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Fusiform aneurysm of the ascending thoracic aorta

Aortic aneurysm: What is it and how is it treated? The aorta is the largest artery in the body and is a blood vessel that carries oxygen-rich blood from the heart to all parts of the body. The part of the aorta that runs through the chest is called the thoracic aorta, and since the aorta moves down through the abdomen, it is called the abdominal aorta. What is a thoracic aortic aneurysm? When the artery wall in the aorta weakens, the wall abnormally expands or bulges as blood is pumped through what causes the aortic aneurysm. Lumps or balloons can be defined as: Fusiform: A uniform shape that appears equally along the enlarged part and edges of the aorta. Sacicular aneurysm: A small, lop-sided blister on one side of the aorta that forms in the weakened area of the aorta wall. An aneurysm can develop anywhere along the aorta: Aneurysms that occur in the part of the aorta that runs through the abdomen (abdominal aorta) are called abdominal aortic aneurysms. Aortic aneurysms that occur in the chest area are called thoracic aortic aneurysms and may include the root of the aorta, ascending aorta, aortic arch or descending aorta. Aneurysms that include the aorta as it flows through the abdomen and chest are called thoracoabdominal aortic aneurysms. Who is affected by the aneurysm of the thoracic aorta? Thoracic aortic aneurysms affect approximately 15,000 people in the United States each year. Up to 47,000 people die each year on all types of aorta; over from breast cancer, AIDS, homicides, or motor vehicle accidents, making aortic disease a silent epidemic. Is the aneurysm of the thoracic aorta serious? A thorax aortic aneurysm is a serious health risk because depending on its location and size it can crack or dissect (tear), causing life-threatening internal bleeding. When detecting at times, thoracic aortic aneurysms can often be corrected with surgery or other less invasive techniques. Small aneurysms place one at increased risk for: Atherosclerotic plaque (fat and calcium deposits) formation at the site of the aneurysm clot (thrombus) can form in place and relax, increase the chance of stroke Increase the size of the aneurysm, causing it to squeeze into other organs, causing pain Autopsy of the aorta or tearing of the layers of the aorta, potentially fatal complication and medical emergency. The aneurysm ruptures because the artery wall thinner at this point is brittle and can burst under stress. Sudden rupture of the aortic aneurysm can be life-threatening and is a medical emergency What is an autopsy of the aorta? An aortic autopsy occurs when the layers of the aorta tear and separate from each other. The presence of an aortic aneurysm increases the risk of autopsy of the aorta, but autopsy of the aorta can also occur in people of normal aortic size. Aortal autopsy can occur suddenly, causing severe sharp, tearing pain in the chest or upper back. Yet, like all types of aneurysms, there may be no symptoms of the aorta Most often associated with high blood pressure, aortic autopsy forces the layers of the aorta wall apart through increased blood flow. If not treated in a timely manner, the autopsy of the aorta weakens the aorta and can lead to an aortic aneurysm by causing weakening of the area of the aorta bulge like a balloon, stretching the aorta. If you experience any symptom of a thoracic aortic aneurysm or autopsy, tell your doctor immediately. If not treated, these conditions could lead to fatal rupture. Symptoms of autopsy of the aorta The most common symptom: Severe chest pain (anterior, posterior or both). Uncommon symptoms: Abdominal pain, numbness or weakness in one or both legs, loss of consciousness, symptoms of stroke. If you have these symptoms, call emergency help (dial 9-1-1 in most areas). What causes thoracic aortic aneurysm? Thoracic aortic aneurysms are most often caused by atherosclerosis, hardening of the arteries that damages the walls of the artery. While your arteries are usually smooth on the inside as you age they can develop atherosclerosis. When atherosclerosis occurs, a sticky substance called atheromatous plaque accumulates in the walls of the arteries. Over time, excess plaque causes the aorta to solidify and weaken. Your risk of atherosclerosis increases if: You are a smoker have high blood pressure Have high cholesterol are obese Have a family history of cardiovascular or peripheral vascular diseases (narrowing of blood vessels) Some diseases can also weaken the layers of the aortic wall and increase the risk of thoracic aortic aneurysms, including: Marfan syndrome (genetic connective tissue disorder), Loeys-Dietz and other familial connective tissue disorders Other non-sucisive connective tissue disorders (characterized by a family history of aneurysms) Presence of bicuspid aortic valve infections Inflammatory disease Rarely, trauma such as severe fall or car accident can cause thoracic aortic aneurysm. As you get older, the risk of developing an aortic aneurysm increases. More men than women are diagnosed with a thoracic aortic aneurysm, and are often affected by the condition at a younger age. Recent research shows that a significant number of aneurysms have familial patterns or are inherited from previous generations. It is important to tell your doctor if there is a history of aortic aneurysm in your family to ensure that the best preventive examinations are completed. What are the symptoms of thoracic aortic aneurysm? Aneurysms of the thoracic aorta often go unnoticed, since patients rarely experience any symptoms. While only half of those with a thoracic aortic aneurysm complain of symptoms, possible warning signs include: Pain in the jaw, neck and upper back Chest or back pain Cough, spewing, or difficulty breathing Last reviewed by a Cleveland Clinic health care professional on October 1,2019. Get useful, useful and relevant health + wellness information enews The clinic is a nonprofit academic health center. Advertising on our site helps support our mission. We do not endud non-Cleveland Clinic products or services. Aortic aneurysm policies are occasionally evaluated and treated by doctors of many specialties. Indeed, while cardiac surgeons act on ascending aorta and arch and vascular surgeons manage abdominal aortic aneurysms, currently the responsibility often falls on cardiologists to oversee the medical care of patients with aortic disease of all types. However, although formally trained in cardiovascular medicine, most cardiologists pay their attention to the heart and its coronary arteries, and relatively few have experience in managing aortic diseases. Therefore, it is important that cardiologists acquire a sufficient knowledge base to be able to confidently evaluate and manage patients with aortic disease and know when it is advisable to refer them to surgery. To this end, the purpose of this review is to summarise the current understanding of thoracic and abdominal aortic aneurysms. Thoracic aortic aneurysmsThoracic aneurysms may include one or more aorta segments (aorta root, ascending aorta, arch, or descending aorta) and are classified accordingly (Figure 1). Sixty percent of thoracic aortic aneurysms include aortic root and/or ascending aorta, 40% include descending aorta, 10% engage arch, and 10% include thorate aorta (with some spanning >1 segment). Etiology, natural history and treatment of thoracic aneurysms vary for each of these segments. Figure 1. Anatomy of the thoracic and proximal abdominal aorta. ©Massachusetts General Hospital Thorate Aortic Center. It is used with consent.) Etiology and pathogenesis Neurysms of the ascending thoracic aorta most often arise from cystic medial degeneration, which appears histologically as a blackout of smooth muscle cells and degeneration of elastic fibers. Medial degeneration leads to weakening of the wall of the aorta, resulting in dilation of the aorta and the formation of an aneurysm. When such aneurysms include the root of the aorta, the anatomy is often referred to as anulaoorticectia. Cystic medial degeneration occurs normally to some extent with aging, but the process is accelerated by hypertension.1Marfan syndromeThe absence of cystic medial degeneration in young patients is classically associated with Marfan syndrome (or with other uncommon connective tissue disorders such as Ehlers-Danlos syndrome). Marfan syndrome is a herifiable autosomal dominant disorder caused by mutations in one of the fibrilline-1 genes, a structural protein that is the main component of elastin microfibrils. Mutations result in a decrease in the amount of elastin in the aortic wall and loss of the normally highly organized structure of elastin. As a result, the aorta exhibits significantly abnormal elastic properties that lead to a gradual increase in Thoracic aortic aneurysm syndromeCystic medial degeneration has also been observed in patients with ascending thoracic aortic aneurysms who do not have connective tissue disorders. In addition, it is now recognized that although cases of thoracic aortic aneurysms in the absence of connective tissue attacks can be sporadic, they are often familial and are now referred to as familial thoracic aortic aneurysm syndrome. In an analysis of their large database of patients with a thoracic aortic aneurysm Coady and colleagues3, they found that at least 19% of patients had a family history of thoracic aortic aneurysm and presented themselves at a significantly younger age than patients with sporadic aneurysms. Most pedigrals suggest an autosomal dominant way of inheritance, but there is significant variability in expression and penetration disorders, so that some inherit and pass on the gene, but show no manifestation of the disease. Several mutations have been identified. Mutation to 3p24.2–25 may cause both isolated and familial thoracic aortic aneurysms, with histological evidence of cystic medial degeneration.4,5 Mutations have also been mapped to 2 other chromosomal loci (5q13–14 and 11q23.2-q24).1,6 The extent of genetic heterogeneity is likely to become more visible as more families with thoragen aortic aortic aneurysm are examined. It is also possible that this is in fact a polygenic condition, thus explaining the variable expression and penetentrance. As a result, it is currently not possible to carry out routine genetic screening of this syndrome. Bicuspid Aortic ValvesMases ascending thoracic aortic aneurysms are associated with the underlying bicuspid aortic valve. It was once thought that such aneurysms were due to poststenotic dilation of ascending aorta, but the data suggest otherwise. Nistri et al7 used echocardiography to evaluate young people with commonly functioning bicuspid aortic valves and found that 52% had theoretic dilation (44% at the tubular part of the ascending aorta and 20% at the sinus level [i.e. root]). Indeed, other studies have shown that the bicuspid aortic valve is associated with dilapidated aorta, regardless of the presence or absence of haemodynamically significant valve dysfunction.8 The underlying cause of aortic dilation associated with the bicuspid aortic valve was found to be the underlying cause of aortic dilation associated with the bicuspid aortic valve. In one study, 75% of those with bicuspid aortic valve undergoing aortic valve replacement surgery had biopsy-proven cystic medial necrosis ascending aorta, compared to only 14% of patients with tricuspid aortic valves undergoing similar surgery. They found that patients with bicuspid aorta had less fibrillin-1 than patients with tricuspid aortic valves, and the reduction in fibrilline-1 was independent of patient age or aortic valve function. Interestingly, lung artery samples of the same individuals showed a similar reduction in fibrilline-1 content among those who have bicuspid aortic valves. This could lead to some patients with bicuspid valve undergoing the Ross procedure developing a late dilation of the pulmonary auto graft (see later sections). In addition, in a recent study in patients with ascending thoracic aortic aneurysms, Schmid et al12 found that compared to tricuspid aortic valve control, aortic tissue aneurysms of those with bicuspid aortic valves showed more lymphocyte infiltration and smooth muscle cell apoptosis. This suggests that wall aneurysms associated with bicuspid aortic valves may be weaker than more typical aneurysms. Since half of those with bicuspid valves have aortic dilation, cardiologists should routinely picture ascending aorta in all bicuspid aortic valve patients. In many cases, this can be achieved by echocardiography. However, while the root of the aorta is easily visualized in most transthoracic echocardiograms, in many cases the middle of the ascending aorta is not. Consequently, if the ascending diameter of the aorta is not reported on the echocardiogram, it cannot be safely assumed that it has been visualised and has been found to have a normal mean. You could examine the images to specifically examine the ascending aorta, but if it wasn't visualized enough, one should instead get a computed tomography (CT) scan or magnetic resonance imaging (MRI) study to determine the diameter of the aorta. AtherosclerosisAtherosclerosis is a rare cause of ascending thoracic aortic aneurysm. On the contrary, atherosclerosis is the predominant etiology of the aneurysm of the descending thoracic aorta. These aneurysms usually arise only distally to the origin of the left subclavian artery. The pathogenesis of atherosclerotic aneurysms in the thoragen aortic may resemble abdominal aneurysms (see later parts), but this is not extensively studied. SyphilisSyphilis was once perhaps the most common cause of ascending thoracic aortic aneurysms, but in an era of aggressive antibiotic treatment, such lueic aneurysms are rarely seen in modern medical centers. The latent period from initial spirochetal infection to complications of the aorta is usually 10 to 30 years, although it can range anywhere from 5 to 40 years. During the secondary phase of the disease, spirochetes directly infect the aortic media, causing smoothing endarteritis of the vassal vasor, especially the proximal ascending thorax aorta. Destruction of collagen and elastic tissues leads to dilation of the aorta, fibrosis and calcification. Longitudinal wrinkles on the aortic wall produces a radiographic pattern of barking trees. Weakening the wall of the aorta leads to aortic dilation, resulting in aneurysms that can be fusiform, but are often sacicular. The ascending aorta is most often affected, but an aneurysm may include an arch or root. Ascending aortic disability often produces secondary aortic regurgitation, and inflammation in the aortic root can result in ostial coronary artery stenoses. Turner syndromeTurner syndrome is associated with a number of cardiovascular anomalies, including bicuspid aortic valve (present in one third of individuals) and aortic coarctation. Thoracic aortic aneurysms are also found and usually include ascending aortics. In a recent systematic study of adults with Turner syndrome, Elsheikh et al13 screened 38 asymptomatic women and found that 42% had dilated aortic root. Of these, 4 out of 38 (11%) had bicuspid aortic valve and 11 out of 38 (29%) Hypertension. This population is at increased risk of aortic dissection and rupture. Indeed, those with both bicuspid aortic valve and aortic dilation may be at particularly high risk, given that both bicuspid aortic valve and Turner syndrome are strong risk factors for aortic dissection. As a result, it has been recommended that all women with Turner syndrome undergo a complete cardiac evaluation and echocardiographic or MRI scan at least every 5 years to detect potential aortic dilation.14Aortic arteritisTakayasu is the arteritis-most pronounced arteritis that affects aorta-is a chronic inflammatory disease of unknown etiology. The disease affects women much more often than men, and the average age at the time of diagnosis is 29 years.15 It usually causes obliterative luminal changes in the aortic and other arteries involved. However, in 15% of cases, aortic dilation can occur and lead to an aneurysm. These can arise either in the acute inflammatory (early) stage of the disease, or in the sclerotic (late) stage of the disease. Therefore, the finding of a thoracic aortic aneurysm in a young woman with symptoms of a systemic inflammatory process should increase the consideration of the disease. Giant-cell arteritis usually affects temporal or crayon arteries, but can also affect the aorta and produce aneurysms. In a long-term cohort of 169 patients in Olmstead County with giant cell arteritis, 30 (18%) aortic aneurysm (18 thoracic and 16 abdominal). Unfortunately, there were no clinically useful predictors of aortic involvement. Ankylos spondylitis is associated with inflammation of fibrocartilage, and it is hypothesis that inflammation is directed at tissues rich in fibrillary-1.17 Thus, it can be the cause of an aneurysm ascending aorta.18Aortic autopsyChronic autopsies of the aorta tend to break down over time. The affected walls of the aorta were weakened at the beginning of treatment (which led to an autopsy) and after autopsy the outer wall of the false lumen further weakened because its inner half (intimacy flap) was disassembled. As a result, persons with chronic aortic dissection are at high risk of developing an aneurysm and should be closely monitored using surveillance imaging studies. TraumaNonpenetrating traumatic aortic injuries usually occur as a result of slowing down the injury. Most often, trauma leads to partial or complete reloading of the descending thoracic aorta at the level adjacent to the left subclavian artery. Most of those with aortic transection die within an hour, and others undergo aortic repair during their initial hospitalization. However, in 1% to 2% of such patients, traumatic aortal transection is not diagnosed at the beginning, and patients can continue to develop chronic pseudoaneurysms at this site. These aneurysms are different in that they are usually sacicular (rather than the more common fusiform shape), relatively discrete, and located immediately distal on the left subclavian artery.19 Over time they tend to calcify. Clinical manifestationsSome patients with thorax aortic aneurysms are asymptomatic at the time of diagnosis, since aneurysms are usually discovered incidentally in imaging studies (chest x-ray, CT scan, or echocardiogram) ordered for other indications. An aortic aneurysm or ascending aorta can produce secondary regurgitation of the aorta, so a diastolic murmur can be detected during physical examination or, less often, patients may have congestive heart failure. When thoralat aortic aneurysms are large, patients may suffer from local effects such as bronchitis or mainstem bronchus (causing coughing, dyspnea, wheezing, or recurrent pneumonitis), compression of the esophagus (causing dysphagia), or compression of a recurrent bulging nerve (causing hoarseness). Rarely, chest or back pain may occur when an aneurysm is not distinguished due to direct compression of other intrathoracic structures or erosion into the adjacent bone. The dreaded consequence of a thoracic aneurysm is an aortic dissection or rupture (often referred to as acute aortic syndrome), which is potentially fatal. Typical symptoms of acute aortic syndrome include the sudden onset of severe pain in the chest, neck, back and/or abdomen. Diagnosis and SizingOften, thoracic aortic aneurysms are evident in chest x-ray films and are characterized by the expansion of mediastinal silhouettes, enlargement of the aortic knob, or tracheal

3rd, Estrera AL, Huynh TT, Porat EE, Allen BS, Sheinbaum R. Staged repairs of extensive aortic aneurysms: morbidity and mortality in elephant strain technique. *Circulation*. 2001; 104: 2938–2942.CrossrefMedlineGoogle Scholar31 Shores J, Berger KR, Murphy EA, Pyeritz RE. Progression of aortic dilation and benefit of long β -adrenergic blockade of Marfan syndrome. *N Engl J Med*. 1994; 1335–1341.CrossrefMedlineGoogle Scholar32 Ejiri J, Inoue N, Tsukube T, Munezane T, Hino Y, Kobayashi S, Hirata-Kawashima S, Imajoh-Ohmi S, Hayashi Y, Yokozaki H, Okita Y, Yokoy M. Oxidative stress in the pathogenesis of the thoracic aortic aneurysm: protective role of statin and angiotensin II receptor blocker. *Cardiovasc Res*. 2003; 59: 988-996.CrossrefMedlineGoogle Scholar33 Thompson MM. Control the expansion of the abdominal aortic aneurysm. *Br J Surg*. 2003; 90: 897-898.CrossrefMedlineGoogle Scholar34 Elefteriades JA, Hatzaras I, Tranquilli MA, Elefteriades JA, Stout R, Shaw RK, Silverman D, Barash P. Weightlifting and rupture of silent aortic aneurysms. *Jama*. 2003; 290: 2803. Letter.CrossrefMedlineGoogle Scholar35 Cayne NS, Veith FJ, Lipsitz EC, Ohki T, Mehta M, Gargiulo N, Suggs WD, Rozenblit A, Ricci Z, Timaran CH. Variability of maximum measurements of the diameter of the aorta aneurysm to CT scan: the importance and methods to minimize. *J. Vasc Surg*. 2004; 39: 811-815.CrossrefMedlineGoogle Scholar36 Multicenter Screening Study Group Aneurysm. Multicentre aneurysm screening study (MASS): analysis of the cost-effectiveness of abdominal aortic aneurysm screening based on four-year results of a randomised controlled trial. *BMJ*. 2002; 325: 1135-1138.CrossrefMedlineGoogle Scholar37 Lederle FA, Johnson GR, Wilson SE, Tastes EP, Littooy FN, Bandyk D, Krupski WC, Barone GW, Acher CW, Ballard DJ. Prevalence and association of abdominal aortic aneurysm detected through screening. Aneurysm Detection and Management (ADAM) Veterans Affairs Cooperative Study Group. *Ann Intern Med*. 1997; 126: 441-449.CrossrefMedlineGoogle Scholar38 Lederle FA, Johnson GR, Wilson SE. Aneurysm Detection and Management of Veterans Affairs Cooperative Study: Abdominal Aortic Aneurysm in Women. *J. Vasc Surg*. 2001; 34: 122–126.CrossrefMedlineGoogle Scholar39 Frydman G, Walker PJ, Summers K, West M, Xu D, Lightfoot T, Codd C, Dique T, Nataatmadja M. Screening value in siblings of patients with abdominal aortic aneurysm. *Eur J. Vasc Endovasc Surg*. 2003; 26: 396-400.CrossrefMedlineGoogle Scholar40 Lederle FA, Simel DL. Rational clinical examination: does this patient have an abdominal aortic aneurysm? *Jama*. 1999; 281: 77–82.CrossrefMedlineGoogle Scholar41 Lederle FA, Wilson SE, Johnson GR, Reinke DB, Littooy FN, Acher CW, Messina LM, Ballard DJ, Ansel HJ. Variability of measurement of abdominal aortic aneurysms. *J. Vasc Surg*. 1995; 21: 945-952.CrossrefMedlineGoogle Scholar42 Sprouse LR 2nd, Meier GH 3rd, Lesar CJ, Demasi RJ, Sood J, Parent FN, Marcinyck MJ, Gayle RG. Comparison of measurements of the diameter of the abdominal aortic aneurysm obtained by ultrasound and computed tomography: Is the difference? *J. Vasc Surg*. 2003; 38: 466-471.CrossrefMedlineGoogle Scholar43 Wilmink AB, Quick CR, Hubbard CS, Day NO. Efficacy and cost of abdominal aortic aneurysm screening: results of the population screening programme. *J. Vasc Surg*. 2003; 38: 72-77.CrossrefMedlineGoogle Scholar44 Kent KC, Zwolak Jaff MR, Hollenbeck ST, Thompson RW, Schermerhorn ML, Sicard GA, Riles TS, Cronenwett JL; Society for Vascular Surgery; American Association of Vascular Surgery; Society for Vascular Medicine and Biology. Screening abdominal aortic aneurysm: consensual statement. *J. Vasc Surg*. 2004; 39: 267-269.CrossrefMedlineGoogle Scholar45 Brown PM, Pattenden R, Vernooy C, Zelt DT, Gutelius JR Selective abdominal aortic aneurysm control in prospective measurement program. *J. Vasc Surg*. 1996; 23: 213-220.CrossrefMedlineGoogle Scholar46 Filling MF, Marra SP, Raghavan ML, Kennedy FE. Prediction of the risk of rupture in the abdominal aortic aneurysm during observation: wall stress versus mean. *J. Vasc Surg*. 2003; 37: 724-732.CrossrefMedlineGoogle Scholar47 UK Small Aneurysm Trial Participants. Mortality outcomes in a randomised controlled early elective surgery study or ultrasonographic monitoring of small abdominal aortic aneurysms. *Lancet*. 1998; 352: 1649–1655.CrossrefMedlineGoogle Scholar48 Lederle FA, Wilson SE, Johnson GR, Reinke DB, Littooy FN, Acher CW, Ballard DJ, Messina LM, Gordon IL, Tastes EP, Krupski TOILET, Busutil SJ, Barone GW, Sparks S, Graham LM, Rapp JH, Makaroun MS, Moneta GL, Cambria RP, Makhoul RG, Eton D, Ansel HJ, Freischlag JA, Bandyk D. Aneurysm Detection and Management Veterans Affairs Cooperative Study Group. Immediate repair compared to the supervision of small abdominal aortic aneurysms. *N Engl J Med*. 2002; 346: 1437–1444.CrossrefMedlineGoogle Scholar49 Brewster DC, Cronenwett JL, Hallett JW Jr., Johnston KW, Krupski WC, Matsumura JS; Joint Board of the American Association for Vascular Surgery and Society for Vascular Surgery. Guidelines for the treatment of abdominal aortic aneurysm: report of the subcommittee of the Joint Council of the American Association for Vascular Surgery and the Society for Vascular Surgery. *J. Vasc Surg*. 2003; 37: 1106-1117.CrossrefMedlineGoogle Scholar50 Dimick JB, Cowan JA Jr., Stanley JC, Henke PK, Pronovost PJ, Upchurch GR Jr. Surgeon specialties and provider volumes are related to the outcome of abdominal aortic contact aneurysm repair in the United States. *J. Vasc Surg*. 2003; 38: 739–744.CrossrefMedlineGoogle Scholar51 Laheij RJ, Butth J, Harris PL, Moll FL, Stelter WJ, Verhoeven EL. Need for secondary interventions following endovascular repair of abdominal aortic aneurysm: mid-term subsequent results of the European Collaborative Register (EUROSTAR). *Br J Surg*. 2000; 87: 1666-1673.CrossrefMedlineGoogle Scholar52 Gadowski GR, Pilcher DB, Ricci MA. Aneurysm of the abdominal aorta rate of expansion: the effect of size β -adrenergic blockade. *J. Vasc Surg*. 1994; 19: 727-731.CrossrefMedlineGoogle Scholar53 Brady AR, Thompson SG, Fowkes FGR, Greenhalgh RM, Powell JT. Enlargement of abdominal aortic aneurysms: risk factors and time intervals for surveillance. *Circulation*. 2004; 110: 16–21.LinkGoogle Scholar54 Lindholt JS, Vammen S, Juul S, Fasting H, Henneberg EW. Optimal interval detection and surveillance aortic aneurysm. *Eur J. Vasc Endovasc Surg*. 2000; 20: 369-373.CrossrefMedlineGoogle Scholar55 McCarthy RJ, Shaw E, Whyman MR, Earnshaw JJ, Poskitt KR, Heather BP. Recommendations for screening intervals for small aortic aneurysms. *Br J Surg*. 2003; 90: 821-826.CrossrefMedlineGoogle Scholar56 Powell JT, Brady AR. Detection, management, and prospect of medical treatment of small abdominal aortic aneurysms. *Arterioscler Thromb Vasc Biol*. 2004; 24: 241-245.LinkGoogle Scholar57 Stonebridge PA, Draper T, Kelman J, Howlett J, Allan PL, Prescott R, Ruckley CV. The rate of growth of the infrarenal aortic aneurysm. *Eur J. Vasc Endovasc Surg*. 1996; 11: 70-73.CrossrefMedlineGoogle Scholar58 Vardulaki KA, Prevost TC, Walker NM, Day NIE, Wilmink AB, Quick CR, Ashton HA, Scott RA. Growth rate and risk of rupture of the abdominal aortic aneurysm. *Br J Surg*. 1998; 85: 1674–1680.CrossrefMedlineGoogle Scholar59 Santilli SM, Littooy FN, Cambria RP, Rapp JH, Tretnyak AS, d'Audiff AC, Kuskowski MA, Roethle ST, Tomczak CM, Krupski WC. Expansion rate and results for 3.0 cm to 3.9 cmfrarenal abdominal aortic aneurysm. *J. Vasc Surg*. 2002; 35: 666-671.CrossrefMedlineGoogle Scholar Scholar

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