


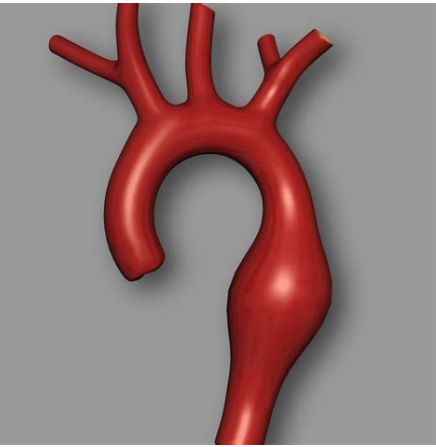
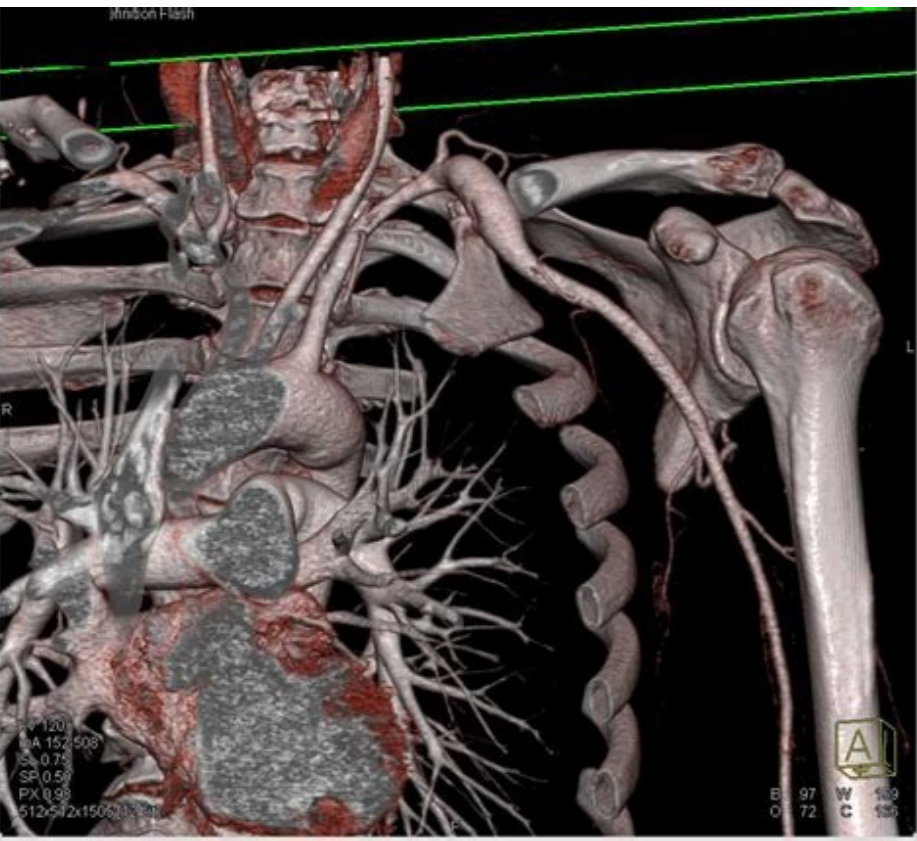
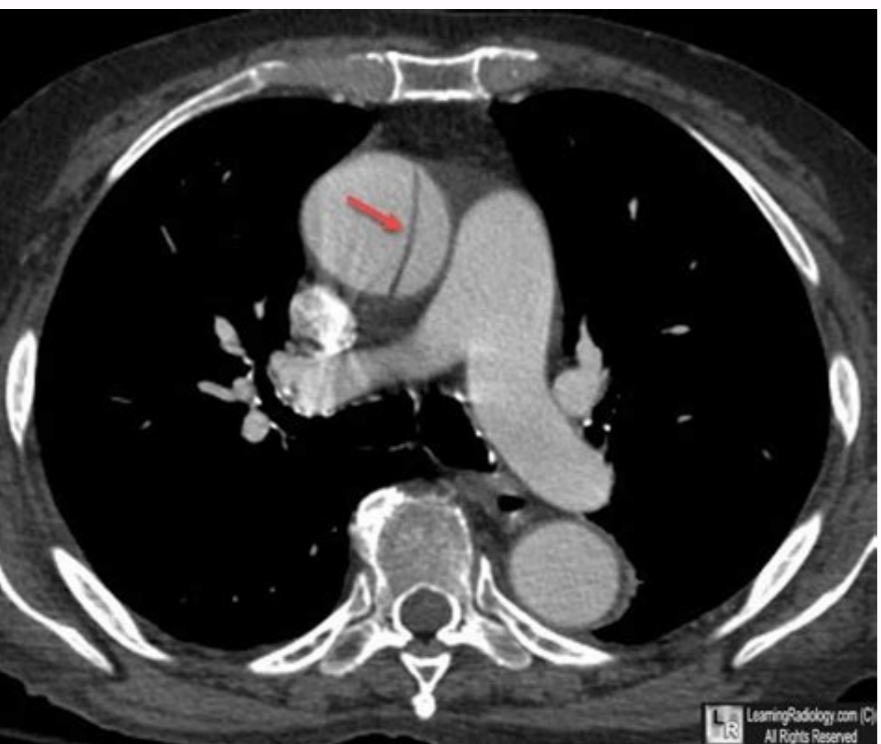
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Abdominal aortic aneurysm ct report

AORTIC ANEURYSMS ENDOVASCULAR REPAIR USING FENESTRATED & BRANCHED ENDOGRAFTS

Gustavo S. Oderich MD
Professor of Surgery
Director of Endovascular Therapy
Division of Vascular and Endovascular Surgery



What is the definitive test used to diagnose an abdominal aortic aneurysm. Would an abdominal ct show an aortic aneurysm. How much does an abdominal aortic aneurysm screening cost. When should an abdominal aortic aneurysm be repaired. Is an abdominal aortic aneurysm dangerous.

It should not be confused with aortic dissection. Medical condition ANEURYSMICO ANEURYSMICO NOMESTRIPLE-ACT Reconstruction Image of an abdominal aortic aneurysm (white arrows) SurgerySymmercularSymptomsNone, abdominal, back or leg pain [1] [2] Typical location of 50 years old [1] Risk factors, high blood pressure, Other blood vessel diseases, family history, Marfan syndrome [1] [3] [4] Methodmedical diagnosis Imaging (abdominal aorta diameter) [1] Prevention Not Smoking, dealing with risk factors [1] Treatment (open or endovascular surgery, Aneurysm repair) [1] Frequency – 658, 200 years (man) Abdominal aortic aneurysm (AAA or triple a) [6] Is a localized extension of the abdominal aorta. So the diameter is greater than 3 cm or more than 50% larger than normal. [1] They usually don't cause symptoms, except during the breakup. [1] Occasionally, abdominal pain, ass or leg may occur. [2] Large aneurysms can sometimes feel pushing the abdomen. [2] Breaking may result in pain in the abdomen or back, low blood pressure or loss of consciousness, and often results in death. [1] [7] AAA occurs more commonly in those over 50 years of age, in men, and among those with family history. [1] Additional risk factors include smoking, high blood pressure and other blood vessel diseases. [3] Genetic conditions with a higher risk include Marfan syndrome and Ehlers-Danlos syndrome. [4] AAA are the most common form of aortic aneurysm. [4] About 85% occurs below the kidneys with the rest, either at the level of or above the kidneys. [1] In the United States, detection with abdominal ultrasound is recommended for males between 65 and 75 years of age with a history of smoking. [8] InUnited Kingdom and Sweden, screening is recommended for all men over 65 years of age. [1] [9] Once an aneurysm is found, additional ultrasounds are usually performed on a regular basis. [2] Not smoking is the single best way to prevent disease. [1] Other Other prevention include the treatment of high blood pressure, the treatment of high blood cholesterol and the non-overweight.[1] Surgery is usually recommended when the diameter of an AAA grows to ≤ 5.5 cm in men and ≤ 5.0 cm in women.[1] Other reasons for repair include the presence of symptoms and a rapid increase in size, defined as more than one centimeter per year.[2] Repair can be done through open surgery or repair of endovascular aneurysm (EVAR).[1] Compared to open surgery, EVAR has a lower risk of short-term death and shorter hospitalization, but it cannot always be an option.[1] [10] [11] There seems to be no difference in long-term results between the two.[12] Repeated procedures are more common with EVAR.[13] AAAs affect 2-3% of men over 65 years of age.[1] The rates among women are a higher quarter[1]. In those with an aneurysm below 5.5 cm, the risk of rupture in the next year is less than 1%. [1] Among those with an aneurysm between 5.5 and 7A cm, the risk is about 10%, while for those with an aneurysm greater than 7A cm the risk is about 33%. [1] Breakdown mortality is 85% to 90%. [1] During 2013, aortic aneurysms caused 168,200 deaths, compared to 100,000 in 1990.[5] [14] In the United States, AAAs caused between 10,000 and 18,000 deaths in 2009.[4] Signs and symptoms Location of abdominal aortic aneurysm The vast majority of aneurysms are asymptomatic. However, as the abdominal aorta expands or breaks, the aneurysm may become painful and cause pulsible sensations in the abdomen or chest pain, the lower back, legs, or scrotum.[15] Complications Complications include rupture, peripheral embolization, acute aortic occlusion and aorticaval fistulas (between aorta and inferior vena cava) or aortoduodenalthe aorta and the duodenum). In the physical examination, a palpable and pulsable abdominal mass is observed. Mores may be present in case of renal or visceral arterial stenosis.[16] Signs and symptoms of AAA AAA ruptureinclude intense pain in the lower part of the back, side, abdomen, or groin. You can also feel a beating mass with the heartbeat.[7] Bleeding can lead to a hypovolemic shock with low blood pressure and a rapid heart rate. This can lead to short fainting.[7] AAA breakdown mortality is up to 90%. From 65 to 75% of patients die before reaching the hospital and up to 90% die before reaching the O.R. [17] Bleeding can be retroperitoneal or abdominal cavity. The rupture can also create a connection between the aorta and the intestine or the inferior vena cava.[18] Equamous from the side (a morbidity of a bruise) is a sign of retroperitoneal bleeding, and is also called a sign of Grey Turner.[16] [19] Causes The exact causes of the degenerative process are not clear. However, there are some well-defined scenarios and risk factors.[20] Smoking tobacco: More than 90% of people who develop an AAA have smoked at some point in their lives.[21] Alcohol and hypertension: Inflammation caused by prolonged alcohol consumption and hypertensive effects of abdominal edema leading to hemorrhoids, esophageal varices and other conditions, is also considered a long-term cause of AAA. Genetic influences: The influence of genetic factors is high. AAA is four to six times more common in male brothers of known patients, with a risk of 20-30%. [22] High family prevalence is more noticeable in men.[23] There are many hypotheses about the exact genetic disorder that could cause a higher incidence of AAA among male members of affected families. Some presumed that the influence of alpha 1-antitripsin deficiency could be crucial, while other experimental works favored the mutation hypothesis linked to X, which would explain the lowest incidence in heterozygotes. Otherof genetic causes.[16] Conjunctive tissue disorders, such as Marfan syndrome and Ehlers-Danlos syndrome, have also been very common with AAA.[18] Both recurrent polycondritis and elastic pseudoxanthoma can cause abdominal aortic aneurysm.[24] Atherosclerosis: For a long time it was considered that AAA was caused by atherosclerosis, because AAA walls often carry an atherosclerotic load. However, this hypothesis cannot be used to explain the initial defect and the development of occlusion, which is observed in the process.[16] Other causes of AAA development include: infection, trauma, arteritis and cystic medial necrosis.[18] Physiology Gray Anatomy Plate with yellow lines that represent the most common infrarenal location of the AAA 3D file that shows an aortic aneurysm The most striking histopathological changes of the aneurysm aorta are observed in the medium tunica and the intima layers. These changes include the accumulation of lipids in foam cells, extracellular free cholesterol crystals, calcifications, thrombosis and ulcerations and layer ruptures. Adventitial inflammatory infiltration.[18] However, the degradation of the tunica media through a proteolytic process seems to be the basic physiopathological mechanism of AAA development. Some researchers report increased expression and activity of the matrix metalloproteinases in individuals with AAA. This leads to the elimination of media elastin, making the aortic wall more susceptible to the influence of blood pressure.[16] Other reports have suggested that the granzima B of the protease of the seine may contribute to the breakdown of the aortic aneurysm through the excision of the decroine, which leads to an organization of the altered collagen and a lesser resistance to the traction of adventium.[25] [26] There is also a lower amount of vasa vasorum in the abdominal aorta (compared) in the abdominal aorta (compared with the chest aorta); consequently, the means of tunica should depend onfrom diffusion for nutrition, making it more susceptible to damage.[27] Hemodynamics affects the development of AAA, which has a predilection for the infrarenal aorta. The histological structure histological characteristics of the infrarenal aorta differ from those of the thoracic aorta. The diameter decreases from the root to the Arctic bifurcation, and the wall of the infrarenal aorta also contains a lower proportion of elastin. The mechanical tension on the abdominal aortic wall is, therefore, higher than on the thoracic aortic wall. Elasticity and distensibility also decrease with age, which may result in a gradual dilation of the segment. Increased intraluminal pressure in patients with arterial hypertension contributes significantly to the progression of the pathological process. [18] Appropriate hemodynamic conditions may be linked to specific patterns of intraluminal thrombus (ILT) along the aortic lumen, which in turn may affect the development of AAA. [28] Diagnostic An abdominal aortic aneurysm is usually diagnosed by physical examination, abdominal ultrasound or CT scan. Simple abdominal x-rays can show the outline of an aneurysm when your walls are calcified. However, the pattern will be visible on the X-ray in less than half of all aneurysms. Ultrasound is used to detect aneurysms and determine the size of any present. In addition, free peritoneal fluid can be detected. It is not invasive and sensitive, but the presence of intestinal gas or obesity may limit its usefulness. Computed tomography has a sensitivity of almost 100% for an aneurysm and is also useful in preoperative planning, which details the anatomy and the possibility of endovascular repair. In the case of suspected rupture, you can also reliably detect the retroperitoneal fluid. The alternative, less often, methods used to visualize an aneurysm include magnetic resonance imaging and angiography. [Quote required] An aneurysm breaks if the mechanical tension (area tension) exceeds the local strength of the wall. As a result, it has been found the maximum wall stress (PWS) [29] and the maximum wall failure risk (PWR) [30] are more reliable parameters than the diameter for assessing the risk of AAA rupture. The medical software makes it possible to calculate these risk rates of breach of the standard CT data and provides a patient-specific AAA rupture risk diagnosis. [31] [32] [33] This type of biomechanical approach has been shown to accurately predict the location of AAA rupture. [32] [33] [34] Aortic measurement of abdominal ultrasonography in the axial plane between the outer margins of the aortic wall. [35] Ultrasonography in the sagittal plane, showing the measurement of the axial plane (degraded red line), as well as the maximum diameter (small yellow line) that is preferred. AAA rotates with an open arupture the aneurysm and closed arupture marking the free blood in the abdomen Image TC Sagittal of a Biomechanical AAA Risk Prediction AAA Acomputer tomography improved by axial contrast that shows an abdominal aortic aneurysm of 4.8 by 3.8 cm The weak outline of the licified AAA X-ray as a veto An aortic aneurysm as seen in the CT with a small remaining Play media Ultrasound blood flow area showing a previously repaired AAA that is filtering with the flow around the graft[36] Ultrasonography of an aneurysm with a mural thrombo. Classification Sorting Size Classification Eighth or similar dilation ± 2.0 cm and 0 cm [37] Moderate 3.0 - 5.0 cm [37] Abdominal aortic aneurysms are commonly divided according to their size and symptomatology. An aneurysm is usually defined as an outer aortic diameter exceeding 3 cm (the normal diameter of the aorta is about 2 cm). [39] or more than 50% of the normal diameter. [40] If the outer diameter exceeds 5.5 cm, the aneurysm is considered to be large. [38] It should be suspected that AAA is broken in an older person (age 60) with collapse, low blood pressure without explaining, or abdominal pain in the back or back. Abdominal pain, shock and a palpable mass are only present in aces. Although an unstable person with a known aneurysm may undergo surgery without more images, the diagnosis will usually be confirmed by CT scan or ultrasound. [The Necessary Vocation] The suprarenal aorta usually measures approximately 0.5 cm larger than the infrarenal aorta. [41] Differential diagnosis The aortal aneurysm rupture can be confused with the pain of the rich stones or back pain related to the muscles. [7] Prevention Smoking The treatment of treatment of hypertension detection The junction task of the United States (USPSTF) recommends an abdominal ultrasound of a single examination for abdominal aortic aneurysm in males from 65 to 75 years Have a smoking story. [42] Between this group that does not smoke, the detection can be selective. [42] It is not clear whether the detection is useful in women who have smoked and the USPSTF recommends against detection in women who have never smoked. [8] [43] In the United Kingdom, the detection program of NHS AAA invites men in England to detect the selection during the year in which they meet 65 years. Men over 65 years can contact the program to organize being examined. [44] In Sweden it is recommended once it is recommended once more in all men over 65 years. [1] [9] It has been found that this decreased the risk of death from AAA by 24% with a number necessary to detect little more than 200. [43] In those with a close relative diagnosed with a aortal aneurysm , Swedish guidelines recommend an ultrasound at around 60 years of age. [45] Australia has no guide in the detection. [46] Ultrasound repetitions must be carried out in those that have a tootic size greater than 3.0 cm. [47] In those whose aorta is between 3.0 and 3.9 ". This should be every three years, if between 4.0 and 4.4 cm every two years, and if between 4.5 and 5.4 cm each year. [47] Gestion Treatment options for AAA Asymptoms are conservative management, surveillance with views of eventual repair and immediate repair. There are two repair modes for an AAA: Aneurysm repair and repair of endovascular aneurysms (EVAR). An intervention is often recommended if the aneurysm grows more than 1 cm per year or is larger than 5.5 cm. [48] Repair is also indicated for symptomatic aneurysms. Ten years after AAA open repair, AAA. The overall survival rate was 59 per cent.[49] Conservative management is indicated in people where repair carries a high risk of mortality and in patients where repair is unlikely to improve life expectancy. The pillar of conservative treatment is to stop smoking. [The necessary vocation] Surveillance is indicated in small asymptomatic aneurysms (less than 5.5 cm) where the risk of repair exceeds the risk of rupture. [48] As AAA grows in size, the risk of rupture increases. Surveillance has not been shown until an aneurysm has reached a diameter of 5.5 cm with a higher risk compared to early intervention.[50] [51] Medication It has not been determined that medical therapy is effective in reducing the growth rate or the breakdown rate of asymptomatic AAAs. [1] However, blood pressure and lipids should be treated as usual. [39] Surgery The repair threshold varies slightly from one individual to another, depending on the balance of risks and benefits when considering repair against continuous surveillance. The size of an individual's native aorta can influence this, along with the presence of comorbidities that increase the operating risk or decrease the life expectancy. The evidence, however, does not usually support the repair if the size is less than 5.5 cm.[48] Open repair Main article: Open aortic surgery Open repair is indicated in young patients as an elective procedure, or in large or large, symptomatic or broken aneurysms. The aorta should be subjected during repair, denying the blood to the abdominal organs and sections of the spinal cord; this may cause a variety of complications. It is essential that the critical part of the operation be quick, so the incision is made big enough to facilitate the quickest repair. Recovery after open surgeryIt takes a long time. The minimums are a few days in intensive care, a total of week in the hospital and a few months before full recovery. [The Necessary Vocation] Abdominal aortic endoprothesis, computed tomography, computerized.Aneurysm marked in the main blue article: the repair of endovascular aneurysms, endovascular repair was made practically practical in the 1990s and, although it is now an established alternative for open repair, its function has not yet been clearly defined. In general, it is indicated in older, high-risk patients or patients who are not fit for open repair. However, endovascular repair is feasible for only one proportion of AAA, depending on the morphology of aneurysm. The main advantages of open repair are that there is lower perioperative mortality, less time in intensive care, less time in the general hospital and before normal activity. The disadvantages of endovascular repair include a requirement for more frequent ongoing hospital checks, and a greater likelihood that more procedures are required. According to the latest studies, the EVAR procedure does not offer any benefit to general survival or health-related quality of life compared to open surgery, although aneurysm-related mortality is lower. [52] [53] [54] [55] In patients not fit for open repair, EVAR PLUS Conservative management was associated with no benefits, more complications, subsequent procedures and higher costs compared to the conservative management alone. [56] Endovascular treatment for paraaortic aneurysms after the aORobotic reconstruction is also a possibility. [57] A review of Cochrane 2017 found attempted evidence of no difference in the results between the endovascular and open repair of the AAA break in the first month. [58] Rupture in those with aortic breakage of AAA, treatment is an immediate surgical repair. There seems to be benefits to allow permissive hypotension and limit the use of intravenous fluids during transport foot operations [59] AAA announcement size (cm) type of growth (cm / year) [60] Annual breaking risk (%) [61] 3.0-3.9 0.39 0.4-0.9 0.36 0.5-5 5.0-5.9 0.43 3-15 6.0-6.9 0.64 10-20 > = 7.0-20-50 Although the current standard of the rupture risk determination is based on the maximum diameter, it is known that the aaa higher \pm os small AAAs lower than this threshold (diameter 5.5A cm) can also be broken, and the larger AAA (diameter 5.5A cm) can remain stable[62] [63] In a report, it was shown that 10A24% of the broken AAAs had a diameter of less than 5A cm[63] It has also been reported that of 473 AAA not repaired examined from autopsy reports, there were 118 cases of rupture, 13% of which were less than 5 cm in diameter. This study also showed that 60% of AAAs over 5 cm (including 54% of AAAs between 7.1 and 10 cm) did not break.[64] Vorp et al. later deduced, from the results of Darling et al., that if the maximum diameter criterion for the 473 subjects were followed, only 7% (34/473) of the cases would have succumbed to the breakage before the surgical intervention, since the diameter was less than 5A cm, and that 25% (116/473) of the cases could possibly be subjected to unnecessary surgical intervention, since these would never have been broken Alternative repair assessment methods have recently been reported. Most of these approaches involve the numerical analysis of AAAs using the common engineering technique of the finite element method (FEM) to determine wall effort distributions. Recent reports have shown that these voltage distributions correlate with the overall geometry of the AAA and not only with the maximum diameter[65] [66] [67] It is also known that the pressure of the wall alone does not completely govern the failure, as an AAA usually breaks when the pressure of the wall exceeds its resistance. In light of this, the evaluation of the breakage can be more accurate if the stress of the patient's specific wall is combined with the resistance of the patient's specific wall. A non-invasive method was recently reported to determine the resistance of the wall on the basis of the patient[68] with more traditional approaches to determinationresistance through tracing tests conducted by other researchers in the field.[69] [70] [71] Some of the most recently proposed methods for assessing the risk of AAA rupture include: AAA wall AAA AAA expansion rate; [74] Grade of Asymmetry; [67] Presence of intraluminal thrombus (ILT); [75] A potential rupture index (RPI); [76] [77] A rupture index of finite elements analysis (feadi); [78] Biomechanical factors coupled with informant analysis; [79] Growth of the geometric parameters of ILT; [80] of the AAA; [81] And also a method to determine the growth and breaking of AAA in function of mathematical models. [82] [83] Postoperative mortality for an AAA already broken has slowly decreased for several decades, but it remains greater than 40%. [84] However, if the AAA is repaired quirologically before breaking, the postoperative mortality rate is approximately 1-6%. [85] Epidemiology The AAA's appearance varies according to ethnicity. In the United Kingdom, the AAA rate in caucasian men greater than 65 years is approximately 4.7%, while in Asian men it is 0.45%. [86] It is also less common in individuals of African and Hispanic heritage. [1] It occurs four times more often in men than women. [1] There are at least 13,000 annual deaths in the US. UU secondary to AAA's breakup. [1] The maximum number of new cases per year between males is around 700 years of age, the percentage of males affected for 60 years is 2-6%. The frequency is much greater in smokers than in non-smokers (8: 1), and risk slowly decreases after cessation of smoking. [87] In the United States, the incidence of AAA is 2. 4% in the adult population. [16] AAA's rupture occurs in 1: 3% of men of 65 years or more, mortality is 70 \dot{a} - 95%. [38] History The first historical records on AAA are Of ancient Rome in the second century AD, when the Greek Antyllus surgeon tried to treat AAA with proximal and distal ligation, central incision and elimination of thrombotic material from the aneurysm. However, attempts to treat AAA They were not successful until 1923. In that year, Rudolph Matas (who also proposed the concept of endoneurymorlaphy) made the first successful aortic ligation in a human. [88] Other methods that were successful.Treatment of AAA included wrapping the aorta with polyethylene cellophane, which induced fibrosis and limited the growth of the aneurysm. Endovascular aneurysm repair was first performed in the late 1980s and has been widely adopted in subsequent decades. Endovascular repair was first used to treat a broken aneurysm in Nottingham in 1994.[89] Society and Culture Theoretical physicist Albert Einstein was operated on for an abdominal aortic aneurysm in 1949 by Rudolph Nissen, who wrapped the aorta with powder cellophane, ethylene. Einstein's aneurysm ruptured on April 13, 1955. He refused the surgery, saying, "I want to go whenever I want. It's in bad taste to prolong life artificially. I've done my part, it's time to go. She died five days later at the age of 76.[90] Actress Lucille Ball died on April 26, 1989 of an Arctic abdominal aneurysm. At the time of her death, she was at the Cedars-Sinai Medical Center recovering from an emergency surgery performed just six days earlier due to a dissecting Arctic aneurysm near her heart. Ball was at greater risk, having been a heavy smoker for decades.[91] Musician Conway Twitty died in June 1993 of an Arctic abdominal aneurysm at age 59, two months before the release of what would be his last studio album, Final Tou. cocks. Actor George C. Scott died in 1999 of a ruptured Arctic abdominal aneurysm at the age of 71. [citation required] In 2001, former presidential candidate Bob Dole underwent surgery for an abdominal aortic aneurysm in which a team led by vascular surgeon Kenneth Ouriel inserted a stent.[92] Ouriel said the team inserted a Y-shaped tube through an

only one timid day of his 42nd birthday. His father also died for the same cause when Robert was a child. Actor Tommy Ford died of abdominal aneurysm in October 2016 at 52 years. [93] Gary Gygas co creator of Dungeons and Dragons died in 2008 by a 69-year-old aortic Abdominal aneurysm. Risk Assessment of Research There have been many calls for alternative approaches to risk assessment for rupture over the past few years, and many believe that a biomechanical approach can be more appropriate than the current diameter approach. Numerical modeling is a valuable tool for researchers that allow to calculate approximate wall tensions, thus revealing the potential for breaking a particular aneurysm. Experimental models are necessary to validate these numerical results and provide a new vision of AAA's biomechanical behavior. In vivo, AAAs exhibit a variety of material fortresses[94] from localized weak hypoxic regions[95] to much stronger regions and calcification areas. [96] Finding ways to predict the future growth of AAA is considered a research priority. [97] Experimental models Experimental models can now be manufactured using a new technique that involves the process of manufacturing lost wax of injection molding to create anatomically correct AAA replicas specific to each patient.[98] The work has also focused on developing more realistic materials analogs for those in vivo, and recently a new range of silicone-rubbers was created that allows the variable material properties of AAA to be more precise. [99] These rubber models can also be used in a variety of experimental situations, from stress analysis using the photoelastic method[100] New endovascular devices are being developedcan treat more complex and tortuous anatomy. [101] prevention and treatment of an animal study showed that the elimination of a single protein prevents blood vessels from causing complications in the posterior stage.Signature protein called cyclophilin A (CYPA) of a strain of mice, researchers could provide full protection against abdominal aortic aneurysm. [102] Other recent research identified the Granzyme B (GZMB) (an enzyme that degrades proteins) as a possible objective in the treatment of abdominal aoral aneurysms. The elimination of this enzyme in mice models delayed the progression of aneurysms and improved survival. [103] [104] Preclinical research The mechanisms that lead to the development of AAA still are not fully understood at the cellular and molecular level. In order to better understand the physiopathy of the AAA, it is often necessary to use experimental animal models. It is often questioned how well these models are translated into human illness. Although there is no animal model that exactly represents human condition, all existing models focus on a different physiopathological aspect of the disease. Combining the results of different animal models with clinical research can provide a better general vision of AAA physiopathology. The most common animal models are rodents (mice and rats), although in some studies, such as the testing of preclinical devices or surgical procedures, large animal models (pigs, sheep) are used more frequently. AAA rodent models can be classified according to different aspects. There are dissection models vs models not dissection and genetically determined models vs models chemically induced. The most used models are the infusion of angiotensin-II in Knockout ApoE mice (dissection model, chemically induced), the calcium chloride model (non-dissection, chemically induced) and the Elastase model (non-dissection, induced model Quemically). 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