

Surgical Management of Aortic Pathology

Current Fundamentals
for the Clinical Management
of Aortic Disease

Olaf H. Stanger
John R. Pepper
Lars G. Svensson
Editors



Springer

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Foreword

Treating aortic disease has always been an important but often complex area of cardiovascular surgery. This is not only because of the aorta's role as the body's largest and most vital artery but also because aortic disease can take a variety of forms with a broad range of consequences, from slowly progressive, asymptomatic disease to sudden death by rupture.

The surgical treatment of aortic disease, particularly aortic aneurysm, is one of my long-time interests. In 1949, during my residency at Johns Hopkins, I was assisting surgeon Grant Ward with an emergency procedure and ended up performing one of the first excisions of an aortic aneurysm. Later, during my early years at Baylor College of Medicine in the 1950s, my friend and mentor Michael E. DeBakey and I, along with some of our colleagues, developed techniques for replacing aneurysmal aortic segments, first with homografts and later with synthetic grafts. In addition to treating aortic aneurysm, we used these techniques in the first successful repair of a case of chronic aortic dissection in 1954. Also of great interest to us were methods of preventing ischemic injury, particularly to the brain and spinal cord, during these procedures. It pleases me to see how much progress has been made in aortic surgical techniques, technology, and protective adjuncts since those days.

In this book, *Surgical Management of Aortic Pathology*, Olaf H. Stanger and my long-time colleagues John R. Pepper and Lars G. Svensson have assembled the knowledge of many great minds (and hands) in the field of aortic surgery and related disciplines. This volume contains valuable information on a wide range of topics, including the biological underpinnings of aortic disease; modern diagnostic methods; open surgical, endovascular, and hybrid techniques of repairing the various aortic segments and the aortic valve; and methods of protecting vital organs against ischemia during these often complex procedures.

I congratulate Drs. Stanger, Pepper, and Svensson, as well as the many contributors, for creating a comprehensive work on a vital subject.

Houston, TX, USA

Denton A. Cooley

Preface

Current knowledge suggests that the aorta, long seen from a purely mechanistic view as a more or less rigid tube, is in fact a highly functional, metabolically active, hemodynamically responsive, and adaptive structure with laminar flow.

The editors have sought to review and understand the aorta as an organ *per se*. Neither disease nor treatment can be understood in isolation because any change caused by pathology or intervention inevitably affects upstream and downstream segments. Accordingly, this comprehensive work not only includes fundamentals of anatomy and development, but is further focused on advances in imaging, genetics, physiology, molecular biochemistry, and all current treatment options and strategies.

Over time numerous concepts have emerged to explain certain aortic pathologies, but most conditions are much more complex than previously thought, with interactions and potential overlap of mechanisms adding to the complexity.

Perhaps the most important evolution has been brought about by advanced imaging tools visualizing flows, forces, lesions, and changes with previously unthinkable precision. Each patient represents a highly individual case with multiple conditions that influence function, morphology, adaptations, interaction, progression, and risk. Genetic understanding has also grown rapidly and patients, particularly those with hereditary diseases, are increasingly well informed. In an era of rapid genetic analysis, they seek advice on how to prevent aortic rupture, dissection, and death. This presents new challenges as it is difficult to counsel individuals who are seen at such an early stage of their disease that means of accurately predicting their specific outcomes have yet to be developed.

Given the great diversity of disease and treatment concepts, the challenge of individual decision-making calls for highly *specialized interdisciplinary management* involving cardiac and vascular surgeons, interventional cardiologists and radiologists, imaging experts, geneticists, and others. Ideally, each patient will have an individually tailored treatment concept.

Aside from acute aortic events, most aortic diseases are indolent and often discovered by chance. At the same time, as more and more patients are diagnosed and undergo treatment, the group of “survivors” grows constantly, with the ever-present risk of pathology progression and exposure to future complications. Since patients must be followed longer, with management of disease

progression and associated complications, specialist care of patients with aortopathy is a *life-long commitment*.

With expanding insight into the mechanisms that underlie aortic diseases, *myths and paradigms* are challenged and questioned, reviewed, and adapted. Clearly, not one technique or standard protocol will fit all patients' conditions. Conventional concepts, i.e., excision and local replacement of an aortic dissection entry tear, are now considered insufficient to cause false lumen collapse and subsequent remodeling with a near normal prognosis in most, if not all, cases. New techniques, particularly interventional and hybrid techniques, are developing rapidly and taking their places in the toolbox. But do they translate into better overall outcome?

"The more we know the less we understand," but progress is clearly being made in parallel with new controversies to resolve. We are honored and thankful that these many experts on aortic medicine and surgery readily agreed to contribute to this comprehensive volume, sharing their knowledge, experience, and modern outlooks. We hope that this book will provide the reader with stimulating and valuable new insights into the ongoing quest for optimal patient care.

We wish to acknowledge the valuable support of Genie Lamont (Graz, Austria), and Wilma McHugh (Springer; Heidelberg, Germany) in the preparation of this book.

And foremost we are very grateful to the editorial skill and persistence of Sara Baumberger (Project Coordinator; Berne, Switzerland) which proved indispensable in the compilation of this extensive work.

London, UK; Munich, Germany
London, UK
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Olaf H. Stanger
John R. Pepper
Lars G. Svensson

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A Brief History of Aortic Pathology and Surgery

1

Olaf H. Stanger

1.1 The Earliest Descriptions and Concepts

“What’s past is prologue” (William Shakespeare, “The Tempest,” Act 1, Scene 1).

Several early authors are credited with the first mention of the aorta and its abnormal variations, but at times when even the function of vessels was obscure, it is difficult to say what role and relevance were attributed to their observations.

The oldest written narrative of aortic pathologies is found in the **Ebers Papyrus**, named after the German Egyptologist **Georg Ebers** (1837–1898), who purchased the 2 meter scroll with 108 columns of text in 1872 in Thebes. He brought the papyrus to Leipzig where it is still kept in the university library. It is among the oldest and most important medical papyri from ancient Egypt, written in hieratic script and dating to 1550 B.C., but believed to have been copied from earlier texts. The papyrus contains a basic description of the human heart with “vessels attached for every member of the body” [1]. Furthermore, the text identified peripheral and abdominal aneurysms, i.e., “when his abdomen palpitates, it is caused by a swelling therein” [2].

The term “aorta” seems to have first been applied by **Aristotle** (384–322 B.C.). In his works

Historia animalium, *De somno*, and particularly *De partibus animalium*, Aristotle refers to the heart as the center and origin of blood connected with two great vessels (later identified as veins and aorta) and defined the aorta as primary (arterial) outflow of the left ventricle. He then describes all the other vessels as branches of the great vessel and the aorta, and he assumed a two-way blood transfer (blood produced from nutrition travels toward the heart only to be collected and distributed into the vessels) [3]. Whereas he envisioned the heart as the container and origin of blood, he stated that all blood leaves the heart but none returns, and so was unaware of circulation. Although he was not fully clear about the function of the mentioned vessels, the existence of a “great artery” and the “aorta” is clear.

At that time veins and arteries were first distinguished, and the term “aneurysma” (ἀνεύρυσμα), meaning widening or dilation, was introduced.

Next, the prolific Greek physician **Galen of Pergamon** (129/131–c. 200/216), a most prominent figure in medicine and arguably the most accomplished of all medical researchers in antiquity, upon physical examination described “localized pulsatile swellings” and a ruptured aneurysm: “when an aneurysm is wounded, the blood is spouted out with so much violence that it can scarcely be arrested” [4].

Galen also recognized distinct differences between venous (dark) and arterial (bright) blood.

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Although his anatomical experiments on animal models led him to a more advanced understanding of the circulatory system, his work contained scientific errors, i.e., the belief that the circulatory system consists of two separate one-way systems of distribution, rather than a single unified system of circulation. He thought venous blood was generated in the liver, whence it was distributed and consumed by all organs of the body. Similarly, he postulated that arterial blood originated in the heart, whence it was distributed and consumed by all organs of the body. The blood was then regenerated in either the liver or the heart, completing the cycle [5]. Galen's understanding of anatomy was importantly influenced by Hippocrates' then prevailing humoral theory, and his reports were the only anatomical reference for many centuries, although mainly derived from animal (monkeys, pigs) vivisections. Particularly Galen's erroneous conception that venous blood passes through tiny pores in the heart's septum, moves from the right to the left chambers, and is mixed with inhaled air from the lungs long inhibited any new thought. Galen's doctrines dominated medical thinking for many centuries, and particularly his writings on anatomy became the mainstay of the medieval physician's academic curriculum. They remained uncontested until printed descriptions and illustrations of human dissections were published in the seminal work *De humani corporis fabrica* by **Andreas Vesalius** (1514–1564) in 1543 and when **William Harvey** (1578–1657) published his treatise entitled *De motu cordis* in 1628, in which he established that the pumping heart drives blood circulation [6, 7].

The works of **Antyllos**, a Greek surgeon of the Roman period and contemporary of Galen (second century A.D.), have only survived in the writings of **Or(e)ibasius from Pergamon** (325–403), who collected most of the fragments of Antyllos' works [8]. He himself wrote *The Synagogue Medica* and classified aneurysms as either due to dilatation of the arteries (with cylindrical form) or caused by rupture of the artery (with round form) emptying blood into tissues [9]. Antyllos allegedly belonged to the “pneumatist” medical school, described false traumatic and true aneurysms, and

proposed that aneurysms were a consequence of clotting. He was the first to recommend surgical treatment of small peripheral aneurysms by proximal and distal arterial ligation followed by central incision of the sac and evacuation of the thrombotic material (Fig. 1.1) [10]. He did not resect the sac, considering it dangerous to do so, because pulsation puts violent tension on the ligatures, potentially displacing them and leading to fatal bleeding, and advised: “Those who tie the arteries, as I advise, at each extremity, but amputate the intervening dilated part, perform a dangerous operation. The violent tension of the arterial *pneuma* often displaces the ligatures.” He also opposed surgery for large aneurysms but did operate on peripheral aneurysms. Antyllos expressed the (still valid) dilemma in stating: “To decline treatment of any aneurysms is foolish, but it is also dangerous to operate on all of them” [8]. Antyllos' detailed technique is the earliest record of therapy of aneurysms. Importantly, this was recommended as state of the art by **Albucasis** (**Abu al-Qasim** al-Zahrawi; 936–1013) 800 years later [11] and, in fact, was the best surgical treatment available until the end of the nineteenth century (Fig. 1.2) [12].

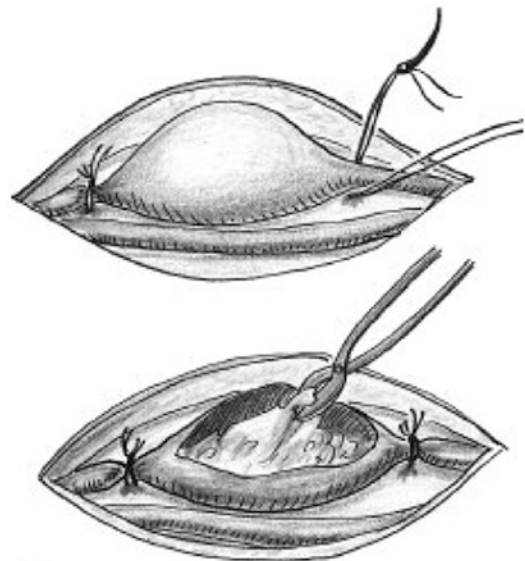
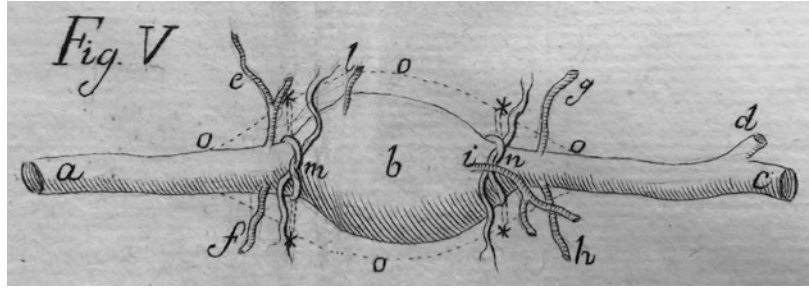


Fig. 1.1 Surgical treatment of aneurysms as described by Antyllos. Proximal and distal ligation followed by central incision of the sac and removal of thrombotic material (from Ref. [10], with permission)

Fig. 1.2 Technique by Antyllos is still the state of the art in the eighteenth century [12]



1.2 The Great Leap Forward from Antiquity

Advancements in the vascular field were negligible until the sixteenth century, with a new breed of scientists whose own studies and observations made them question the received wisdom from antiquity. Flemish physician **Andreas Vesalius** laid the overdue foundation of exact modern human anatomy and was the first author to rely solely on his own observations of actual human anatomical dissections (Fig. 1.3a–d) [13]. He could thereby identify many errors passed down from Galen and others that derived from conflicting information from animal vivisection. Vesalius identified aneurysms of the thoracic and abdominal aorta (and considered them untreatable) and diagnosed a traumatic aneurysm in a rider who fell off his horse [14]. His colleague and friend, French surgeon **Ambroise Paré** (1510–1590) of Paris, is best known for reintroducing (and establishing) vascular ligation as treatment of choice for injured blood vessels (Fig. 1.4a–f) [15]. He warned of opening an aneurysm due to the inevitable fatal bleeding and considered aneurysms of internal parts to be incurable: “we cannot cure large aneurysms of the armpit or groin, for on cutting into them so large quantity of the blood and vital spirits escapes that the patient dies” [16]. He thought of vascular calcifications as “a gift from God” to prevent rupture of the aneurysm. His was one of the earliest accounts of aneurysms presumably caused by the spirochete that causes syphilis, then called the “French disease”: “The aneurismaes which happen in the internall parts are incurable. Such as frequently happen to those who have often had the unction

and sweat of the cure of the French disease, because the blood being so attenuated and heated therewith that it cannot be contained in the receptacles of the Artery, it distends it to that largeness as to hold a man’s fist; Which I have observed in the dead body of a certain Taylor, who by an Aneurisma of the Arterious veine suddenly whiles hee was playing at Tennis fell downe dead, the vessel being broken; his body being opened I found a great quantity of blood powred forth into the capacity of the chest, but the body of the Artery was dilated to that largeness I formerly mentioned, and the inner Coate thereof was bony. For which cause within a while after I shewed it to the great admiration of the beholders in the Physitions Schole whilst I publicly dissected a body there; the whilst he lived said he felt a beating and a great heate over all his body by the force of the pulsation of all the Arteryes, by occasion whereof he often swounded” [17].

German physician **Daniel Sennert(us)** (1572–1637) dealt with aortic dissections as separation of the aortic wall layers in a broader context [18], and German barber surgeon **Matheus Gottfried Purmann** (1648–1711) operated on a series of antecubital aneurysms in the 1680s, putting ligatures above and below the aneurysm and opening and removing the sac (Fig. 1.5) [19, 20]. **Giovanni Maria Lancisi** (1654–1720) of Rome published *De motu cordis et aneurysmatibus* in 1728, providing descriptions of the etiology and pathology of aneurysms, including case studies (Fig. 1.6a, b) [21].

The founder of modern pathology, **Giovanni Battista Morgagni** (1682–1771), is best known for introducing the concept that every disease



Fig. 1.3 (a–d) *De humani corporis fabrica* (Andreas Vesalius, 1543). (a, b) Title page. Vesal looks to us while performing an autopsy, (c) depiction of the main arterial

blood vessels of the human body, (d) descending aorta (arteria magna) with side branches [13]



Fig. 1.4 (a–f) Opera chirurgica (Ambroise Paré, 1594). (a) Title page, (b) abdominal anatomy, (c) most arterial injuries were traumatic in origin, (d–f) surgical instruments suggested by Paré [15]

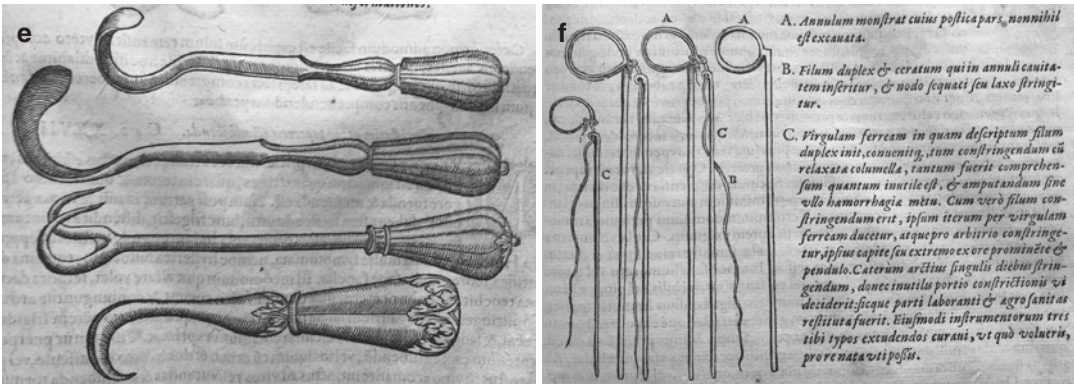


Fig. 1.4 (continued)

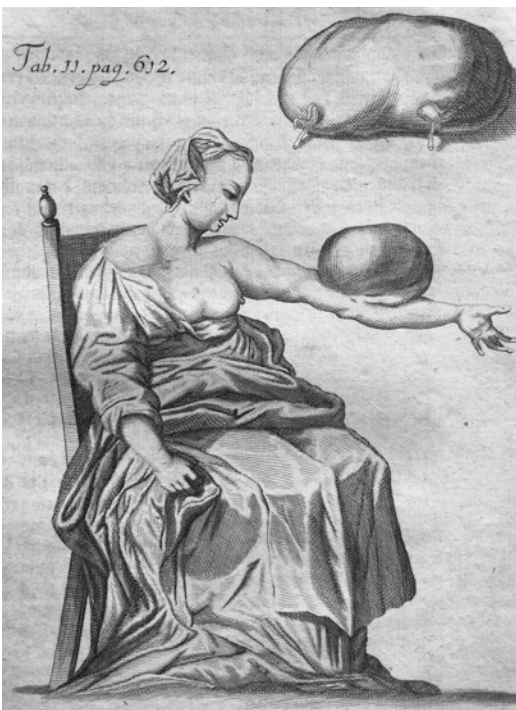


Fig. 1.5 *Chirurgia curiosa* (Gottfried Purmann, 1699). Antecubital aneurysm [19]

originates in a distinct anatomical location, as indicated by the title of his most important work (Fig. 1.7a–c). He left a study on dissecting aneurysms in 1761 [22]. He reported several cases in which blood forced its way through the wall “coming out under the external coat of the artery,” and on a patient with acute aortic dissections, “A man ... was taken by pain in the right arm and shortly thereafter in the left arm, ... soon

to be followed by a tumor on the upper part of the sternum. ... He was instructed to think serious and humble of his departure from life, which was inevitable and very soon to occur” [22]. To Morgagni it was beyond doubt that the syphilitic toxin corrodes the vessel wall, causing dilatation.

The previous year, **King George II of England** (1683–1760) had died suddenly at Kensington Palace from pericardial tamponade caused by a ruptured aortic dissection. The case was very accurately described in the autopsy report by **Frank Nicholls** (1699–1778), the King’s personal physician: “... the pericardium was found distended with a quantity of coagulated blood, nearly a pint...; the whole heart was so compressed as to prevent any blood contained in the veins from being forced into the auricles; therefore the ventricles were found absolutely void of blood...; and in the trunk of the aorta we found a transverse fissure on its inner side, about an inch and a half long, through which some blood had recently passed under its external coat and formed an elevated echymosis” [23, 24].

The Italian surgeon-anatomist **Antonio Scarpa** (1752–1832), secretary to Morgagni, quoted the writings of **Sennert** in his own book and thought of atherosclerosis as the main driver for aneurysms (Fig. 1.8) [25].

Arterial injuries were common when phlebotomy and bloodletting were applied for a wide range of diseases. In the armpit, the needle frequently lacerated the brachial artery instead of

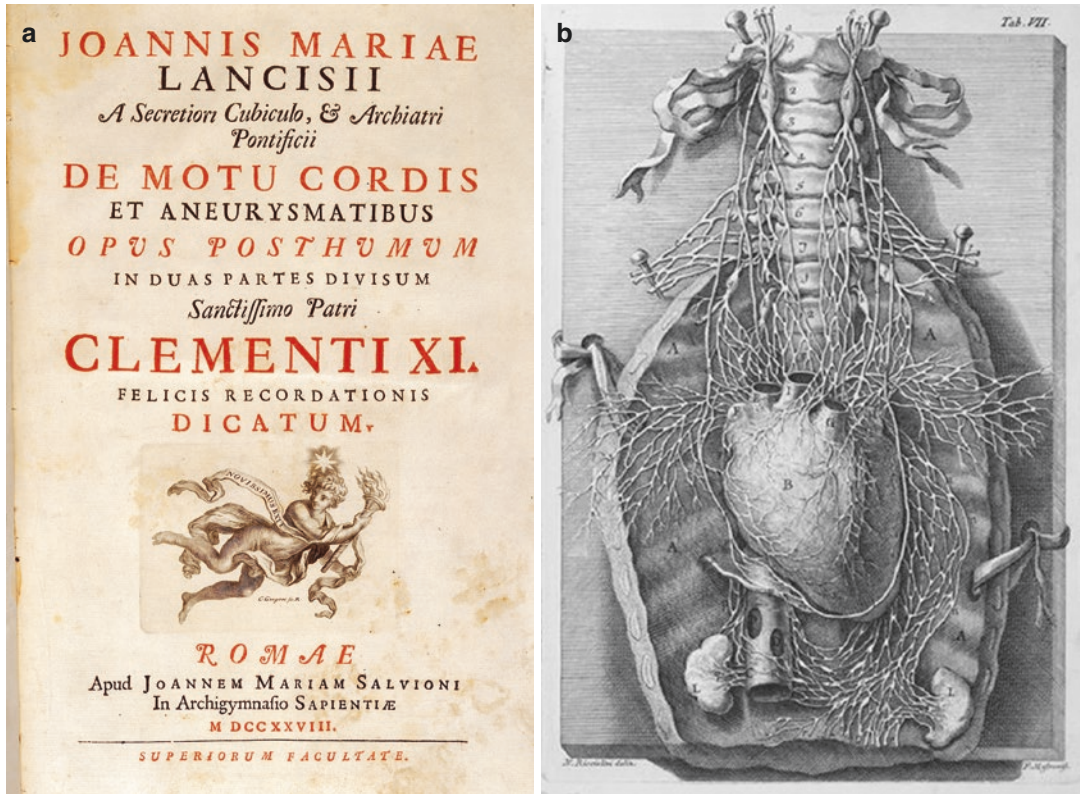


Fig. 1.6 (a, b) De motu cordis et aneurysmatibus (Giovanni Maria Lancisi, 1728). (a) Title page, (b) thoracic anatomy [21]



Fig. 1.7 (a–c) De sedibus et causis morborum per anatomen indagatis (Giovanni Battista Morgagni, 1761). (a) Portrait of Giovanni Battista Morgagni. (b) Title page. (c) Thoracic anatomy [22]

the veins, with false aneurysms, arteriovenous fistula, and potentially fatal rupture as sequelae. In the eighteenth century, riding boots often

caused painful aneurysms of the popliteal artery, which, unsurprisingly, were particularly common in coachmen.

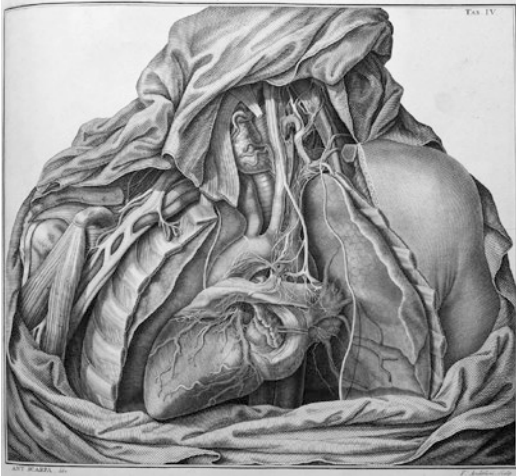


Fig. 1.8 A treatise on the anatomy, pathology, and surgical treatment of aneurysms (Antonio Scarpa, 1808). Illustration of thoracic anatomy [25]

English surgeon **John Hunter** (1728–1793) is well known for his famed ligation of the popliteal artery. He acquired a critical attitude toward traditional medical practice under the influence of his teacher **William Cheselden** (1688–1752), who was fundamental in establishing surgery as medical science, breaking away from the Company of Barbers to form the Company of Surgeons in its own right, which later became the Royal College of Surgeons (of London, 1800; of England, 1843). Accordingly, Hunter’s work represents the emergence of surgery as a scientific discipline based on anatomy and physiology. He thought that an aneurysm develops when the arterial wall loses elasticity and becomes too weak to withstand the force of blood, which, however, does not say much about the cause. From experiments in peeling off the outer part of the carotid artery in dogs without aneurysm formation, he concluded that trauma was not an important cause [26].

For the treatment of femoral aneurysms, he assumed that ligation above and below the aneurysm would suffice, and perfusion to the lower limb would find its way through smaller collateral side vessels. To prove his theory, Hunter

conducted a series of experimental femoral artery occlusions in animals [27]. Upon sacrifice a few weeks later, he injected colored resin into the artery, demonstrating collaterals ensuring sufficient perfusion. Based on this finding, he concluded that with sufficient collaterals, an artery could be safely ligated and that ligation should be done at a distance from the diseased part of the aneurysm to avoid erosion and rupture [28].

Opportunity for proof of concept came with the treatment of a popliteal aneurysm in a 45-year-old coachman, Samuel Smart, on December 12, 1785. Hunter put a ligature on the superficial femoral artery high in the thigh in the area now known as Hunter’s canal (*Canalis adductorius*). The patient survived for 15 months, the aneurysm having shrunk to a hard knot and the limb surviving. Afterward Hunter was able to buy the leg from Smart’s widow and found “a completely thrombosed aneurysm, somewhat larger than a hen’s egg” [29]. This postmortem specimen can still be seen in the Hunterian Museum of the Royal College of Surgeons in Lincoln’s Inn Fields (Fig. 1.9).

Opposition to Hunter’s method was soon to come, and the surgical establishment including **Percival Pott** (1714–1788) intensively defended the common treatment of symptomatic popliteal aneurysm, which meant limb amputation [30].

Although, with reference to Antyllos, Hunter was not the first to treat peripheral aneurysms by ligation, he pioneered the idea of justifying application of surgical techniques on the basis of experimental evidence and clinical success.

Anatomist **William Hunter** (1718–1783), John’s elder brother, had produced a manuscript in 1757 [31], in which he discriminated between true and false aneurysms, describing them as dilated and pulsatile vessels, and also named a third type of aneurysm “that was formed partially by a wound or rupture of some



Fig. 1.9 Popliteal aneurysm surgically treated by John Hunter (1785) (original specimen). Hunterian Museum of the Royal College of Surgeons in Lincoln's Inn Fields

of the coats of the artery, and partly by a dilatation of the rest" [32]. He was the first to describe arteriovenous fistulae, predominantly the result of phlebotomy and injuries to the brachial artery, along with the hissing noise heard on auscultation. Another type of aneurysm was caused by infections, most frequently as a complication of syphilis, although their infectious nature was not yet known and they were rather attributed to a dissolute lifestyle, particularly among soldiers.

Post-venesection brachial artery pseudoaneurysms were treated surgically with proximal ligation by French surgeons **Dominique Anel** (1678–ca. 1730) [33] and **Pierre-Joseph Desault** (1744–1795) in 1785 [34].

John Hunter had taught that ligation could be used for aneurysms of the subclavian, carotid, and femoral arteries. One of his students was **Sir Astley Cooper** (1768–1841), who after experimentation developed retroperitoneal access to the aorta in a cadaver model. In 1805 he performed one of the earliest ligations of the right common carotid artery in a human. And in 1817, in analogy to Hunter's concept, he ligated the distal aorta to control a large ruptured left-sided external iliofemoral aneurysm in a 38-year-old porter, expecting thrombosis and obliteration of the lesion. He managed to get his finger around the aorta through a small transperitoneal incision, passed a single heavy silk ligature around with a needle, and tied the knot. In consequence, however, one leg became ischemic, and the patient survived for barely 48 h. The lesson was that Hunterian ligation was appropriate for aneurysms of small- and intermediate-sized vessels but proved universally fatal in patients with aortic aneurysms. The postmortem specimen is preserved and remains on display in the Gordon Museum of Pathology at King's College London (Fig. 1.10a, b).

Jean-Nicolas Corvisart des Marets (1755–1821), personal physician to **Emperor Napoléon I** (1769–1821), was prominent in medical circles and had an interest in cardiology. He published works on diseases and organic lesions of the heart and the great vessels [35]. Besides describing dilatative cardiomyopathy and congestive heart failure, he provided a detailed evolution of aneurysms of the aorta [36]. In an ascending aortic aneurysm, he described the thrill and retromanubrial dullness to percussion.

French surgeon **René Théophile Laennec** (1781–1826) not only invented the stethoscope but reported several cases of chronic aortic dissection diagnosed using that instrument [37]. He

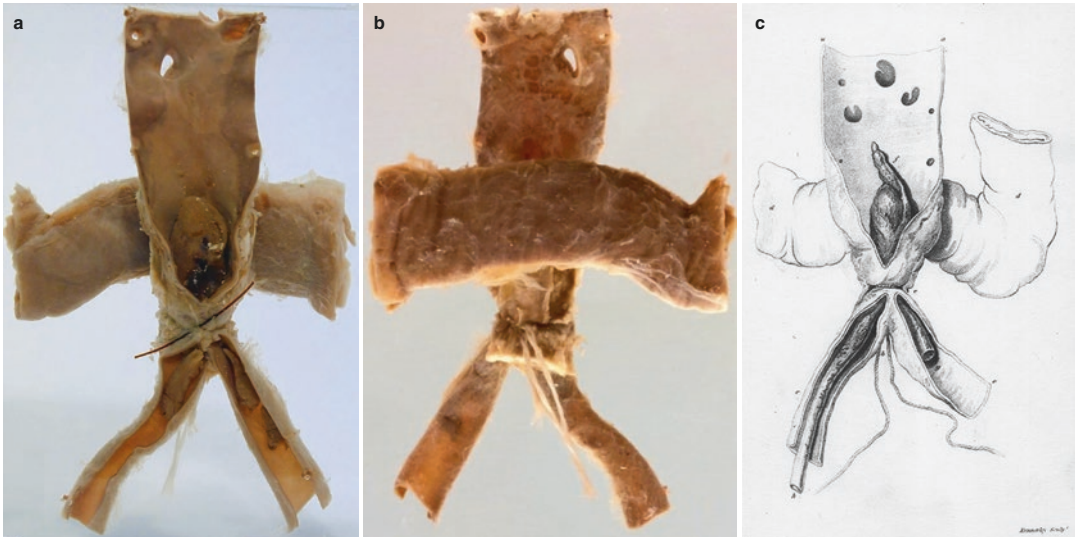


Fig. 1.10 (a and b) Ligature of abdominal aortic aneurysm performed by Sir Astley Cooper 1817 (original specimen). Gordon Museum of Pathology at King's College London

was the first to use the term “dissecting aneurysm” (“L’anévrysme disséquant”) (Fig. 1.11a, b) [37], although, a few years earlier in 1802, **Jean Pierre Maunoir** (1775–1830) had described the pathological process more precisely: “Elles se rompent dans un point, et la tunique externe ou celluleuse, fait poche et s’oppose seule à l’effusion du sang qui passe par la déchirure des tuniques internes” [38], but this went largely unnoticed.

Single cases of fatal ascending aortic aneurysm ruptures were reported by Scottish surgeons **Allan Burns** (1781–1813) [39] and **Joseph Hodgson** (1788–1869) (Fig. 1.12a, b) [40]. In fact, these two works were the first and most important textbooks on heart disease and cardiovascular pathology in English, notably written by two surgeons. Burns had then dissected 14 cases of aortic aneurysm and gave an excellent account of the symptoms. While praising Scarpa’s work on aneurysm, he disagreed with the view that it always resulted from a localized rupture of the inner coat and described diffuse cylindrical dilatation of the aorta with intact coats, a condition later described by Hodgson [41]. In 1824 **Adolph Wilhelm Otto** (1786–1845) provided probably the first description of coarctation of the aorta complicated by

aortic dissection and the presence of a bicuspid aortic valve (BAV) (Fig. 1.13) [42].

In Vienna, **Joseph Škoda** (1805–1881), an expert in physical diagnosis and representative of the legendary Second Vienna Medical School, applied auscultation and percussion with unheard-of precision. He was once called for consultation to Pierre Louis Jean Casimir de Blacas d’Aulps (1771–1839), a French nobleman and former French Minister to Austria, who suffered from unexplained abdominal pain. Whereas three other famous fellow authorities diagnosed liver disease, **Škoda** found a leaking abdominal aneurysm instead based on auscultation and percussion and predicted imminent death. His diagnosis was confirmed by necropsy soon afterward, including the precise dimensions of the aneurysm [43, 44]. His diagnostic skills were precise enough to allow the first pericardial puncture by Viennese surgeon **Franz Schuh** (1804–1865) in 1840 [45].

Descriptions of splitting of the aortic tunica media in cases of chronic dissection were presented by Scottish pathologist **William Henderson** (1810–1872) [46] and the presence of a distal reentry with a false aortic lumen by **Thomas Bevil Peacock** (1812–1882) [47, 48].

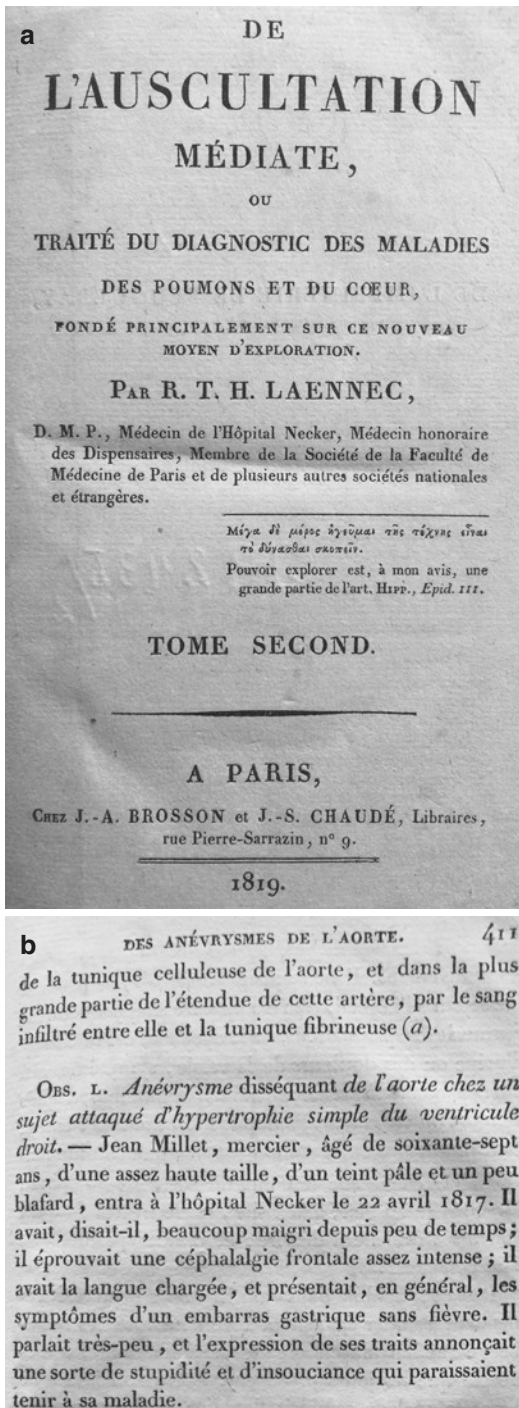


Fig. 1.11 (a, b) De l'Auscultation Médiante, ou Traité du Diagnostic des Maladies des Poumons et du Cœur, Fondé Principalement sur ce Nouveau Moyen d'Exploration. (a) Title page. (b) One of the earliest mentions of "Anévrysme disséquant de l'aorte" (René Théophile Laennec, 1819) [37]

1.3 Concepts and Theories of Pathological Mechanisms

In 1839, Viennese pathologist **Carl von Rokitansky** (1804–1878) explained the difference between dissection and spontaneous rupture on the basis of two types of degenerative changes: either through delamination when the adventitia loses its supporting function for the inner walls or by longitudinal rents due to brittleness and breakdown in the intima and media [49–51]. For dissection, Rokitansky favored causal inflammation, while **Karl Köster** (1843–1904) suggested that mesarteritis extending along the vasa vasorum weakens the media [52].

Rudolf Virchow (1821–1902) in Berlin primarily thought of atheromatous ulceration as the cause of dissection [53]. Another German pathologist, **Friedrich von Recklinghausen** (1833–1910), in 1883 explained dissection as a consequence of inflammation [54], with “molecular changes of the elastic structures or subcellular events” along with stress from elevated blood pressure occurring in the aortic wall [55].

Then in 1875 a relatively high prevalence of aortic aneurysms was observed in army soldiers as compared to sailors [56]. British army surgeon **Francis Henry Welsh** (1839–1910) had studied the records of 53 men who had died from ruptured aortic aneurysm and noted that two-thirds had a documented history of syphilis [57]. Welsh felt this frequency was greater than would be expected in the general population and wondered whether syphilis could be the cause of the aortic aneurysm. It was not until 1905 that the German zoologist **Fritz Schaudinn** (1871–1906) together with dermatologist **Erich Hoffmann** (1868–1959) in Berlin first observed, in autopsy specimens of aorta, the causative spirochete that later became known as *Treponema pallidum* [58].

Sir William Osler (1849–1919) coined the term “mycotic aneurysm” in 1885, and in 1909 he argued for syphilis as an important cause of aneurysm [59]. In 1920 **Friedrich Ernst Krukenberg** (1871–1946) first suggested rupture of the aortic vasa vasorum to be responsible for aortic dissection [60].

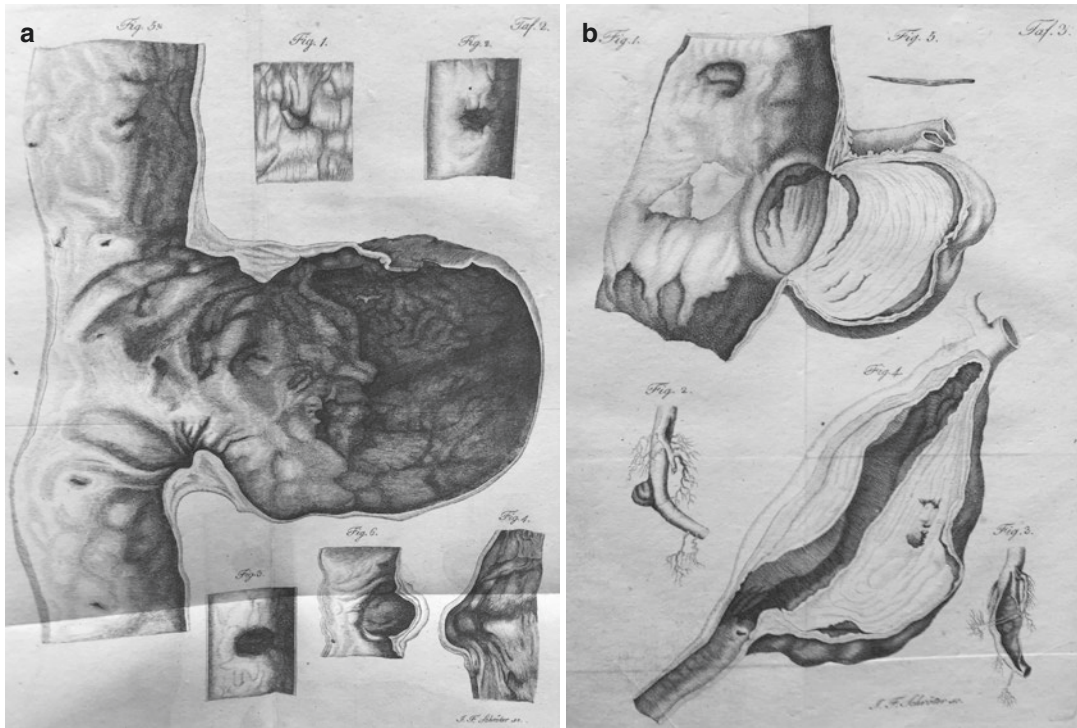


Fig. 1.12 (a, b) Tables depicting aortic saccular and atherosclerotic aneurysms (Joseph Hodgson, 1815) [40]



Fig. 1.13 First description of aortic coarctation complicated by aortic dissection in the presence of bicuspid aortic valve (BAV) (Adolph Wilhelm Otto, 1824) [42]

Again, it was **Thomas Peacock** who reported on 19 cases of aortic dissection in 1843 [61], recognized the importance of the intimal tear, and hypothesized that dissection was the result of disruption of the “internal coats of the vessel” [61]. Peacock even described experiments in which fluids were injected between the adventitia and media of the aorta simulating dissection and observed a tendency to see the canal reopen into

the original vessel; he wrote that this might be seen as “an imperfect natural cure of the disease” [62]. He also noted the difference in prognosis between dissections originating in the ascending aorta and those in the descending aorta. Having continued to collect cases, he published a series of 80 cases of dissection, dividing the process into three stages: (1) rupture of the internal aortic coats, (2) dissection and possible external rupture, and (3) recanalization [61]. Peacock made great contributions toward the understanding of aortic dissection with his experiments and observations. In fact his data on mortality, gender and age distribution, location of dissection, and symptoms are in close agreement with current literature [24]. Thus **Peacock** [47, 48, 63], Baltic German pathologist **Eugen Bostroem** (1850–1928) [64], and **Franz Schede** (1882–1976) [65] all proposed penetration of the aortic wall by blood entering from the lumen as the primary event of aortic dissection. In contrast, **Victor Babes** (1854–1926) and **Teodor Mironescu** (1876–1954) reported a case of dissecting

mesaortitis [66] and had also observed cases of dissection without tears and thus questioned the theory of primary penetration of the aortic wall as proposed by Peacock [67] and others. They rather thought of primary cleavage of the media as the triggering event of aortic dissection. Intramural hematoma (IMH) was subsequently described by Austrian pathologist-anatomist **Hans Eppinger** (1848–1916) [68, 69].

Pathologist **Eduard v. Rindfleisch** (1836–1908) described the breakthrough of blood into the vessel wall due to wear and tear at certain predilected sites [70]. He assumed that pathologically reduced elasticity and resistance of the wall produced this tendency to rupture.

In 1934, pathologist **Theodore Shennan** (1869–1948) from Aberdeen published the data of the largest necropsy series (300 cases) collected at the time and proposed four separate causal theories for aortic dissection: mechanical, inflammatory, degenerative, and congenital [71]. He noted that primary degenerative changes in the media with subsequent loss of elasticity were an important factor underlying the dissection process. The series by pathologist **Albert E. Hirst** (1915–) in 1958 even included 508 such cases [72]. Both reports were fundamental in providing important clinical information and essential for the understanding of the etiology and pathogenesis. The classic form of dissection is defined as entry of blood into the wall of the aorta with subsequent separation of the mural layers.

French pediatrician **Antoine Marfan** (1858–1942) studied the symptoms of the syndrome that would later bear his name and also reported the first case of arachnodactyly in 1896 [73], but it was only in 1943 that **Helen Taussig** (1898–1986) pointed out an association between Marfan disease and aortic medionecrosis [74]. Also in 1943, the association between Marfan syndrome and aortic dissection was first noted by **Lewis E. Etter** (1901–1979) and **Lewis Pellman Glover** (1900–1953) [75].

Swiss physician **Otto Gsell** (1902–1990) reported an aortic wall pathology in 1928 characterized as cystic medionecrosis with focus on degeneration of the muscle elements in the media [76]. Nearly identical “idiopathic aortic medio-

necrosis,” later known as cystic medial degeneration, was described in detail by Austrian pathologist **Jakob Erdheim** (1874–1937) [77]. This pathology, characterized by vacuolization of the media with noninflammatory loss of muscle cells and elastic fibers in the arterial wall, was subsequently accepted as the underlying cause for aortic dissection and rupture. This, however, came to be questioned as experience with larger case series increased and difficulties surfaced due to lack of adequate control series as the histological appearance of the aorta varies considerably with age and even at different levels within the same aorta [78, 79].

From the time of Laennec, numerous theories of causality have developed, namely, that dissecting aneurysms are due to trauma, chronic high blood pressure, infection, degenerative changes, inflammation, or disease of the vessel wall (of either the intima or the media or both). However, the sequence of events in the course of dissection was (and still is) a matter of debate, suggesting multiple and possibly interacting etiological factors.

1.4 Endovascular Treatment with Foreign Bodies

The standard treatment for thoracic aneurysms in the eighteenth and nineteenth century was complete rest, systemic administration of potassium iodide, and starvation diet regulated according to Mr. Tufnell’s method [80]. Other recommendations included vinegar, iron perchloride, alcohol, zinc chloride, gelatin, sodium chloride, or ergot salts [81], albeit with inconsistent success and rather reflecting lack of better options. A new (indirect) approach to prevent rupture was the introduction of foreign material into the aneurysmatic lesion to induce clotting.

1.4.1 Needles

This concept followed the idea that foreign bodies, i.e., needles, would induce irritation and inflammation followed by clotting, thus reduc-

ing flow into the aneurysm sac and stabilizing the aneurysm through subsequent obliteration of the artery.

Surgeon **Benjamin Philipps** (1772–1838) caused clot formation in the femoral and carotid arteries of dogs by inserting needles (1832), later to be supplemented by electrical current (one needle attached to the copper, the other to the zinc pole of a galvanic battery) in an effort to increase clot formation [82]. After sacrificing his experimental animals, he found coagula around the needles and adherent to the vessel wall, which stimulated enthusiasm for the procedure. Simultaneously in Paris, **Alfred-Armand Velpeau** (1795–1867) conducted similar coagulation experiments [83]. Even aneurysms of the ascending aorta were treated with puncture for the sake of scratching the inner layer of the vessel and promoting thrombus, though without success [84]. Results with simple needle puncture were unpredictable, and the technique was ultimately abandoned, only to give way to wires and current.

1.4.2 Wires

To further enhance clot formation, English surgeon **Charles H. Moore** (1821–1870) and British physician **Charles Murchinson** (1830–1879) began to pack aneurysms with wire. They were inspired by a fibrin-covered bullet recovered from an autopsy on a sailor who had died of a gunshot wound to the chest. The metallic bullet that Moore found within the ascending aorta was embedded in fibrin, and it was concluded that a foreign body would attract fibrin, support the mass entangling it, and lead to the eventual filling of the cavity of the aneurysm. A foreign body that would be less irritating and superior to simple needle insertion was thought to be a wire that might be passed in through a small cannula [85]. Moore and Murchinson were the first to attempt percutaneous endovascular aneurysm repair by causing sac thrombosis through direct needle cannulation and wire packing. In 1864 they inserted 26 yards (!) of wire coils into a large thoracic aneurysm via direct aneurysm puncture (Figs. 1.14 and 1.15) [84–87]. They recounted

that they observed a declining pulse, reduction in the size of the aneurysm, and overall clinical improvement. One might call it a success because the aneurysm had indeed partially thrombosed; however, the patient succumbed to sepsis and distal embolism. At autopsy, the coils of wire were filled with “fibrinous coagulum” and were “firmly adherent” [85].

Other investigators amended Moore’s techniques by developing coils from different materials. **Richard Levis** (1827–1890) from Philadelphia and **John Henry Bryant** (1867–1906) from London used horsehair to treat subclavian and popliteal aneurysms, respectively, but both patients suffered rupture shortly after treatment [86]. Other surgeons used iron wire, steel wire, silvered copper wire, gold wire, coil, and metal watch springs, but with none or very limited success (Fig. 1.16) [88]. Instead, the full range of complications became obvious, such as hemorrhage from subtotal packing and distal embolization of wire or thrombus [81].

Sir D’Arcy Power (1855–1941) attributed the disappointing and often variable results to underestimation of the aneurysm size and hence incomplete wire insertion. He therefore used the “Colt apparatus” invented by **George Herbert**

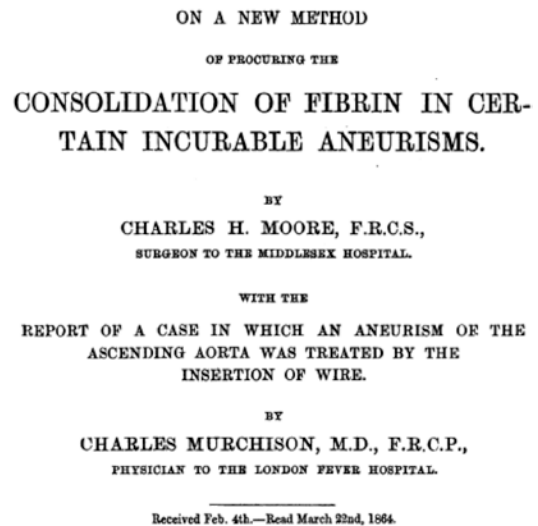


Fig. 1.14 Description of a case in which an aneurysm of the ascending aorta was treated with insertion of wire (1864) [85]

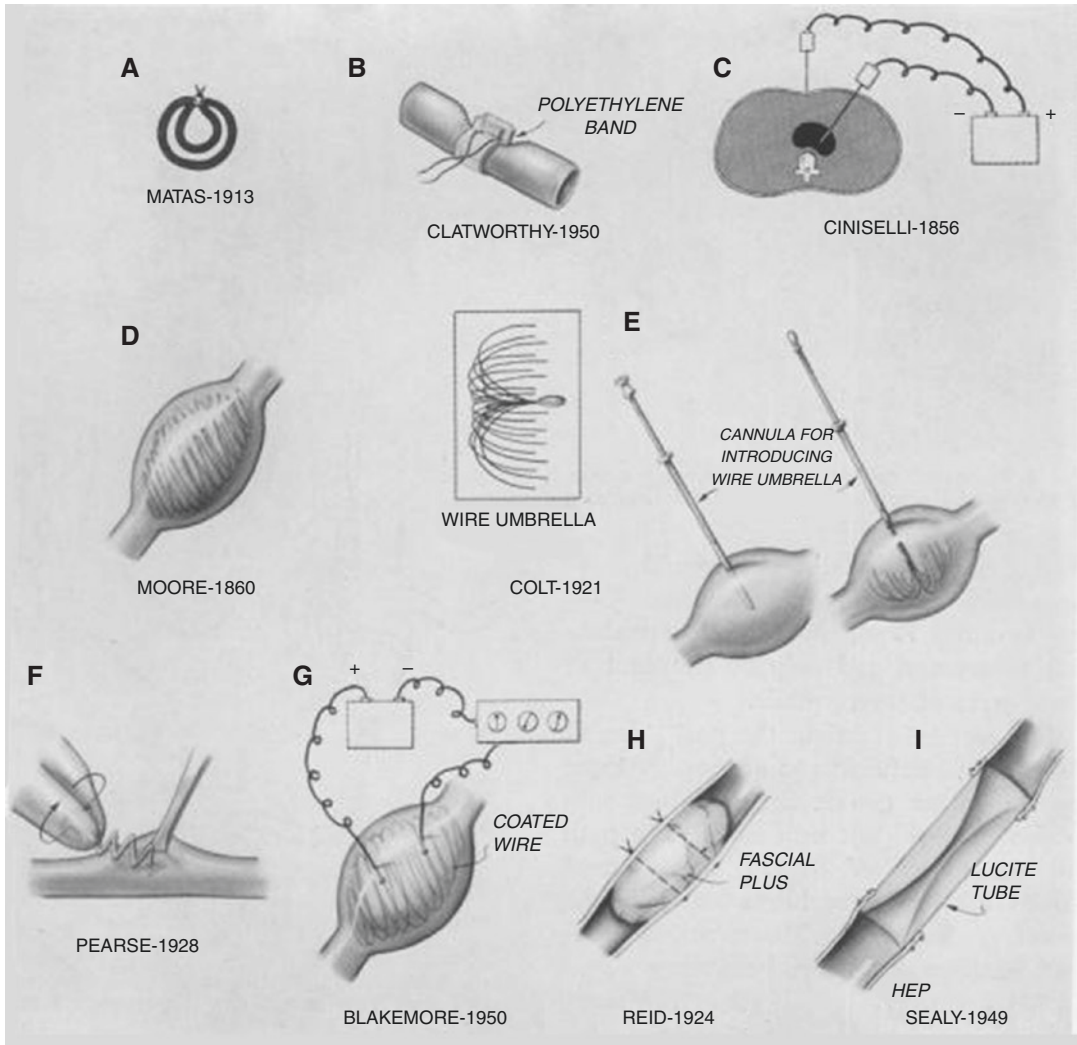


Fig. 1.15 Endovascular treatment with foreign bodies. Endovascular treatment concepts for blood vessel constriction in aneurysms [87]

Colt (1878–1957) of Aberdeen, which consisted of “a trocar and cannula, a ramrod, a tube and a wisp that contained fine steel wires that expanded to form a miniature umbrella” (Figs. 1.15 and 1.17a–c) [87, 89], and reported “a case of aneurysm of the abdominal aorta treated by the introduction of silver wire” in 1903 [90]. The Colt device was remarkably advanced for the time because it opened into a three-dimensional shape. Although ultimate results were poor, an author concluded that “wiring is a good method of relieving pain, ... , but one that may not alter the natural history of the disease” [91].

1.4.3 Electrothrombosis (Galvanopuncture)

The next step was galvanopuncture, or the attempt to induce inflammation and clot formation with electrical current around the electrodes. Others theorized that coagulation resulted from the oxidation of blood cells and proteins or from the chemical deposition of albumin or through decomposition of salts in the blood through the acid produced at the positive pole [81]. Whatever the exact mechanism is, it was thought that current could potentially lead to occlusion of aneurysms.

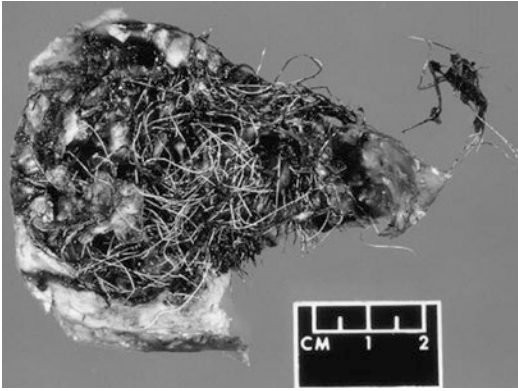


Fig. 1.16 Wire introduced into an aneurysm to promote coagulation (from Ref. [88], with permission)

In one of the earliest contributions in 1824, **Charles Scudamore** (1779–1849) passed a galvanic current through blood, which then formed a dense black coagulum at the positive electrode [92]. In 1832, German physiologist **Johannes Müller** (1801–1858) published investigations describing the effects of galvanism on blood and egg white, among others [93].

Peripheral aneurysms were sometimes treated then by electrocoagulation, but thoracic aneurysms were tackled in similar fashion only in 1846, first by **Luigi Ciniselli** (1803–1878), who is credited with having popularized the technique of galvanic puncture. In 1856 he published data from 50 cases involving electropuncture of aortic aneurysms with a mortality rate of only 14% (as compared with 33% for ligature) and a 50% success rate overall in comparison (Fig. 1.15) [81, 87, 94], stimulating interest in the technique. He also reported a case of aneurysm of the descending aorta that he treated by galvanopuncture across the chest. In 1870 he published 23 cases that he had collected over time, whereby 6 patients recovered (though 3 of them relapsed a few months later) [95].

The experiments by surgeon **John Duncan** (1839–1899) and **Sir Thomas Richard Fraser** (1841–1920) using egg albumin and canine arteries supported the concept of albumin decomposition as the mechanism of current-related coagulation [96]. Duncan used to introduce both poles (steel needles) into an aneurysm and pass a

current for 20 min [97]. The positive needle was covered with gutta-percha (alternatively vulcanite), the negative with glass, and both were inserted into the aneurysm through the skin and thorax (Fig. 1.18a–c) [98, 99]. The electric circuit was closed with a battery of Bunsen cells. Typically, blood clotted and the puncture sites bled. Duncan concluded that the operation delayed death only slightly, if at all. Other surgeons also had fatal outcomes. Complications included distal migration of the wires, formation of emboli, end limb ischemia, sepsis, and formation of distal aneurysms from the altered hemodynamics [100].

Rather than giving up on this technique, **Alfonso Corradi** (1833–1892) of Bologna added electrical current in 1879 to Moore’s original wire work, with silver and copper wire coils causing (electro)thrombosis. The passage of current through the coil was intended to encourage thrombosis, and it was suggested that an electrical current applied to a permanently inserted metallic coil would combine the dual benefits of wire insertion and electrothrombosis. The method became widely referred to as “Moore-Corradi method” and was widely used for years [101].

Surgeon **Joseph Ransohoff** (1853–1923) of Cincinnati also used this procedure by passing electric current through wire to enhance coagulation [102]. **Guy LeRoy Hunner** (1869–1957) in 1900 compiled 28 cases of aneurysms of the aorta treated by wiring according to the Moore-Corradi method [101]. Although most cases at the time generally died less than 1 year after the procedure due to rupture and sepsis, one case in this particular series managed to survive for at least 38 years [103]. Several accounts of galvanopuncture in thoracic (arch) aneurysms were given, some with encouraging results [104], where it was felt that the walls of the sac have had become stronger, thus lessening the risk of external rupture. Others were less fortunate, with fatal outcomes [105].

Arthur Blakemore (1897–1970) of New York explained the unsatisfying results with underestimation of the aneurysm size and incomplete wire insertion. Subsequently he proposed a novel method of determining the amount of wire

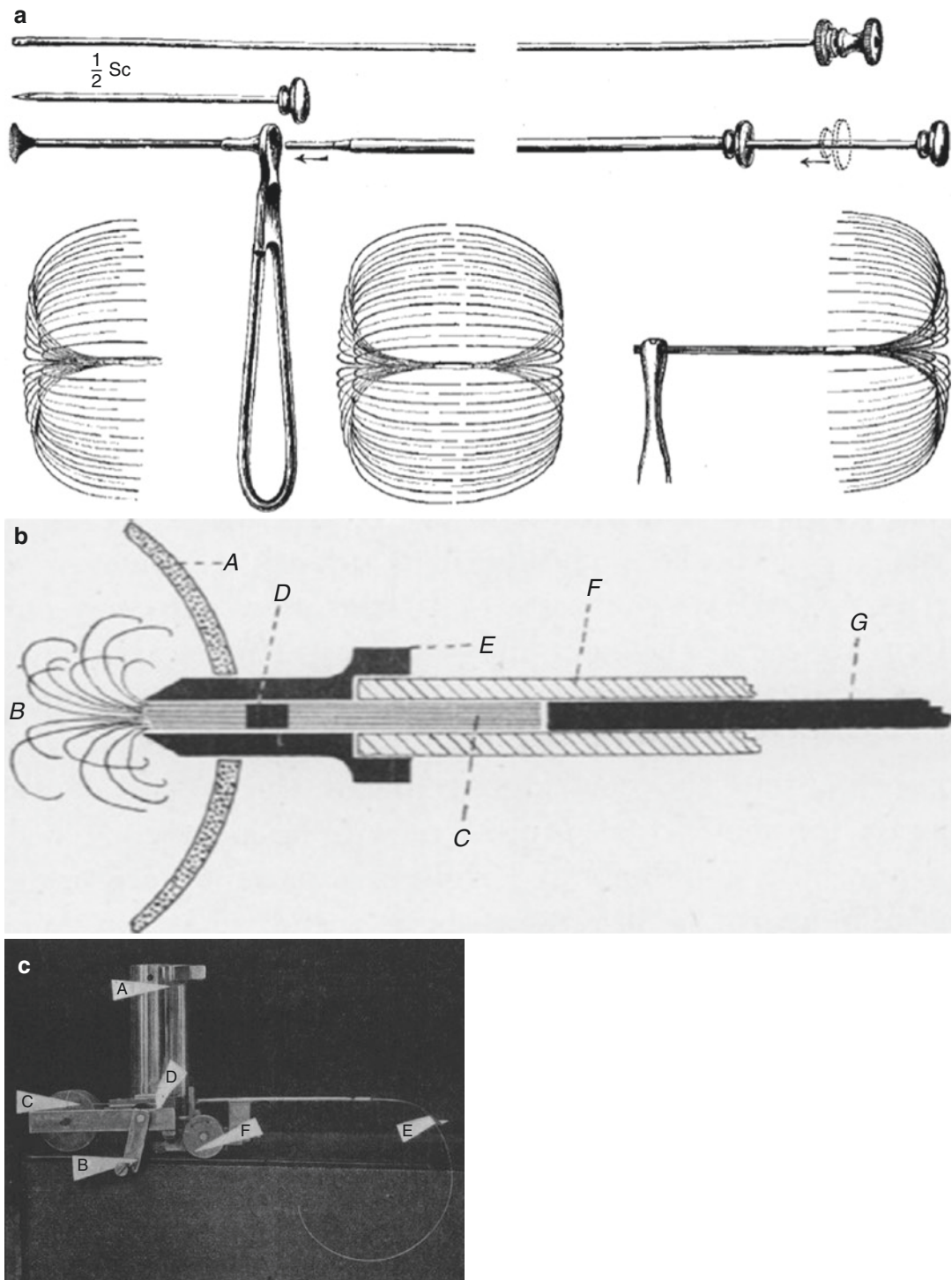


Fig. 1.17 (a–c) Colt’s apparatus for wiring aneurysms. (a) Early instrument with cages for wiring aneurysms; (b) diagrammatic section through Colt’s instrument III in situ. Sac of aneurysm (A), expanding cage (B), compressed cage (C), solder at the center of the cage (D), collar on the

cannula (E), cartridge (F), ramrod (G); (c) flag-labelled side view of Colt’s instrument No. 2. A. Fixed handle (A), moveable handle (B), reel (C), milling tool (D), coil of wire (E), stud (F) [90]

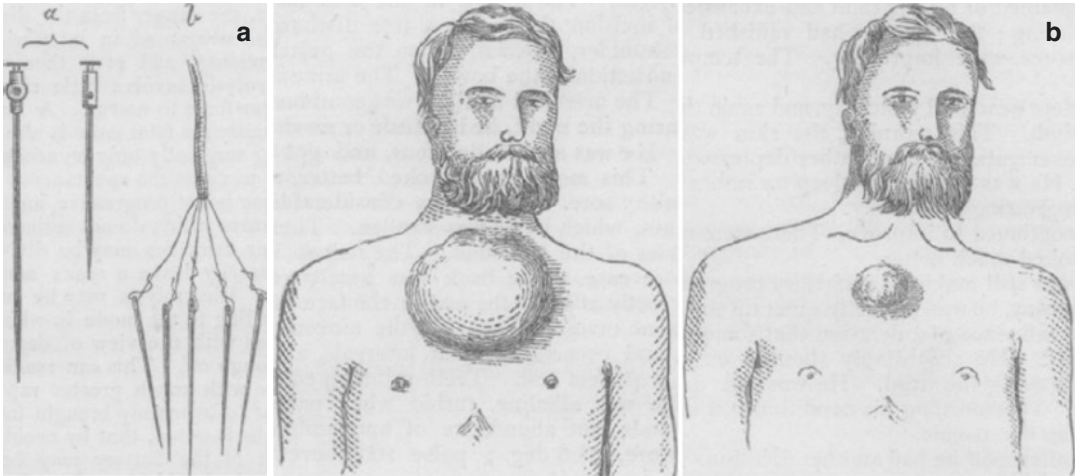


Fig. 1.18 (a–c) Electrolysis. (a) Galvanopuncture needles. (a) Insulated by vulcanite. (b) Uninsulated and multiple. The size varies with the case. (b) Aortic aneurysm

before operation (left) and the same 2 months after the operation by electrolysis (right) [99]

required to achieve thrombosis. Instead of the size of the aneurysm, he used blood velocity as a guide to the amount of wire required by heating the wire to 80 °C and estimating wire length from the difference of the diminished current required for reheating it in a second step. The rationale was that the rate of cooling of the first segment of wire inserted was relative to the velocity of blood flow (Fig. 1.19a–c) [107]. It was still in 1938 that **Blakemore** rediscovered and applied the previously described method of wire and application of an electrical current to induce thrombosis of the aortic aneurysm sac and reported 11 so treated cases with thoracic or abdominal aortic aneurysms [108].

But in general, results from wire insertion in aneurysms were poor, and the method was ultimately abandoned. Summarizing the merits of electrolysis (electrothrombosis), English surgeon **Timothy Holmes** (1825–1907) noted that “the circumstances which are favorable to a perfect success occur very rarely in practice” [109]. Words of warning also came from **David Agnew** (1818–1892), **Ransohoff**, and **Rudolph Matas** (1860–1957) who cautioned against the wires [81]. Matas even described wire insertion as “semisurgical” or “quasimedical” and regarded galvanopunctures as technique that “appeal to us

more as placebos than as real remedies” [110]. Power believed that “electrolysis seemed reminiscent of a time when little was known of the physiological processes connected with the clotting of blood” [91], and Ransohoff stated that “electrolysis fails, as a rule.” Instead, he recommended total extirpation of superficial aneurysms [102].

Very clearly, new concepts were needed.

1.5 Dawn of a New Era

1.5.1 Endoaneurysmorrhaphy

When **Rudolph Matas** of New Orleans reported an internal repair technique known as “endoaneurysmorrhaphy,” it represented a major step forward in the surgical treatment of aneurysms. He thought that aneurysms could be cured by a radical operation that would replace ligature and first performed his procedure in May 1888 on a patient with a large brachial artery aneurysm of the left arm [111]. After ligation of the proximal and distal arteries, an incision was made into the aneurysm and the clot removed. The orifices of the blood vessels that entered the sac were then sutured from within, which preserved the collateral blood supply to the extremity.

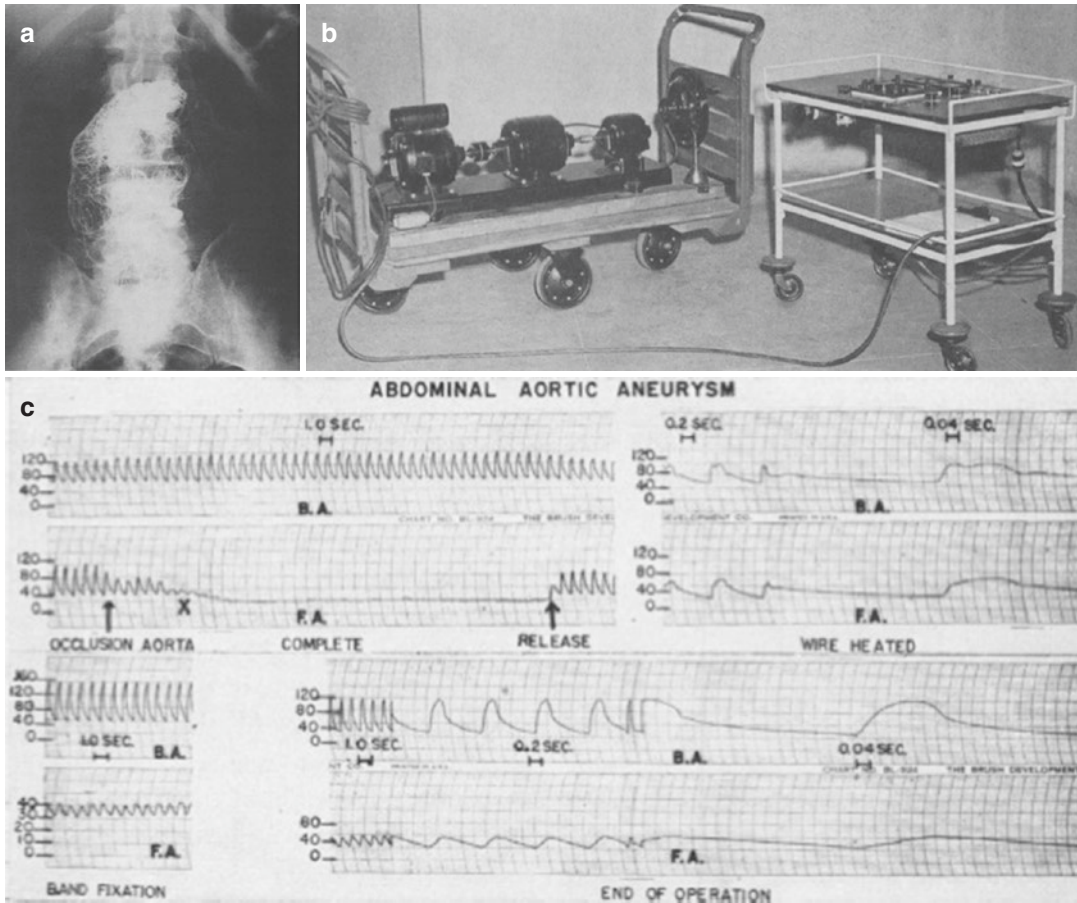


Fig. 1.19 (a–c) Progressive constrictive occlusion of the aorta with wiring and electrothermic coagulation. (a) A roentgen ray of the abdomen taken after wiring and electrothermic coagulation of a very large arteriosclerotic aneurysm. The lesion has been stabilized now in excess of 8 years since operation. Note concentration of wire at the upper aortic-aneurysm junction for its impedance effect. (b) The electrical equipment employed in electrothermic coagulation of aneurysms. The equipment used to convert

AC current to ungrounded DC current is illustrated on the left. Mounted on the portable table is an ohmmeter, ammeter, voltmeter, and ratiometer. The latter is calibrated to show the average temperature of wire imbedded within an aneurysm during heating. (c) Lilly capacitance manometric tracings taken simultaneously from the brachial artery and the femoral artery via fine plastic catheters. Note the rise in brachial artery pressure upon gradual occlusion of the aorta distal to the renal arteries [106, 107]

Matas subsequently described using obliterative, restorative, and reconstructive techniques of endoaneurysmorrhaphy (Fig. 1.20a–c) [112]. In the obliterative form (used mainly in fusiform aneurysms), sutures were placed from within the sac aneurysm so as to occlude the proximal and distal artery; the walls were sewn together to obliterate the sac [113]. The other two techniques were modifications preserving arterial patency and were used preferably in sacciform aneurysms. This could be achieved by placing a cath-

eter in the main arteries and obliterating the aneurysm sac around the catheter with sutures.

With regard to the etiology of vascular aneurysms, Matas wrote that “the sins, vices, luxuries and worries of civilization clog the arteries with the rust of premature senility, known as arteriosclerosis or atheroma, which is the chief in the production of aneurysms” [114]. There was another major leap forward in aortic surgery when in 1923 Matas successfully ligated the infrarenal aorta proximal to a large leaking luetic

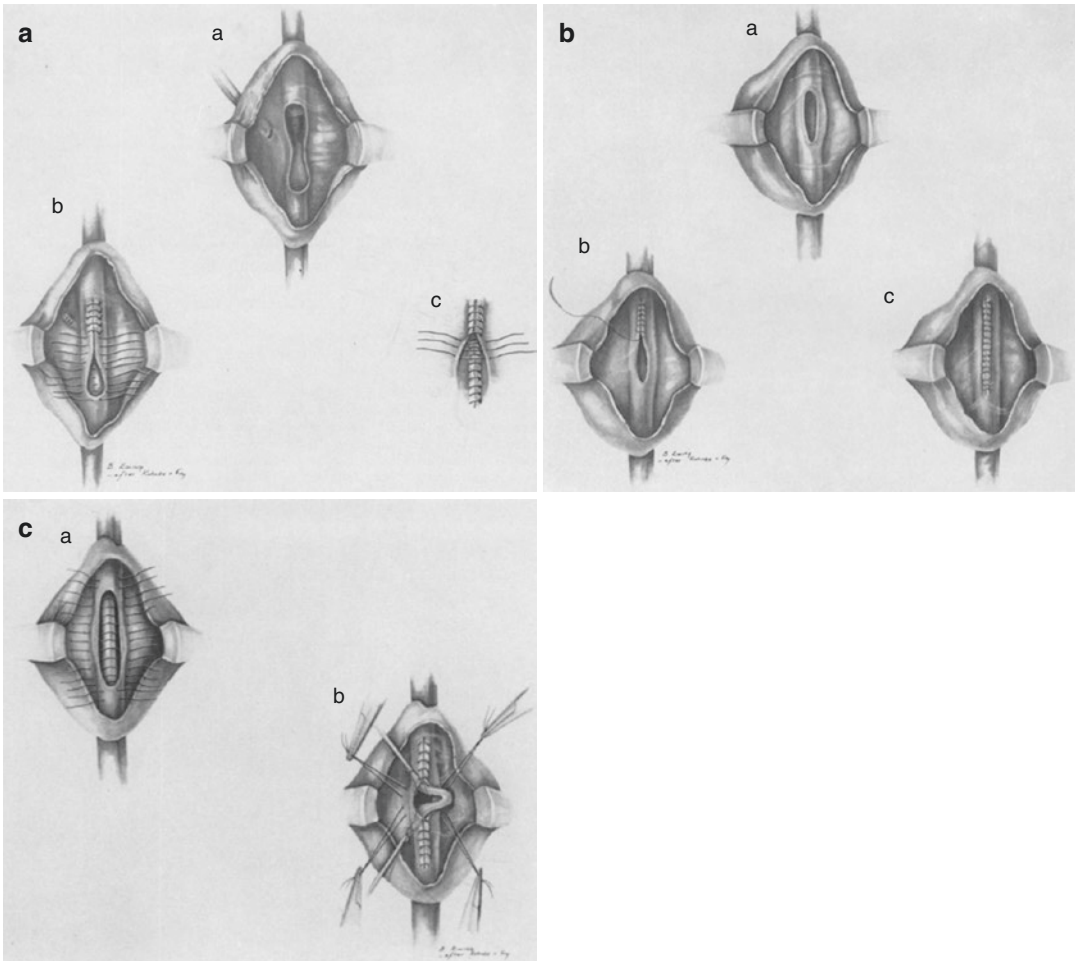


Fig. 1.20 (a–c) Endoaneurysmorrhaphy as described by Matas. (a) Technique of obliterative endoaneurysmorrhaphy. The aneurysms opened to expose orifices of the parent artery and a large branch artery (a). These orifices are closed by sutures (b), and the aneurysmal cavity is obliterated by bringing the walls together (c). (b) Technique of restorative endoaneurysmorrhaphy. The aneurysm is opened to expose the communication with the parent artery (a). This opening is closed with a continuous suture

(b). The aneurysmal cavity is then obliterated (c). (c) Technique of reconstructive endoaneurysmorrhaphy. With the aneurysm opened widely, the communication with the parent artery is closed by suture, a portion of the aneurysmal wall being used to prevent narrowing of the arterial lumen (a and b). A segment of rubber tubing is used as a guide in placing the sutures (from Ref. [112], with permission)

abdominal aortic aneurysm [115]. The patient survived nearly 18 months, and this original report, of the first successful ligation of the abdominal aorta since Cooper in 1817, later was supplemented with very detailed pictures [116]. Over the years Matas acquired vast expertise in treating aneurysms and reported his personal experience of 620 cases in 1940 [117]. Of these 101 were endoaneurysmorrhaphies, and he further emphasized the importance of testing the

collateral potential before proximally ligating an aneurysm. The reconstructive endoaneurysmorrhaphy that involved removing the diseased part and reconstructing the tunnel through the remaining healthy part was used until direct repair with graft replacement was introduced in the 1950s.

In the same year that Matas performed his first ligation on an abdominal aneurysm, French Surgeon **René Leriche** (1879–1855) stated that, “the ideal treatment of arterial thrombosis is the

replacement of the obstructed segment with a vascular graft” [118]. Later, in 1936, he advocated bilateral sympathectomy for treatment of aortic occlusive disease, but it was abandoned in due course. In 1948, he coined the term “Leriche syndrome” for occlusive disease of the terminal abdominal aorta [119].

Jose Goyanes (1876–1964) of Madrid performed the first successful replacement of a human artery (with an interposition graft from the popliteal vein) in 1906 to bridge an excised popliteal aneurysm [120]. Other surgeons such as **James Hogarth Pringle** (1863–1941) of Glasgow, **Bertram Bernheim** (1880–1958) of Baltimore, and **Erich Lexer** (1867–1937) soon followed, using saphenous vein grafts to bridge defects in popliteal and axillary arteries [121, 122].

Surgical treatment was hampered by difficult imaging and diagnosis; in fact, most cases of aortic dissection were postmortem findings [72]. Substantial advance was made with the introduction of clinical angiography with sodium iodide contrast medium by **Barney Brooks** (1884–1952), at Vanderbilt University, in 1923 [123]. **António Egas Moniz** (1874–1955) of Lisbon performed the first cerebral arteriography in 1927 (although Moniz was nominated twice for the Nobel Prize for his groundbreaking work in cerebral imaging, it was his work in psychosurgery that won him the Prize in 1949), and fellow Portuguese **Reynaldo dos Santos** (1880–1970) used translumbar aortography in 1929 (Fig. 1.21a, b). These pioneering achievements preceded today’s imaging methods and remained the only clinical tools for early diagnosis at the time.

1.5.2 Fenestration

In an effort aimed to relieve acute arterial ischemia in the lower extremities in patients with aortic dissection, **David Gurin** of Great Neck (1904–1992), **James W. Bulmer** (1892–1975), **Richard Derby** (1881–1963, husband of President Theodore Roosevelt’s daughter Ethel), and colleagues performed the first fenestration through localized reentry in the right external artery in 1935 [125]. Upon opening the vessel

through the non-dissected anterior wall, they found the true lumen narrowed by the dissection. They then incised the intima and media from within the vessel, creating an opening into the false lumen with flow into the lower extremities after removal of the clamp; closing the vessel restored pulsation in the extremity. A minor modification of fenestration was proposed by **Robert S. Shaw** (1920–2003), who opened the abdominal aneurysm sac and extracted a soft clot from its lumen, so permitting free bleeding from above, and then created a small window into the true aortic lumen [126]. Shaw also coined the term “fenestration.”

Whereas others like Matas operated on true aneurysms, the fenestration by Gurin was the first attempt to tackle acute aortic dissection. Nevertheless, flap fenestration was soon recognized to be palliative as it failed to restore the mural integrity of the ascending aorta and arch.

1.5.3 (Cellophane) Wrapping

Cellophane film was invented by the Swiss textile engineer **Jacques E. Brandenberger** (1872–1954) in 1908. It was produced as a polymer of cellulose and subsequently became an invaluable material for waterproofing products. The ability of cellophane to constrict blood vessels was first demonstrated by physiologist **Irvine Page** (1901–1991) who, besides discovering the serotonin and the renin-angiotensin system (RAS), created an experimental model of hypertension by wrapping cellophane around dog’s kidneys, as first described in 1939 [127]. In a subsequent necropsy study of the wrapped kidneys, they were found to be shrunken and encased in a dense fibroblastic and collagenous layer 4 mm thick [128]. The development of polyethylene cellophane was an important breakthrough as it produced a more intense fibrotic reaction than other types of the polymer.

Based on these observations, cellophane wrapping was further investigated by **Herman E. Pearse** (1899–1983) of Rochester using ordinary alcohol-soaked DuPont cellophane No. 300 T [129]. He demonstrated that cellophane

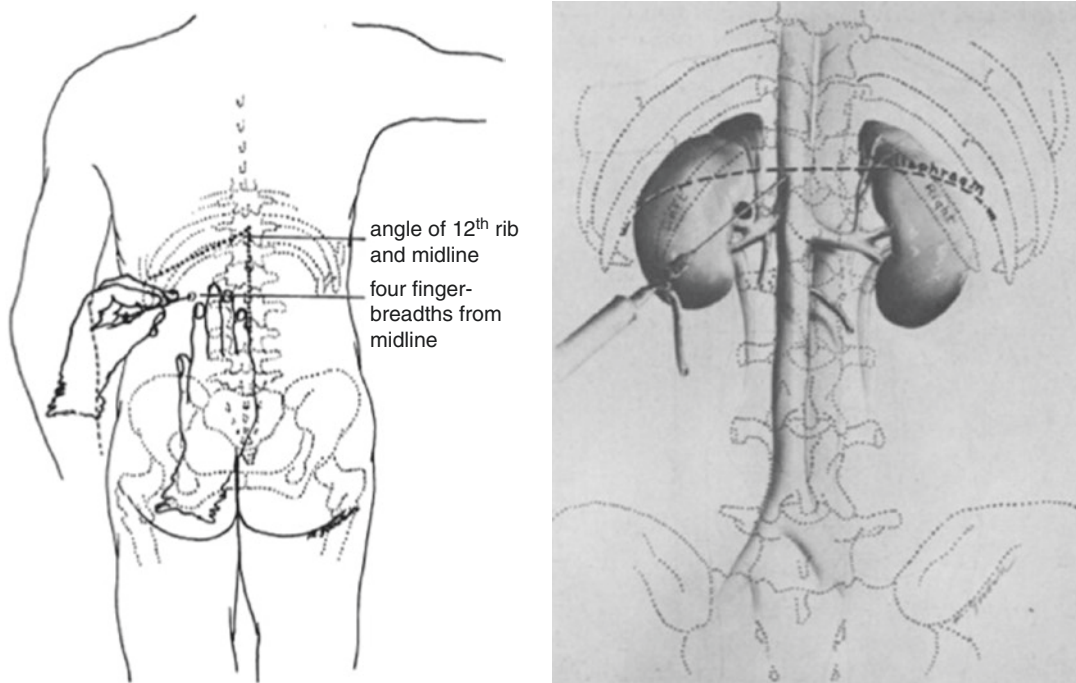


Fig. 1.21 Translumbar aortography. Left: Site of needle injection for translumbar aortography. Patient is prone. Right: Approximate area of aorta which is needed (from Ref. [124], with permission)

could gradually obliterate the lumen of blood vessels, such as the internal carotid artery or aorta in dogs. The reaction produced a partly hyalinized fibro-collagenous layer with progressive constriction and obliteration of the lumen.

Cellophane was first used clinically for aneurysms in 1943 by **Paul W. Harrison** (1883–1962) and **Jacob Chandy** (1910–2007) who successfully treated two arteriovenous aneurysms of the subclavian arteries with cellophane, resulting in their gradual elimination (Fig. 1.22) [130]. First attempts to palliate the aneurysmal dilatation of a chronic dissection of the descending aorta with cellophane wrapping were reported by **Osler Abbott** (1912–1976) [131] and **James Edgar Paullin** (1881–1951), both at Emory [132]. **W. Dean Warren** from Charlottesville (1924–1989) tried Orlon fabric (Fig. 1.23a, b) [133]; others used fascia lata (Fig. 1.24) [134], polyvinyl sponge, and dermal wrapping, but these were soon abandoned because the aneurysms grew relentlessly.

Several reports indicated that pure polyethylene cellophane was nonreactive, whereas the standard

“impure” material obtained from the primary manufacturer, E.I. DuPont Nemours Company of Wilmington, Delaware, proved highly reactive, according to **John K. Poppe** of Portland (1911–2012), who reported excellent results with the compound in treating syphilitic aneurysms [135–137].

Arguably the most prominent patient to receive cellophane wrapping for treatment of an abdominal aneurysm was physicist and Nobel laureate **Albert Einstein** (1879–1955). In December 1948, surgeon **Rudolf Nissen** (1896–1981) treated his “grapefruit-sized” abdominal aneurysm by wrapping it to induce a “foreign body reaction” potentially leading to scarring and reinforcement of the aortic wall, so limiting expansion. Einstein recovered and left the hospital to return home and continue his physics work symptom-free until he died from complications after the inevitable rupture more than 5 years later.

Later, **Michael E. DeBakey** (1908–2008) found polyethylene wrapping unsuitable as a treatment for aortic aneurysms and rejected the technique [138].

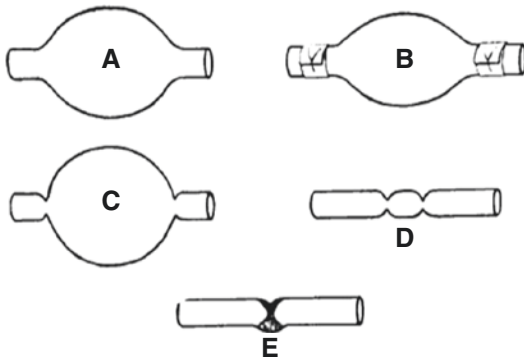


Fig. 1.22 Subclavian aneurysm cured by cellophane wrapping and fibrosis. Illustration of changes in aneurysm. Preoperative impression (A), immediately postoperative with applied cellophane (B), condition at time of first follow-up examination after 2 months (C), marked shrinkage of the aneurysm after 7 months (D), and last observation 11 months post operation (E) (from Ref. [130], with permission)

So far, the various procedures that have been proposed and used in the surgical treatment of aneurysms of the aorta have been classified into three major categories: (1) those designed to promote thrombosis and fibrotic organization by partial, complete, or gradual occlusion or ligation of the aorta, by the introduction of foreign material, or by the stimulation of periarterial fibroblastic reaction (cellophane), (2) endoaneurysmorrhaphy, and (3) extirpation of the lesion.

1.6 Working Toward Definitive Surgical Solutions

A new era in the treatment of aneurysms began in the 1950s with a shift from indirect (palliative) treatments to direct repair. The groundwork was

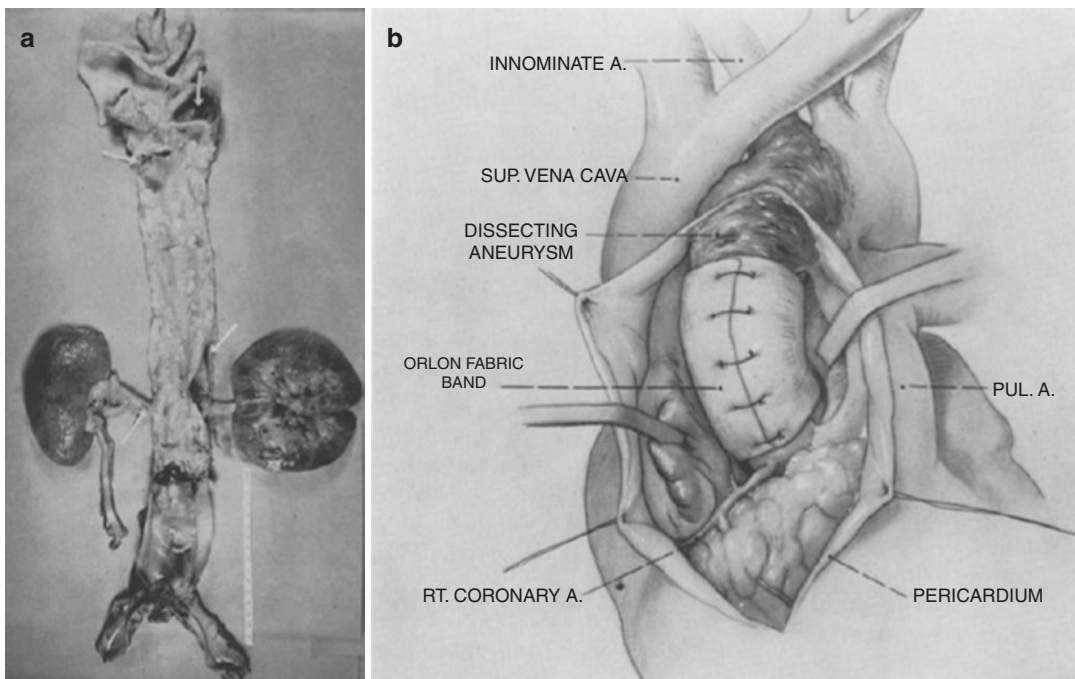


Fig. 1.23 (a, b) Aortic wrapping with Orlon fabric. Left: Photograph of the opened aorta at autopsy. Note (1) false aneurysm just below the subclavian artery, (2) transverse tear of proximal internal opening, (3) minor involvement of renal arteries, (4) Orlon prosthesis with

surrounding fibrous sheath, and small thrombus at aortic suture line. Right: A band of Orlon is applied to the intrapericardial aorta (from Ref. [133], with permission)

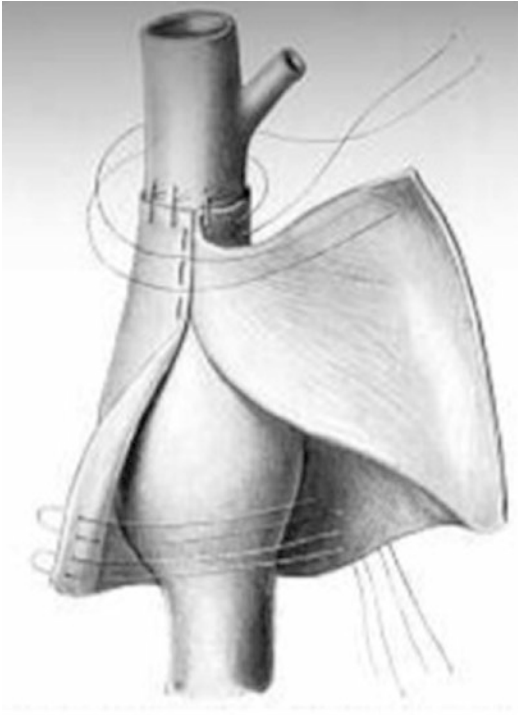


Fig. 1.24 Wrapping of the aorta. Wrapping with fascia to prevent aneurismal expansion [134]

laid by French surgeon **Alexis Carrel** (1873–1944) and **Charles Guthrie** (1880–1963), who experimented with homograft aortic substitutes and vascular anastomosis techniques in the early 1900s. In 1912, the Nobel Prize for Physiology and Medicine was awarded to **Alexis Carrel** “in recognition of his work on vascular suture and the transplantation of blood vessels and organs.” He had worked together with **Guthrie** in refining vascular anastomotic techniques for vein grafts in the arterial system, demonstrating that arterial suturing and reconstruction with xenografts were feasible. Carrel used grafts of vena cava to replace segments of the thoracic aorta in experimental animal models [139]. Recognizing the dangers of spinal cord ischemia, he used paraffin tubes as shunts for distal blood flow. Taken together, this was important and fundamental work for what was yet to come, but aortic replacement was far from reality.

It took a long time until October 19, 1944, when **Clarence Crafoord** (1899–1984) of Sweden pioneered the resection of coarctation

with the first successful end-to-end reanastomosis and restoration of continuity of the aorta [140], followed by **Robert Gross** (1905–1988), of Boston, on July 6, 1945 (Fig. 1.25a) [141], and **Harris B. Shumacker** (1908–2009) [142]. **Gross** was also the first (in 1948) to successfully replace a longer segment of a resected coarctation with a preserved arterial homograft (Fig. 1.25b) [143, 144].

Before the introduction of extracorporeal circulation in 1953, direct excisional repair of the thoracic aorta was limited to cases where side clamping was possible. The first were direct excisions of aneurysms of the subclavian artery, the innominate artery, and the aortic arch. **Denton A. Cooley** (1920–2016) did three spectacular operations as early as the 1940s, one in 1945 with clamping of the ascending aorta, excision of an eroded part, and oversewing of it. In 1949, as a resident working with **Alfred Blalock** (1899–1964), he operated on a patient who had just recently undergone coarctation resection but then developed a massive and paper-thin false aneurysm of the right subclavian artery. Blalock at the time was away, and Cooley excised the aneurysm successfully. On his return to Baltimore, Blalock remarked that, “if you are confronted with a serious surgical problem that has no proven solution, take a trip to Hawaii and your resident will handle it” [145]. And then in 1951, just having joined **DeBakey** in Houston, he had the opportunity to resect an aneurysm of the aortic arch with the same tangential clamp-and-resection technique and oversewing of the defect. In fact, this is believed to have been the first aneurysm repair of its kind (Fig. 1.26a, b) [146] and became the preferred technique for sacciform aneurysms of the thoracic aorta. **Henry Bahnson** (1920–2003) in Pittsburgh reported the first successful excision of a saccular aneurysm of the ascending aorta in 1953 [147].

First repair attempts at dissections of the descending aorta by **DeBakey** et al. were excision of the dilated part, reunion of dissected wall layers, and restoration of continuity using end-to-end anastomosis [148]. The same was attempted in the dissected ascending aorta with excision of the entry, followed by reunion of the dissected

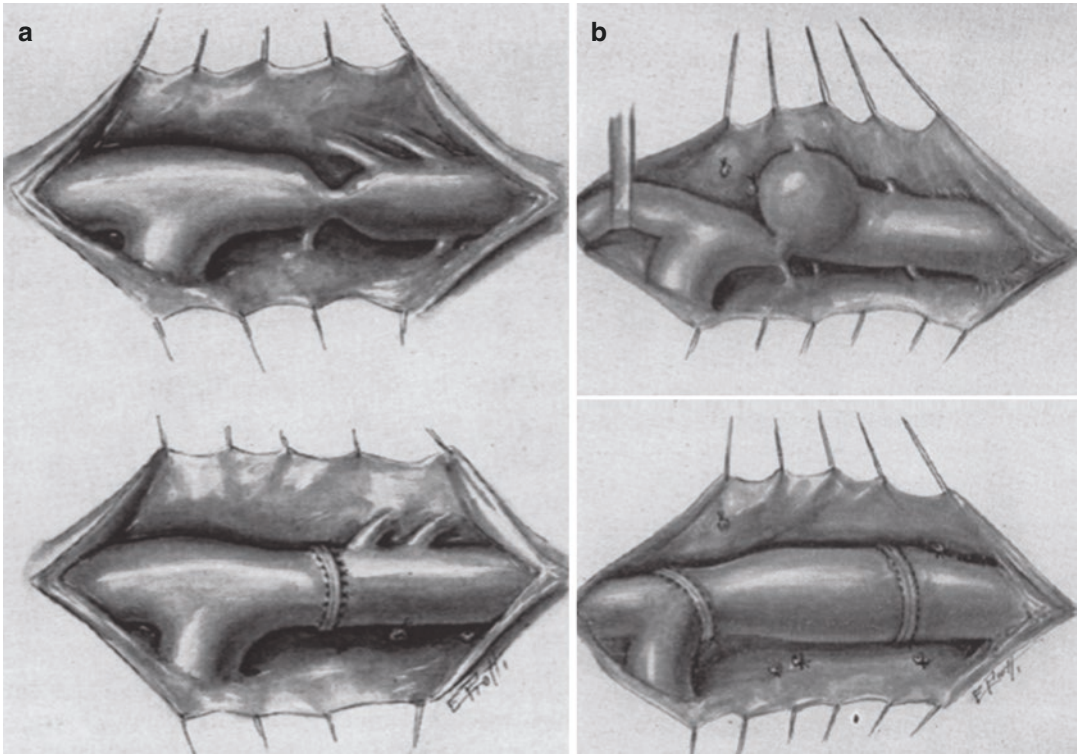


Fig. 1.25 (a, b) Treatment of aortic coarctation with homologous grafts. Left: Ideal form of therapy for coarctation of the aorta; above, thoracic aorta with a rather short zone of constriction; below, removal of the narrowed area and reconstruction of a full-sized aortic pathway by end-

to-end, everting anastomosis using interrupted mattress stitches of silk. Right: above, the findings at operation; below, complete removal of involved segment and replacement by a graft (from Ref. [144], with permission)

layers both proximally and distally and ultimately creation of an end-to-end anastomosis, as done by **Charles Hufnagel** (1916–1989) and **Peter W. Conrad** (1927–2013) [149] of Georgetown University (Washington, D.C.) and **George C. Morris** (1924–1996) in Houston [150]. Alternatively, patch reconstruction after resection of the false channel was used [151].

1.6.1 (Homo)graft Interposition

Another shift from direct excisional repair to graft replacement began with the use of cadaveric allografts (homograft) as no synthetic material was yet available. Based on early work by **Carrel** and **Guthrie** [152, 153] and **Gross** (Fig. 1.27) [154], homograft preservation was perfected, and artery banks were established in the 1940s and 1950s.

Homograft replacement of the aorta had initially been used in children with congenital heart disease after resection of aortic coarctation, first by **Henry Swan** in Colorado (1913–1996) [155], **Russel Brock** (1903–1980) in London [156], **Gross** (Fig. 1.25b) [144], and **Paul W. Schafer** (1915–) in Kansas [157].

Just after it had become clear that the 3-year survival rate for patients with untreated abdominal aortic aneurysms (AAAs) was only 50%, with two-thirds of deaths resulting from aneurysmal rupture (Fig. 1.28) [158], several surgeons independently performed successful abdominal aortic aneurysm reconstruction within just 1 month's time.

On November 14, 1950, **Jacques Oudot** (1913–1953) performed the first homograft replacement of an obstructed (thrombosed) aortic bifurcation, followed by the first crossover bypass

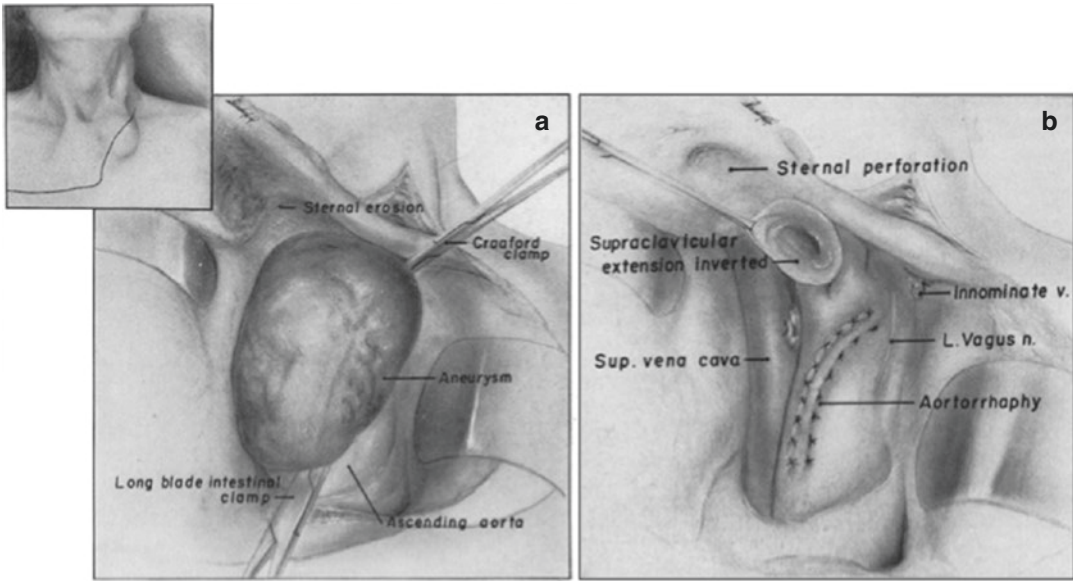


Fig. 1.26 (a, b) Aneurysmectomy (innominate artery and adjacent aorta). Left: Incision for thoracocervical approach (inset). Aneurysm arising in the innominate artery at its origin from the aortic arch. A Crawford clamp has been applied tangentially across the superior border of the aortic arch to occlude the origin of the innominate artery and aneurysm. Distal control of circulation in the

aneurysm is obtained by temporary occlusion with tape around the right common and subclavian arteries. Right: Lateral aortorrhaphy following excision of aneurysm. The supraclavicular extension of the aneurysm through the eroded manubrium has been inverted and the thrombus evacuated (from Ref. [146], with permission)

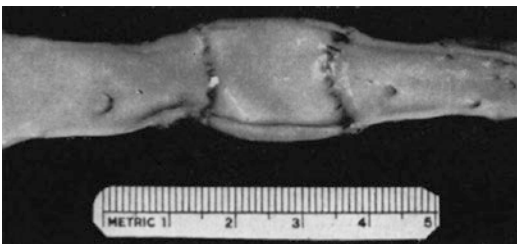


Fig. 1.27 Early homograft preparation and implantation experiments. Graft of abdominal aorta (dog to dog). The graft section had been stored in 10% homologous serum and balanced salt solution for 6 days and then had been implanted into a recipient animal which was kept for 6 months before sacrifice (from Ref. [154], with permission)

in the same patient with insertion of a graft between the two external iliac arteries [159]. Autopsy of the patient 3 years later revealed a thrombosed homograft [160]. On February 26, 1951, **Norman Freeman** (1903–1975) and **Frank Leeds** (1914–2003) in San Francisco successfully treated an aortic aneurysm with a vein inlay autograft from the left common iliac vein

sutured into the abdominal aorta and iliac arteries and then wrapped the aneurysmal sac around the reconstruction for external support of the vein graft [161, 162].

And on March 2, 1951, **Paul W. Schafer** and **Creighton A. Hardin** (1918–2013) in Kansas resected an abdominal aneurysm with an indwelling polyethylene bypass shunt after clamping the aorta and replacing it with a human homograft. The patient died after 29 days from a leak in the native aortic wall [157]. **Freeman** reported reestablishing circulation in the legs with a splenoiliac anastomosis as an extra-anatomic bypass technique [163]. But likely most memorably, on March 29, 1951, French surgeon **Charles Dubost** (1914–1991) successfully resected an abdominal (infra-renal) aortic aneurysm with a 15-cm-long homograft replacement via a left extraperitoneal approach (Fig. 1.29) [160, 164–166]. The patient survived for 8 years. The report from Paris in 1951 that an abdominal aortic aneurysm had been successfully resected greatly influenced surgeons throughout the world who, until then, had

Fig. 1.28 Survival rates for patients with abdominal aortic aneurysm. Survival rates for traced patients who had abdominal aortic aneurysm as compared to the survival rates of the normal population of age 65 years (from Ref. [158], with permission)

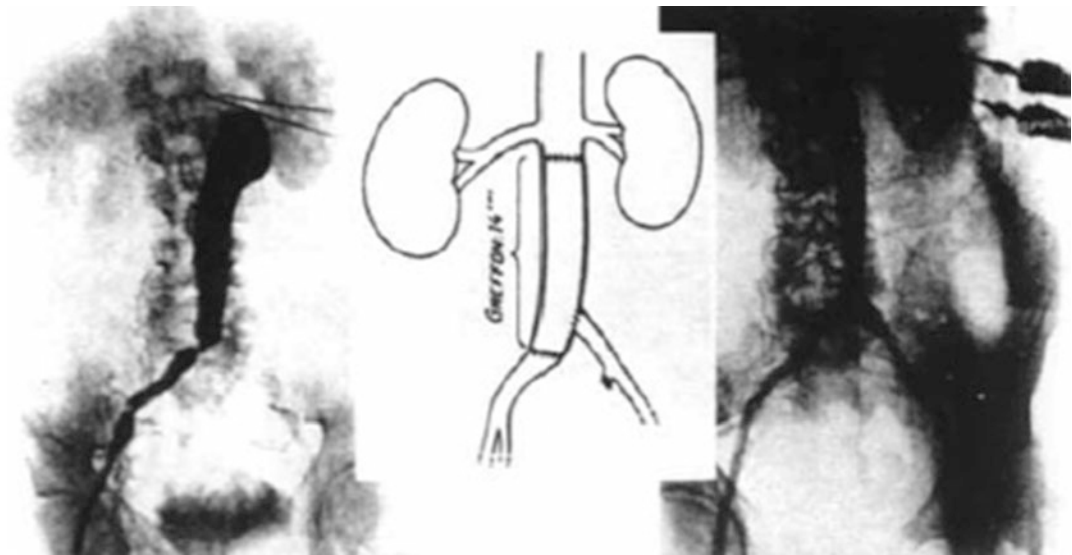
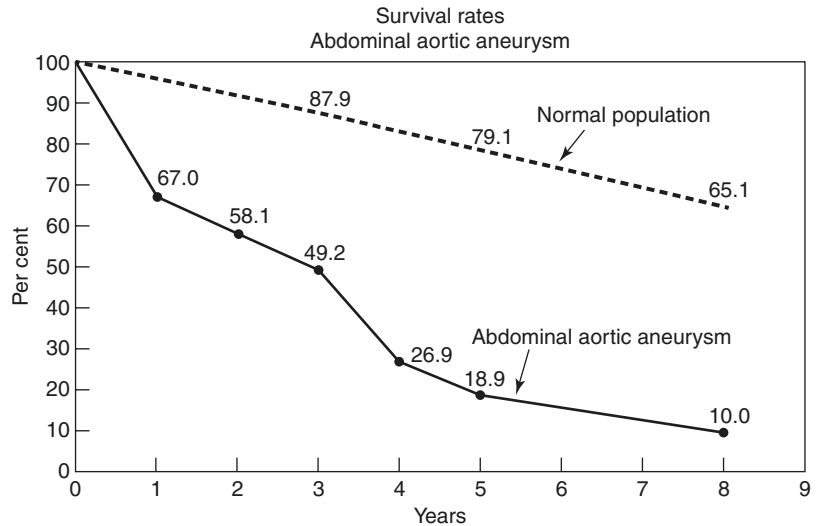


Fig. 1.29 Charles Dubost. Diagram of Dubost's first aortic aneurysm replacement with homograft (March 29, 1951) (from Ref. [160], with permission)

regarded such an operation as being beyond the limits of surgery.

Although the first procedure by **Schafer** and **Hardin** resulted in the patient's death after 29 days due to hemorrhage from a leak [157], the following operations by **Dubost** [164], **Ormand Julian** (1913–1987) in Chicago [167], **Brock** [168], **DeBakey** and **Cooley** [169], and **Bahnson** [147] were successful. Likewise, ruptured abdominal aneurysms were successfully treated

between March 1953 and December 1954 by **Bahnson** [170], **Frank Gerbode** in Stanford (1907–1984) [171], **Cooley** and **DeBakey** [172], and **Hushang Javid** in Chicago (1921–) [173].

The first replacement of a descending thoracic aortic aneurysm with resection and homograft replacement was performed on April 2, 1951 by **Conrad Lam** (1905–1990) in Detroit, and the patient survived for 3 months before succumbing to infection [174]. During the procedure, blood

flow distal to the operative site was maintained through a polyethylene tube inserted into the lumen of the vessel above and below the aneurysm (Fig. 1.30a, b). Lam concluded that leaving the aneurysmal sac intact as Matas suggested predisposes to infection, and full resection would be the preferable technique.

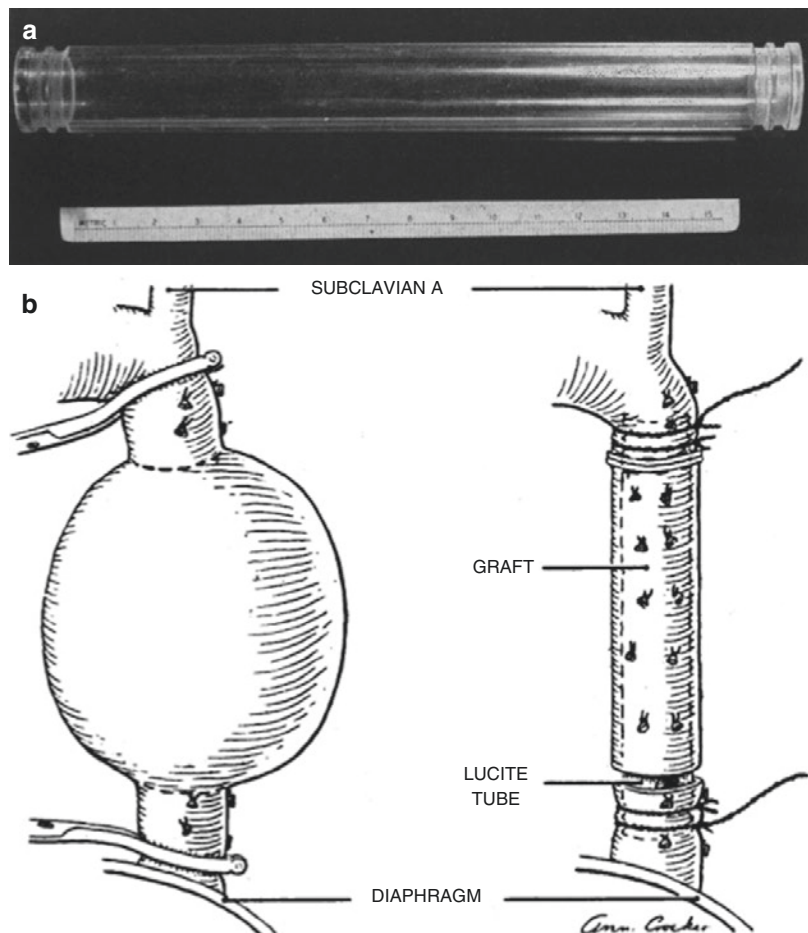
DeBakey and **Cooley** had removed abdominal aneurysms completely in their first patients in 1952 [169]. Removal was also strongly advocated by **Bahnson** in 1953 [147], and it became clear that resection and complete replacement of the diseased aorta would eventually be the ultimate treatment of choice. **DeBakey** and **Cooley** had been developing techniques for complex aneurysm repair and spinal cord protection during thoracic surgery for some years prior, and they performed a successful resection and seg-

mental graft replacement for fusiform aneurysms of the descending aortic aneurysm on January 5, 1953 [175], followed by **Shumacker** and **Harris** in 1956 [176].

The next major breakthrough took place in 1954, when the Houston team performed a series of successful surgical treatments of dissecting thoracic aortic aneurysms (Fig. 1.31) [177]. **DeBakey** and his associates went on to accumulate vast clinical and surgical experience in the management of AD patients, reporting a 20-year follow-up of 527 surgically treated patients as early as 1980 [178]. Ironically, Michael DeBakey himself underwent and survived open surgery for type A aortic dissection at the age of 97, arguably the oldest patient in history to do so.

Ascending aortic replacement required the development of cardiopulmonary bypass and was

Fig. 1.30 (a, b) Resection of the descending aorta and replacement with homograft. (a) Lucite tube used to conduct blood through the graft during the suturing. (b) The operative procedure for resection and replacement of the descending aorta (from Ref. [174], with permission)



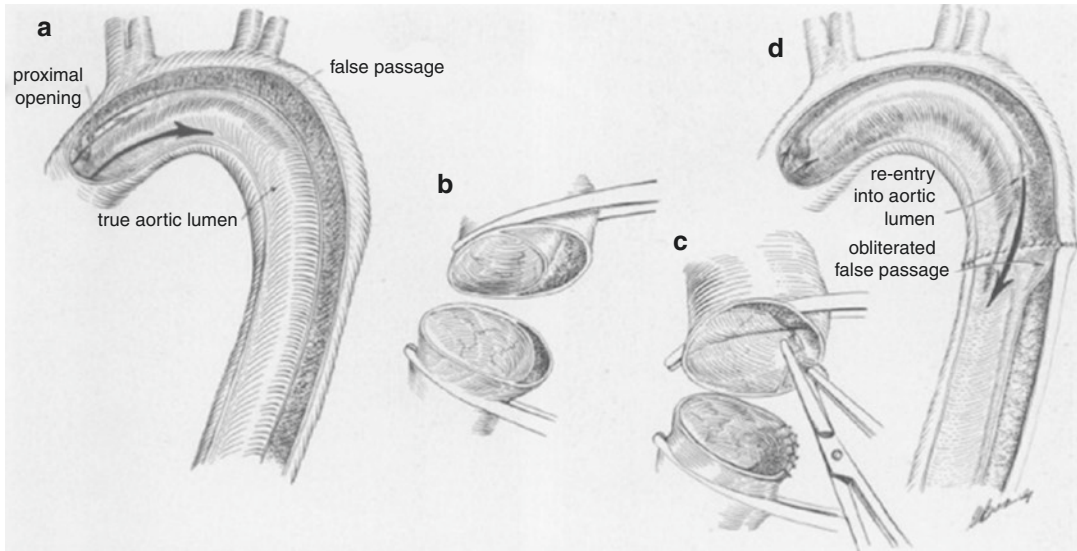


Fig. 1.31 Surgical treatment of dissecting thoracic aortic aneurysm. Illustration showing the site of origin and extent of the dissecting process in the thoracic aorta (a). The aorta has been divided (b), the false lumen has been

obliterated distally (c), and proximally a segment of the inner layer is being excised to create a reentry passage. The anastomosis is completed (d) (from Ref. [177], with permission)

first performed in 1956, again by **Cooley** and **DeBakey** [179], successfully replacing the ascending aorta with a homograft. Replacement of the aortic arch, with its inherent risk of cerebral ischemia, was understandably more challenging. **Schafer** and **Hardin** in 1951 [157] and **Cooley, Mahaffey, and DeBakey** in 1955 [180] failed in performing arch replacements using bypass shunts and hypothermia only. It was only with cardiopulmonary bypass that **DeBakey** and colleagues were first able to successfully replace the aortic arch as reported in 1957 [181].

By now, every section of the thoracic aorta from the arch to the diaphragm had been resected successfully and replaced by homografts [182]. Enthusiasm for homografts had swelled, and use was widely accepted in the early 1950s but then waned because of short supply and difficulty with the harvesting and banking of the grafts but foremost because of frequent structural degeneration and late complications of the grafts [182]. In fact, short-term results up to 3 years had been gratifying, but long-term outcome with homografts was poor, and aorta banks began to be closed [144]. At this point it was obvious that further progress would not be possible without a suitable flexible

conduit to replace resected segments of the aorta, and a search was begun for a more stable, long-term, synthetic conduit material.

1.6.2 Synthetic Grafts

The use of prosthetic grafts leads to a new standard of care, starting with **Arthur Voorhees** (1921–1992), who made his momentous contribution in 1952 using a vinyon-N cloth as a plastic arterial substitute, and ending with **Michael DeBakey** and **Denton Cooley** who refined the design of the Dacron graft in 1954.

Arthur Blakemore was known for performing portacaval shunts in patients with portal hypertension and developed the **Sengstaken-Blakemore tube** for management of hemorrhaging esophageal varices [183]. He was used to depressing cases with copious bleeding (venous and arterial) and is quoted as having said, “The only time I worry about bleeding is when I can hear it” [184]. When dealing with arterial occlusive disease, he tried injections of vasodilator drugs and performed lumbar sympathectomies but was frustrated over abdominal aortic

aneurysms, having tried wires in the 1930s [108], also using the Colt apparatus. Now in the 1950s, **Blakemore** was involved in discovering a suitable graft material for aortic surgeries. Working in his animal laboratory in 1947, **Voorhees** incidentally discovered that a silk suture inadvertently left in a ventricular cavity of an animal was covered with a slick layer resembling natural endocardial tissue cells and speculated that “a piece of cloth might react in a similar way” [185]. He was unaware of **Julius Dörfler** (1872–1952) [186] and **Herbert W. Carson** (1870–1930), who had observed earlier that silk sutures left in the lumen of an artery become encapsulated by a fine veil-like coating [187], and of **Guthrie**, who 30 years earlier had suggested that an implant need serve only as scaffolding for ingrowth of host tissue [188]. But as these findings went largely unnoticed, the important step was made when Voorhees proposed the concept of a fabric tube that “had to be strong, inert, stable, of the right porosity, supple, and yet easily transversed by a fine needle” [185]. The idea was that a fine mesh cloth could be used as an arterial graft, with fibrin plugs forming to stop leakage of blood through the walls of the prosthesis [189].

His first artificial artery was fashioned from a silk handkerchief. Next he turned to a bolt of

vinyon-N cloth that worked even better. According to another source, an orthopedic resident, **James Wallace Blunt** (1918–2003), offered Voorhees the vinyon-N cloth after it had failed as a tendon replacement. It had originally been designed as sail or parachute cloth but proved too inert to hold dye. Voorhees constructed a tube resembling the silk model and began using it as an aortic prosthesis in dogs in demanding and tedious procedures. By the end of 1950, 30 dogs had received implants with satisfying early patency, and in 1951 he had enough material to publish an optimistic preliminary report [189]. Pore size turned out to be critical for ingrowth of fibroblasts, and without the latter, neo-endothelium could not form. In February 1953, **Blakemore** at Columbia Presbyterian Medical Center used a vinyon-N graft from his lab to replace a ruptured abdominal aneurysm, only because the local homograft bank was unable to supply material. It became the first synthetic graft ever used to replace the human aorta [184]. The outcome encouraged further implantations, and over the following year, 16 additional aneurysms were similarly treated with a 56% survival rate (Fig. 1.32) [190].

Nevertheless, vinyon-N rapidly gave way to competitive fibers with more favorable physical

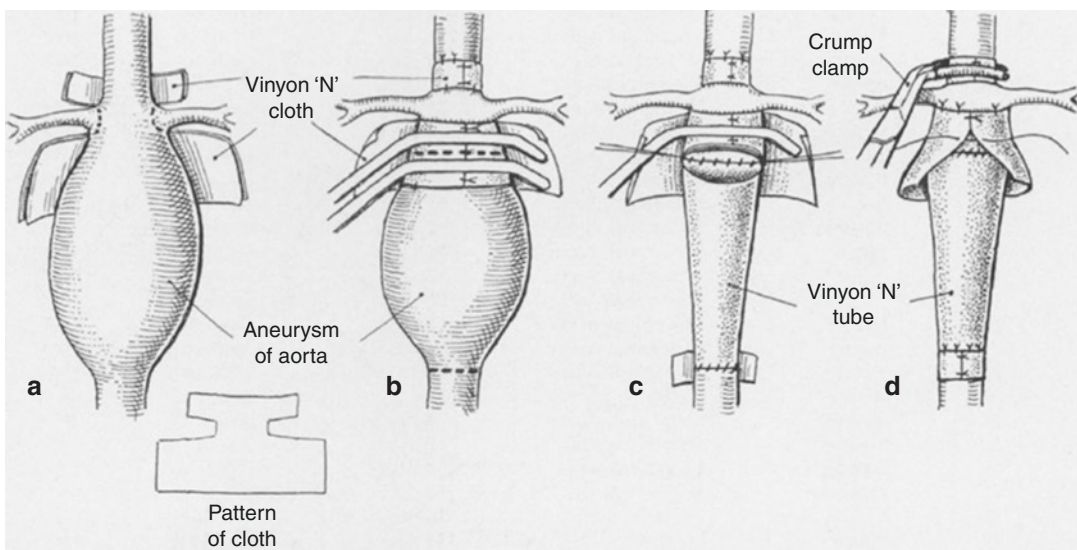


Fig. 1.32 Aortic tubes constructed from vinyon-“N” cloth. Use of reinforcing cuffs over the proximal aortic segment and reinforcing strips about the line of anastomosis (from Ref. [190], with permission)

properties, including Orlon, Teflon, Ivalon, Nylon, and finally Dacron. **Norman Shumway** (1923–2008) at Stanford University experimented with rolled sheets of polyvinyl sponge (Ivalon) [191], and **Shumacker** used layered Nylon, incorporating a thin polyethylene film for hemostasis [192], whereas the Houston group used braided Nylon tubes experimentally (Fig. 1.33) [193]. To properly respect these early achievements, one must realize that prostheses were far from being delivered perfectly and manufactured in all sizes. Instead, “tubes were cut and sewn in scrub rooms, ... unsophisticated and often cranky” [194]. With all these fabrics, durability remained a problem. Some fibers deteriorated rapidly, while others failed to form a strong bond with surrounding tissues.

While **Crawford** in Houston worked on a technique for freeze-drying human arteries taken from autopsies, in 1954 **W. Sterling Edwards** (1920–2004) was inspired by Voorhees’ enthusiasm for synthetic cloth for arterial grafts. Telling one of his patients, who happened to be an execu-

tive at Chemstrand Corporation, about his difficulties with creating easy-to-sew and wrinkle-free nylon grafts, the patient helped set up a collaboration with a physical chemist at the company [195]. They soon introduced the concept of crimping cylindrical grafts to allow greater flexibility without kinking and to provide better handling characteristics [196]. The Edwards-Tapp braided nylon graft was manufactured by US Catheter and Instrument Corp., until Edwards switched his preference from nylon, with its disappointing durability and degeneration in the phase of body fluids, to Teflon because of its superior tensile strength profile. Teflon prostheses remained commercially available until 1979.

However, it was the discovery and introduction of Dacron that opened a new chapter.

1.6.3 Dacron

Tubes of various plastic materials were employed, but all were found to have certain

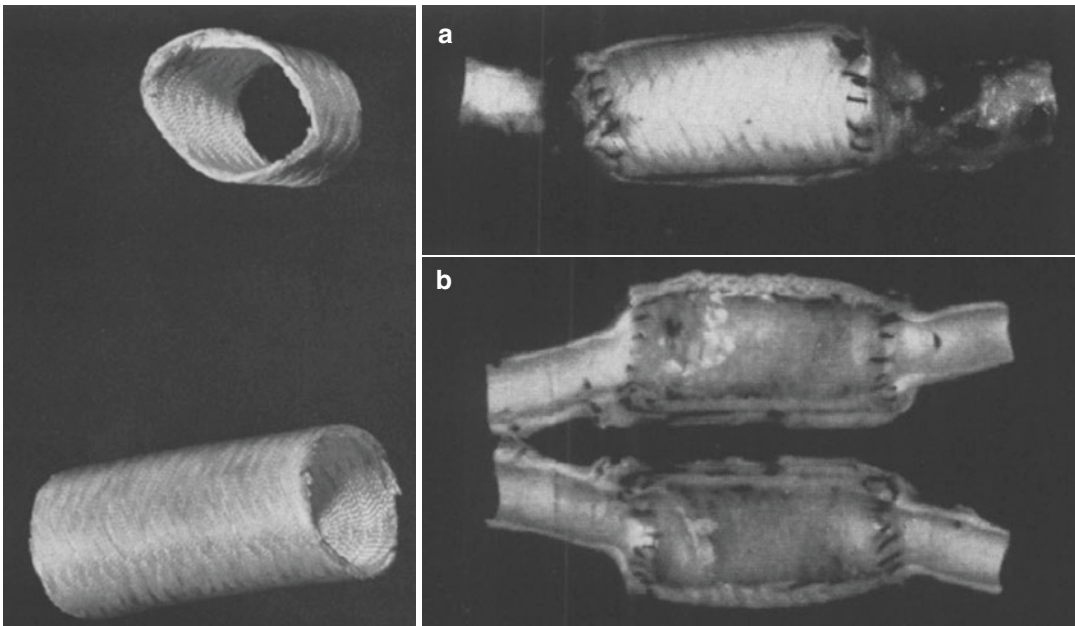


Fig. 1.33 Braided Nylon tube for implantation into the thoracic aorta. Left: Braided Nylon tube for implantation into the thoracic aorta. Right: Braided Nylon prosthesis 196 after implantation into the thoracic aorta (dog). (a) The outer connective tissue sheath has been peeled away

from prosthesis. (b) Longitudinal section of prosthesis and adjacent aorta showing loosely adherent, outer fibrous connective tissue sheath and the smooth adherent inner lining (from Ref. [193], with permission)

disadvantages owing to design and physical characteristics that limited their adaptability and hindered their practical application. Beginning in 1954, **DeBakey** and his group began to experiment with Dacron. This material was a polyester polymer that was developed around 1939 and had been introduced in the USA by E. I. DuPont de Nemours and Company, Inc., in 1946. According to legend, DeBakey discovered the material in a department store more or less by accident when he was actually looking for nylon, but it was sold out and the clerk suggested Dacron instead. Eventually DeBakey came to prefer this textile and used it to create the first artificial arterial patches and tubes using his wife's sewing machine (Fig. 1.34).

After 2 years of testing on animals, **DeBakey** was satisfied with Dacron tubes that were easier to sew than vinyon [197, 198]. Cooperation with the Philadelphia College of Textiles and Science led to the development of a knitting machine capable of producing seamless knitted (instead of braided) Dacron grafts in various sizes and with bifurcations, made flexible by proper crimping [139, 199].

The new material was now widely used by the group around **DeBakey**, **Cooley**, **Morris**, **Oscar Creech** (1916–1967), and **Crawford**. In fact, clinical experience was so highly gratifying that DeBakey employed this graft exclusively. Within less than 4 years, the group had implanted more than 1000 synthetic grafts with a 90% success

rate, and this new arterial substitute was introduced to the medical community in 1958 with the landmark paper reporting their highly satisfactory results [200]. **DeBakey** et al. had collected 803 cases of occlusive disease of the aorta and iliac and femoral arteries including 448 cases with aortoiliac (complete and incomplete) occlusion. At first, the group had also routinely performed lumbar sympathectomy as was standard at the time as a supplemental procedure but gave up on it because of the high incidence of distressing post-sympathectomy neuralgia. Ultimately, flexible knitted **Dacron** tubes were judged to be the best arterial substitute available and came into wide use [200].

A review of chemical and physical data as well as in vivo experiments on a wide range of fabrics in 1955 concluded that “Dacron appeared to have the most desirable qualities in the overall evaluation” and was thus the best material for aortic substitution [198]. In 1956, vinyon-N was no longer commercially available, and both nylon and Orlon exhibited significant loss of tensile strength over time [201].

The **Meadox Weaving Corp.**, an upholstery and drapery fabric manufacturer in New Jersey, collaborated with **Ormond Julian** and **Ralph Deterling** (1917–1992) of New York to design and fabricate grafts. Beginning in 1954, they produced the first woven grafts, and in 1961, Meadox Medical Inc. teamed up with **Cooley** to produce a graft line carrying his name [202]. Bleeding control remained an issue, particularly in the fully heparinized patient undergoing CPB. Grafts needed to be tightly woven with low porosity but at the cost of less desirable handling and suture characteristics. **Cooley** introduced the method of autoclaving a porous graft soaked with autologous plasma, which renders it completely impervious to blood. Better sealing later became available, including impregnation with bovine collagen or albumin.

Since **Cooley** and **DeBakey**'s first successful replacement of the ascending aorta with a tube graft [179], this has become the standard procedure for dealing with dissecting aneurysms and chronic nondissecting dilatations.



Fig. 1.34 Michael DeBakey at home sewing a Dacron vascular graft (c. 1955)

1.7 The Aftermath

The ground had now been laid by the availability of a reliable substitute and the concept of complete removal of the diseased segment. Since then, too many technical advances have been made to cover them all, and to mention the names of the many who have contributed to our current understanding of aortic disease and our management concepts would be beyond the scope of this survey.

Clearly, the most fundamental advancement was the development and introduction of the cardiopulmonary bypass by **John H. Gibbon** (1903–1973) in 1953 (Figs. 1.35 and 1.36) [203–209]. **DeBakey**'s contribution, while still in medical school in 1932, had been to assemble a hand-cranked roller pump, first used to transfuse blood directly from a donor to a patient and later adapted for use in the heart-lung machine. In 1957 **Cooley** introduced the left heart bypass to replace the descending aorta [210].

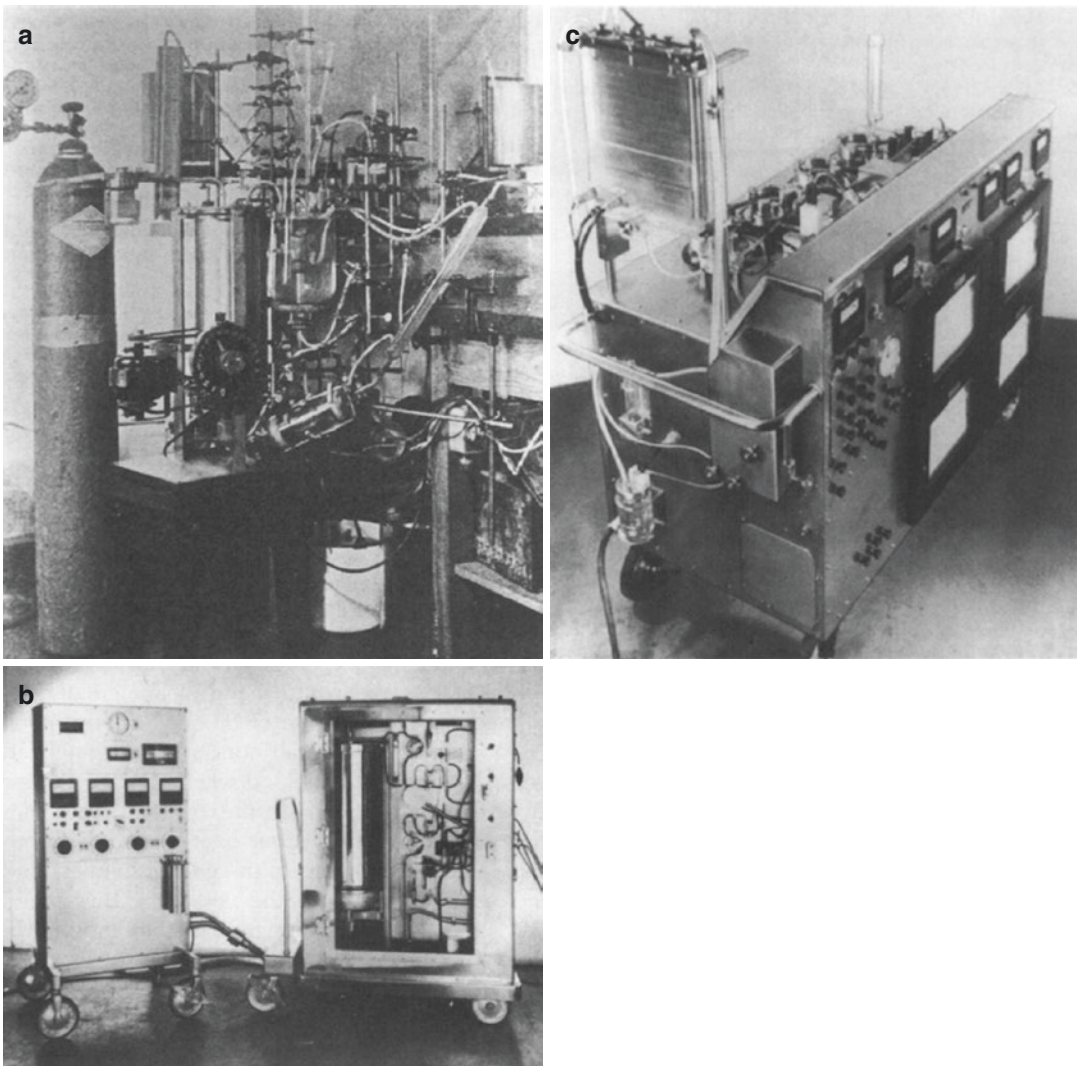


Fig. 1.35 (a–c) John Gibbon's heart-lung machine. (a) Equipment used by John Gibbon in early laboratory experiments in extracorporeal circulation. (b) Heart-lung

machine Gibbon Model I (1949). The first oxygenator built by IBM. (c) Heart-lung machine Gibbon Model II (1951) (from Ref. [207], with permission)

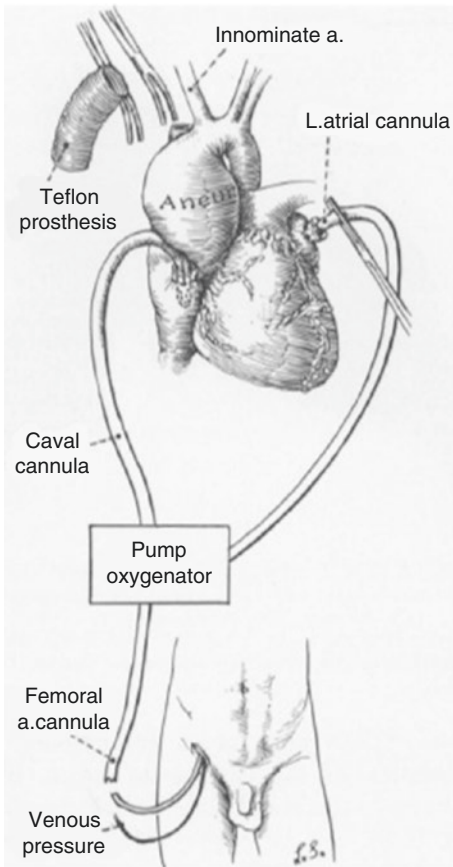


Fig. 1.36 Cardiopulmonary bypass. Plan of cardiopulmonary bypass used for prosthetic replacement of the ascending aorta (from Ref. [208], with permission)

Routine use of cardiopulmonary bypass greatly simplified aortic surgery allowing, among other things, controlled (deep) hypothermia and brain perfusion. **Wilfred G. Bigelow** (1912–2005) of Toronto developed the idea of reducing a patient's body temperature before an operation to lower metabolism and oxygen need [211]. After basic research in animal models, **Floyd John Lewis** (1916–1993) at the University of Minnesota performed the first successful human open-heart operation (September 2, 1952), closing an atrial septum defect in a child, after inducing hypothermia by wrapping the child in cooling blankets [212]. Notably, this and subsequent operations, also by **Henry Swan**, who carried on with this technique, were performed without cardiopulmonary bypass in large patient series. As mentioned

before, **Cooley** failed in replacing an aortic arch using bypass shunts and hypothermia only [180], and a solution for organ protection, particularly the brain, remained paramount. After cardiopulmonary bypass became widely available, **C. Walton Lillehei** (1918–1999), who had participated in Lewis's historic operation, and **John W. Kirklin** (1917–2004) in Rochester observed spontaneous cooling in patients undergoing surgery with beneficial consequences. Rather than relying on this "side effect," **Will C. Sealy** (1912–2001) at Duke University introduced the heat exchanger to the DeWall oxygenator for controlled induction of hypothermia and rewarming [213], progressively allowing more complex and time-consuming procedures. **Donald Ross** (1922–2014) and **Brock** in London advocated the use of deep hypothermia induced with the heart-lung machine [214] and so stimulated wider interest in further study of this technique.

The breakthrough for wide acceptance came with the work of **Randall B. Griepp** (1940–) and colleagues at Mount Sinai in New York. The introduction of deep hypothermic circulatory arrest (DHCA) in the mid-1970s dramatically reduced the incidence of neurological damage following aortic surgery [215]. Griepp, however, also stressed the limits of hypothermic circulatory arrest for cerebral protection [216].

Subsequently, the **Crawford** [217, 218] and **Cooley** [219, 220] groups used deep hypothermia for arch interventions and also suggested using moderate hypothermia for open repair of proximal aortic arch anastomoses [221]. **Nicholas Kouchoukos** (1937–) in St. Louis pioneered the use of profound hypothermic circulatory arrest for repair of descending thoracic and thoracoabdominal aneurysms [222, 223]. The combination of cardiopulmonary bypass and total circulatory hypothermic arrest provided a major advance that greatly enhanced the safety of distal aortic procedures.

The use of cerebral perfusion was reconsidered and then revived by **William H. Frist** (1952–) and colleagues [224]. Subsequent advances focused on improving brain protection by defining an approximately 30-min time limit for circulatory arrest [225], which could be

extended with cerebral perfusion techniques such as uni- and bilateral retrograde (RCP) and antegrade cerebral perfusion (ACP) [226].

To simplify clinical management of aortic dissections, many classification systems were suggested, but only the Stanford and DeBakey nomenclatures have prevailed over the time. The “Stanford classification” differentiates among aortic dissections based on whether the ascending aorta is involved, regardless of the site of tear and irrespective of the distal extent of dissection [227]. The “DeBakey classification” [228], which was modified in 1982 to more closely resemble the Stanford classification [178], classifies dissections not involving the ascending aorta as type III; those limited to the ascending aorta are DeBakey type II, and dissections involving the ascending, arch, and descending aorta are classified as type I.

Treatment of aortic dissection was greatly influenced by **Myron W. Wheat** (1924–2012) and others who evaluated the merits of open versus pharmacological management against the background of persistently high operative mortality. In contrast to aortic dissection with involvement of the ascending aorta, the majority of patients with uncomplicated type B aortic dissection treated medically were found to survive the acute phase, thus giving rise to medical therapy rather than surgery [229, 230]. Management of patients with type B dissection was fundamentally modified and later came to include interventional treatment modalities. **Wheat** in 1965 also emphasized the role of blood pressure control in the medical management of acute aortic dissection [229], still the mainstay for aortic dissections in absence of complications.

Treatment of proximal aortic dissection with concomitant valve insufficiency was managed by narrowing of the annulus and valve bicuspidalization [133] and with the concept of commissural resuspension and attenuation of the sinotubular junction [149, 231]. **Ross**, in 1962, and **Sir Brian Barratt-Boyes** (1924–2006) in 1964 successfully implanted the aortic homograft in the orthotopic position [232, 233].

Albert Starr (1926–) in 1963 excised the incompetent aortic valve in aortic root aneurysm, replacing it with a Starr-Edwards valve and

replacing the aneurysmal ascending aorta with a graft [234]. In 1964 **Wheat** reported the first successful replacement of the entire ascending aorta including the valve with a separate Starr-Edwards valve and a woven Teflon aortic prosthesis; a flap of aortic tissue around the coronary ostia was left to incorporate into the graft [235].

Some patients, however, required replacement of the aortic root as well. Subsequently, combined operations were introduced that replaced the ascending aneurysm in conjunction with replacement of the aortic valve and reimplantation of the coronary arteries. In 1968, **Hugh Bentall** (1920–2012) at Hammersmith Hospital and **Anthony De Bono** reported their technique for complete replacement of the ascending aorta, using a composite mechanical valve and a Dacron conduit with reimplantation of the coronary ostia (Fig. 1.37) [236]. In cases with ascending aortic aneurysms with associated functional aortic insufficiency (but otherwise normal cusps), “aortic valve sparing operations” were developed with the aim of preserving the native aortic valve. These procedures are known as the “reimplantation” technique as introduced by **Tirone E. David** (1944–) of Toronto [237, 238] and the “remodeling” technique as described by **Sir Magdi Yacoub** (1935–) in London [239, 240] in the early 1990s.

Surgery on the aorta, except for its arch portion, had become well established in the 1960s, including introduction of the “island technique” of brachiocephalic vessel reattachment, which simplified the procedure and reduced the number of anastomoses required [241]. However, the risk of multiple-stage operations required for the frequently encountered aneurysms extending distally from the aortic arch remained a problem. In 1983, **Hans Georg Borst** (1927–) in Hannover introduced the two-stage elephant trunk principle to simplify the second stage by leaving an extended vascular graft free within the descending aorta during the first-stage operation [242, 243]. The technique was then refined [244] and later complemented with the “frozen elephant trunk” technique to allow repair of concomitant aortic arch and proximal descending aortic aneurysms in a single-stage procedures with a

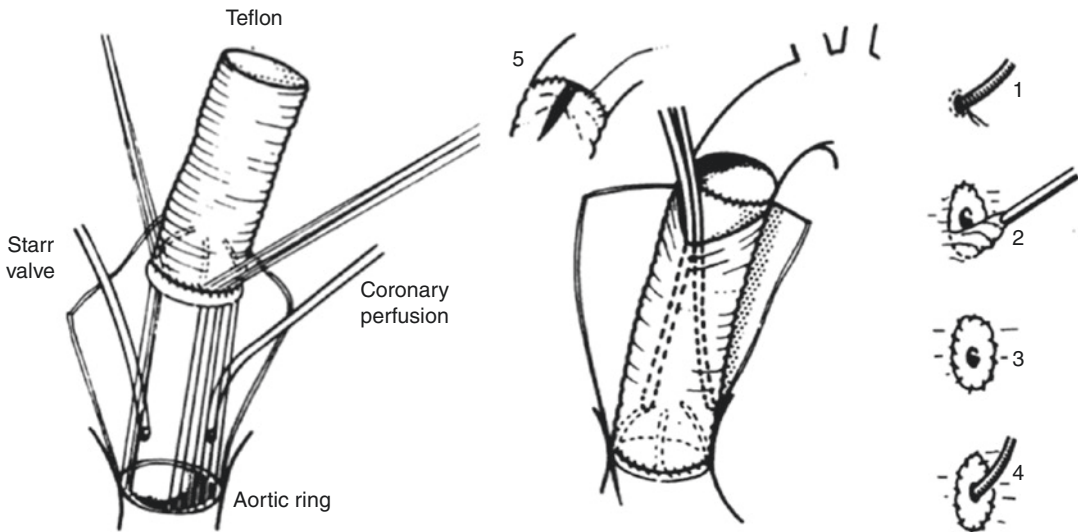


Fig. 1.37 Complete replacement of ascending aorta (Bentall and De Bono). Left: Starr valve has been sutured to aortic prosthesis: sutures have been placed in aortic ring before fixing the combined prostheses. Right: Combined prostheses in situ. Insets 1–4 show details of holes fash-

ioned in the side wall of the Teflon tube to reincorporate the coronary ostia within the lumen of the new ascending aorta. Inset 5 shows the vertical slit in the prosthesis (from Ref. [140], with permission)

“hybrid” vascular graft [245]. One of the most promising recent innovations in aortic arch repair is the “trifurcated graft” technique (Fig. 1.38) [246, 247], along with a large number of debranching hybrid repair concepts using concomitant endovascular stent grafts [248, 249].

Arguably the last major development to date in treating aortic disease has been the evolution of endovascular stent grafting. That new era of treatment started in 1986 when an alternative to surgically placed grafts emerged. **Harrison Lazarus** (1939–) of Salt Lake City had conceived and essentially completed the design of an endovascular graft for abdominal aneurysm repair by the mid-1980s and filed for a US patent in 1986 (awarded in 1988) [250, 251]. The pioneering clinical work is first and foremost associated with **Nikolai Volodos** (1934–) in Kiev, Ukraine [252–255], and **Juan Parodi** (1940–) of Argentina [256]. **Volodos** reportedly performed the first-ever aortic repair with a stent graft in the 1980s (in a patient suffering from a post-traumatic aneurysm of the distal descending thoracic aorta) (Fig. 1.39) [255], but this pioneering work

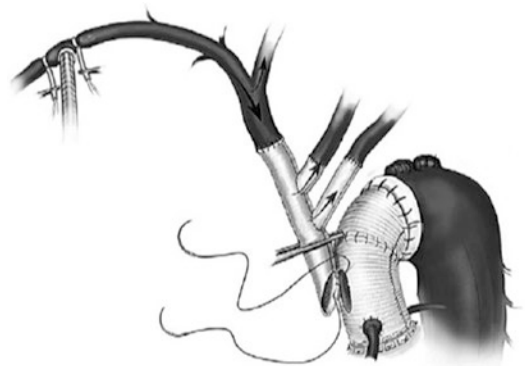


Fig. 1.38 Aortic arch replacement with a trifurcated graft. With the main limb of the trifurcated graft clamped, antegrade selective cerebral perfusion is initiated through the axillary artery. The elephant trunk technique is used to reconstruct the arch, and the graft is anastomosed to the proximal repair. The trifurcated graft is then anastomosed to the reconstructed aorta (from Ref. [247], with permission)

has become widely known in the Western world only since the mid-1990s. Argentinian **Julio Palmaz** (1945–) of San Antonio invented and patented the balloon-expandable stent, which was later approved for use in peripheral arteries in 1991. Human endovascular abdominal



Fig. 1.39 Pioneer of vascular stent graft design. Nikolai L. Volodos (from Ref. [255], with permission)

aneurysm repair (EVAR) was performed by **Parodi** and associates in Buenos Aires and **Palmaz** on September 6, 1990, following extensive experiments with stainless steel stents hand sewn to thin-walled Dacron tube grafts [257, 258]. They launched the modern endovascular treatment revolution that led to profound transformations that have changed everything in vascular surgery.

Treatment modalities for thoracic aneurysms followed successful repairs of abdominal aortic aneurysms. **Michael D. Dake** at Stanford reported the first endovascular thoracic (descending) aortic repair with a homemade endograft in 1994 [259, 260], soon followed in 1999 by a report of earliest clinical experience with endovascular stent graft intervention for acute type B aortic dissection (Fig. 1.40) [261]. As such, TEVAR intervention has added an entirely new dimension to the management of AD and aortic disease (Fig. 1.41) [262–264].

Rapid diagnosis and appropriate management decisions were greatly advanced with improved

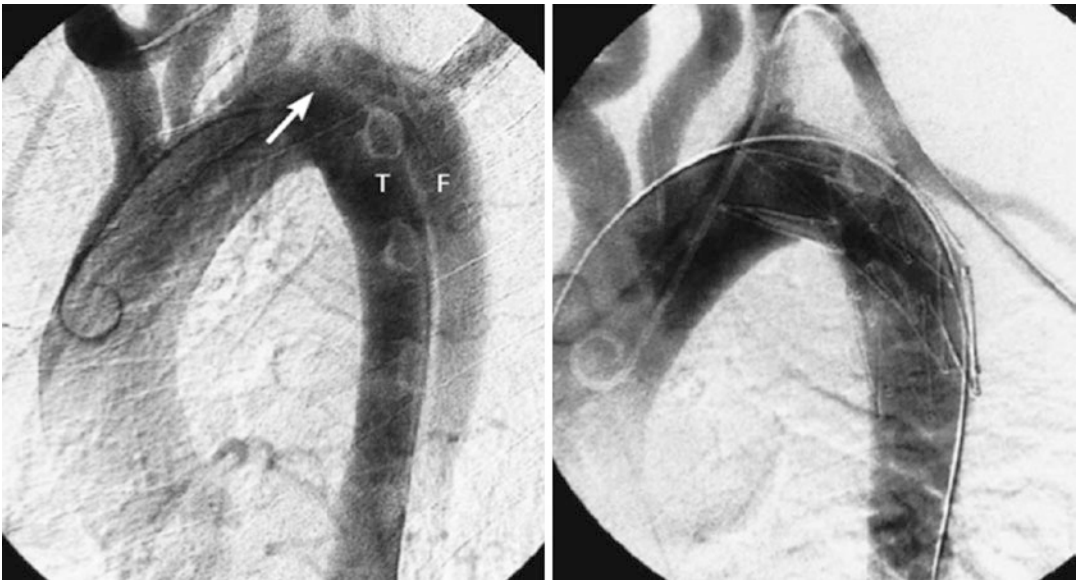


Fig. 1.40 Thoracic aortograms obtained before and after stent graft placement over the primary entry tear in aortic dissection. Left: Before stent graft deployment showing flow of contrast medium from the true lumen (T) across

the entry tear (arrow) into the false lumen (F). Right: After stent graft placement. Only the true lumen is evident (from Ref. [261], with permission)



Fig. 1.41 Stent graft with side branches. Left: *A.* Branched stent graft with one free-flow stent, one internal sealing stent, followed by reducing stents, from which emerge the proximal side branches. *B.* Detail of the emergence of the side branches from the main stent graft module. *C.* Iliac side branch device. Right: The

three-dimensional CT angiography reconstruction shows a thoracoabdominal stent graft with four branches, in adjunct to left internal iliac artery revascularization with iliac side branch device and contralateral embolization of internal iliac artery (from Ref. [262], with permission)

imaging modalities, such as CTA, CT, TEE, TTE, and MRI. Today's clinical management is further greatly enhanced by recognition of "aortic syndromes" and distinct pathologies such as penetrating atherosclerotic ulcers (PAU), IMH, aortic ruptures, and dissection, apart from congenital syndromes.

The author is well aware that this brief review of aortic surgery cannot be complete, especially with

regard to all the individuals who have contributed to the field over the centuries: "history, as it lies at the root of all science" can be understood as "the essence of innumerable biographies" [265].

This textbook looks deep into the past and, from there, toward the future with all its promising new directions, and as such it is to be hoped that it will help to take the history of aortic management one step further.

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