, Lammer J, Huk I, Nanobachvili J, Kretschmer G. Repair of abdominal aortic aneurysms: the benefits of offering both endovascular and open surgical techniques. Perspect Vasc Surg Endovasc Ther. 2006 Sep;18(3):238–46.

36. Overview of open surgical repair of the thoracic aorta - UpToDate [Internet]. [cited 2022 May 12]. Available from: https://www.uptodate.com/contents/overview-of-open-surgical-repair-of-the-thoracic-aorta?topicRef=8189&source=see_link#H113878156

37. Kadakol AK, Nypaver TJ, Lin JC, Weaver MR, Karam JL, Reddy DJ, et al. Frequency, risk factors, and management of perigraft seroma after open abdominal aortic aneurysm repair. J Vasc Surg. 2011 Sep;54(3):637–43.

38. Ma H, Xiao Z, Shi J, Liu L, Qin C, Guo Y. Aortic arch cannulation with the guidance of transesophageal echocardiography for Stanford type A aortic dissection. J Cardiothorac Surg. 2018 Oct 11;13(1):106.

39. Boccalini S, Swart LE, Bekkers JA, Nieman K, Krestin GP, Bogers AJJC, et al. Peri-aortic fluid after surgery on the ascending aorta: Worrisome indicator of complications or innocent postoperative finding? Eur J Radiol. 2017 Oct;95:332–41.

40. Andrade D, Vinck EE, Torres LN. Two-stage Omental Flap Approach for Ascending Aortic Graft Infection. Braz J Cardiovasc Surg. 2020;35(3):XII–XIV.

41. Kunibe Y, Ando M, Komae H, Shimada S, Kinoshita O, Yamauchi H, et al. Perigraft seroma after total aortic arch replacement using Triplex graft. Gen Thorac Cardiovasc Surg. 2022 Mar 29;

42. Safi HJ. Beware of the "Medusa." J Thorac Cardiovasc Surg. 2015 Apr 1;149(4):964.

43. Fazel SS, David TE. Aortic valve-sparing operations for aortic root and ascending aortic aneurysms. Curr Opin Cardiol. 2007 Nov;22(6):497–503.

44. Veldtman GR, Connolly HM, Orszulak TA, Dearani JA, Schaff HV. Fate of bicuspid aortic valves in patients undergoing aortic root repair or replacement for aortic root enlargement. Mayo Clin Proc. 2006 Mar;81(3):322–6.

45. Alonso Pérez M, Llaneza Coto JM, del Castro Madrazo JA, Fernández Prendes C, González Gay M, Zanabili Al-Sibbai A. Debranching aortic surgery. J Thorac Dis. 2017 May;9(Suppl 6):S465–77.

46. Thomas M, Li Z, Cook DJ, Greason KL, Sundt TM. Contemporary results of open aortic arch surgery. J Thorac Cardiovasc Surg. 2012 Oct;144(4):838–44.

47. Becquemin JP, Pillet JC, Lescalie F, Sapoval M, Goueffic Y, Lermusiaux P, et al. A randomized controlled trial of endovascular aneurysm repair versus open surgery for abdominal aortic aneurysms in low- to moderate-risk patients. J Vasc Surg. 2011 May;53(5):1167-1173.e1.

48. Moll FL, Powell JT, Fraedrich G, Verzini F, Haulon S, Waltham M, et al. Management of abdominal aortic aneurysms clinical practice guidelines of the European society for vascular surgery. Eur J Vasc Endovasc Surg Off J Eur Soc Vasc Surg. 2011 Jan;41 Suppl 1:S1–58.

49. Chaikof EL, Brewster DC, Dalman RL, Makaroun MS, Illig KA, Sicard GA, et al. SVS practice guidelines for the care of patients with an abdominal aortic aneurysm: executive summary. J Vasc Surg. 2009 Oct;50(4):880–96.

50. Dawson J, Vig S, Choke E, Blundell J, Horne G, Downham C, et al. Medical optimisation can reduce morbidity and mortality associated with elective aortic aneurysm repair. Eur J Vasc Endovasc Surg Off J Eur Soc Vasc Surg. 2007 Jan;33(1):100–4.

51. Faggiano P, Bonardelli S, De Feo S, Valota M, Frattini S, Cervi E, et al. Preoperative cardiac evaluation and perioperative cardiac therapy in patients undergoing open surgery for abdominal aortic aneurysms: effects on cardiovascular outcome. Ann Vasc Surg. 2012 Feb;26(2):156–65.

52. Uflacker R, Robison JD, Schonholz C, Ivancev K. Clinical experience with a customized fenestrated endograft for juxtarenal abdominal aortic aneurysm repair. J Vasc Interv Radiol JVIR. 2006 Dec;17(12):1935–42.

53. Ishimaru S. Endografting of the aortic arch. J Endovasc Ther Off J Int Soc Endovasc Spec. 2004 Dec;11 Suppl 2:II62-71.

54. Franzese I, Petrilli G, Puppini G, Bacich D, Giambruno V, Faggian G. Total Endovascular Aortic Arch Repair with Branched Graft. AORTA J. 2019 Nov 26;7(4):121–4.

55. Endovascular repair of the thoracic aorta - UpToDate [Internet]. [cited 2022 May 15]. Available from: https://www.uptodate.com/contents/endovascular-repair-of-the-thoracic-aorta?search=endovascular%20aneurysm%20repair&source=search_result&selectedTitle=6~150&usage_type=default&display_rank=6#H67146303

56. Yang X, Dai XC, Zhu JC, Luo YD, Fan HL, Feng Z, et al. Treatment for thoracoabdominal aortic aneurysm by fenestrated endovascular aortic repair with physician-modified stent graft. J Int Med Res. 2018 May;46(5):2014–22.

57. Bulder RMA, Bastiaannet E, Hamming JF, Lindeman JHN. Meta-analysis of long-term survival after elective endovascular or open repair of abdominal aortic aneurysm. Br J Surg. 2019 Apr;106(5):523–33.

58. Powell JT, Sweeting MJ, Ulug P, Blankensteijn JD, Lederle FA, Becquemin JP, et al. Meta-analysis of individual-patient data from EVAR-1, DREAM, OVER and ACE trials comparing outcomes of endovascular or open repair for abdominal aortic aneurysm over 5 years. Br J Surg. 2017 Feb;104(3):166–78.

59. United Kingdom EVAR Trial Investigators, Greenhalgh RM, Brown LC, Powell JT, Thompson SG, Epstein D, et al. Endovascular versus open repair of abdominal aortic aneurysm. N Engl J Med. 2010 May 20;362(20):1863–71.

60. Brown LC, Thompson SG, Greenhalgh RM, Powell JT, Endovascular Aneurysm Repair trial participants. Incidence of cardiovascular events and death after open or endovascular repair of abdominal aortic aneurysm in the randomized EVAR trial 1. Br J Surg. 2011 Jul;98(7):935–42.

61. Blankensteijn JD, de Jong SECA, Prinssen M, van der Ham AC, Buth J, van Sterkenburg SMM, et al. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. N Engl J Med. 2005 Jun 9;352(23):2398–405.

62. EVAR trial participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. Lancet Lond Engl. 2005 Jul 25;365(9478):2179–86.

63. Mell MW, Wang NE, Morrison DE, Hernandez-Boussard T. Interfacility transfer and mortality for patients with ruptured abdominal aortic aneurysm. J Vasc Surg. 2014 Sep;60(3):553–7.

64. Brattheim BJ, Eikemo TA, Altreuther M, Landmark AD, Faxvaag A. Regional disparities in incidence, handling and outcomes of patients with symptomatic and ruptured abdominal aortic aneurysms in Norway. Eur J Vasc Endovasc Surg Off J Eur Soc Vasc Surg. 2012 Sep;44(3):267–72.

65. Moore R, Nutley M, Cina CS, Motamedi M, Faris P, Abuznadah W. Improved survival after introduction of an emergency endovascular therapy protocol for ruptured abdominal aortic aneurysms. J Vasc Surg. 2007 Mar;45(3):443–50.

66. Larzon T, Lindgren R, Norgren L. Endovascular treatment of ruptured abdominal aortic aneurysms: a shift of the paradigm? J Endovasc Ther Off J Int Soc Endovasc Spec. 2005 Oct;12(5):548–55.

67. Alsac JM, Desgranges P, Kobeiter H, Becquemin JP. Emergency endovascular repair for ruptured abdominal aortic aneurysms: feasibility and comparison of early results with conventional open repair. Eur J Vasc Endovasc Surg Off J Eur Soc Vasc Surg. 2005 Dec;30(6):632–9.

68. Woodburn KR, Chant H, Davies JN, Blanshard KS, Travis SJ. Suitability for endovascular aneurysm repair in an unselected population. Br J Surg. 2001 Jan;88(1):77–81.

69. Kent WDT, Appoo JJ, Bavaria JE, Herget EJ, Moeller P, Pochettino A, et al. Results of type II hybrid arch repair with zone 0 stent graft deployment for complex aortic arch pathology. J Thorac Cardiovasc Surg. 2014 Dec 1;148(6):2951–5.

70. Li Y, Li Z, Wang S, Chang G, Wu R, Hu Z, et al. Endovascular versus Open Surgery Repair of Ruptured Abdominal Aortic Aneurysms in Hemodynamically Unstable Patients: Literature Review and Meta-Analysis. Ann Vasc Surg. 2016 Apr;32:135–44.

71. van Beek SC, Conijn AP, Koelemay MJ, Balm R. Editor's Choice - Endovascular aneurysm repair versus open repair for patients with a ruptured abdominal aortic aneurysm: a systematic review and meta-analysis of short-term survival. Eur J Vasc Endovasc Surg Off J Eur Soc Vasc Surg. 2014 Jun;47(6):593–602.

72. Veith FJ, Lachat M, Mayer D, Malina M, Holst J, Mehta M, et al. Collected world and single center experience with endovascular treatment of ruptured abdominal aortic aneurysms. Ann Surg. 2009 Nov;250(5):818–24.

73. IMPROVE Trial, Powell JT, Thompson SG, Thompson MM, Grieve R, Nicholson AA, et al. The Immediate Management of the Patient with Rupture: Open Versus Endovascular repair (IMPROVE) aneurysm trial--ISRCTN 48334791 IMPROVE trialists. Acta Chir Belg. 2009 Dec;109(6):678–80.

74. Chaikof EL, Dalman RL, Eskandari MK, Jackson BM, Lee WA, Mansour MA, et al. The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. J Vasc Surg. 2018 Jan;67(1):2-77.e2.

75. Wanhainen A, Verzini F, Van Herzeele I, Allaire E, Bown M, Cohnert T, et al. Editor's Choice - European Society for Vascular Surgery (ESVS) 2019 Clinical Practice Guidelines on the Management of Abdominal Aorto-iliac Artery Aneurysms. Eur J Vasc Endovasc Surg Off J Eur Soc Vasc Surg. 2019 Jan;57(1):8–93.

10. BIOGRAPHY

My name is Maor Meir Pichadze, I was born in Israel on January 1st, 1994. After high school, I served in the army as a combat medic and clinic manager for 3 years, which I enjoyed very much.

During my military service, I decided that I want to take my passion, love, and knowledge of medicine to the next level and become the best version of myself in the field, so I decided to chase a career as a medical doctor. After the military service, I was accepted to the University of Zagreb, School of Medicine in 2016. In the year 2018, I volunteered to join the "Team 5" medical team to perform a medical mission in Guatemala in which we gave medical assistance to remote villages after the devastating volcanic eruption. This paper is my graduation thesis and the last step to becoming officially a Doctor of Medicine.