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Screening Program of Abdominal Aortic Aneurysm

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Abstract

In Europe, the prevalence of abdominal aortic aneurysms (AAAs) in the elderly population (≥65 year old) has declined in the past decades to <4%. Aneurysmal degeneration of the aorta is a serious and potentially life-threatening vascular disease. Abdominal aortic aneurysms typically develop subclinically and often only become symptomatic when complicated by impending rupture. Most AAAs are discovered incidentally while investigating for an unrelated pathology. Ruptured AAA is the tenth leading cause of death in Belgium (0.32% of all deaths in 2014). Health-care providers have emphasized the importance of early detection of AAA and elective repair when the rupture risk outweighs operative risk (usual diameter threshold of 55 mm). Routine AAA screening programs, consisting of a single abdominal ultrasonography at the age of 65 years, aim to reduce the number of AAA-related deaths. Does population-based ultrasound screening for AAA achieve its objective and is it cost-effective? This literature review tries to answer these challenging questions.

Keywords

abdominal aortic aneurysm, preventive medicine, screening program, follow-up, ultrasound, cost-effectiveness

Introduction

Community-based or nationwide screening programs for the prevalence of abdominal aortic aneurysms (AAAs) have been introduced by health-care authorities in many countries[1] (eg, Sweden in 2006,[2] England in 2009,[3] and the United States in 2008).[4,5] In the 90s, standard screening programs usually enrolled men in their 65th year of age for a single ultrasonography (US) of the infrarenal abdominal aorta.[1,3] But today, most recent guidelines recommend to limit AAA screening for men and women, aged 65 years or older, whoever smoked or have a family history of AAA.[6,7] In well-organized screening programs, an attendance rate of 70% to 80% is achieved.[2,8-15] Some measures could optimize the attendance rate, such as invitation to screening by the general practitioner rather than from a hospital or national screening program, improved public awareness of AAA disease by a better advertising campaign, and screening US availability close to patients' homes.

Detection of AAA using US has been shown to be accurate and reliable. It is an inexpensive, noninvasive, and effective imaging modality.[12] The infrarenal aorta is considered aneurysmal if the anteroposterior diameter exceeds 30 mm.[13] This fixed diameter (30 mm) is universally applied in screening programs. It has the shortcoming not to consider the widening of the aorta in relation to adjacent aortic segments nor in relation to aging, sex, or body size. Considering the rather small proportion of AAA-related deaths (0.32% of all deaths of citizens aged ≥65 years in Belgium in 2015),[16] the long-term merits of systemic mass screening for AAA are not self-evident.

Three issues will be debated in this review: What is the current prevalence of AAA in Western populations? Once an AAA is screen detected, what should be the optimal surveillance program? Does a single US screening for AAA improve life expectancy?

Prevalence of AAA

In the 90s, population-based screening studies estimated that approximately 3.5% to 7.7% of men aged 65 to 74 years had an

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AAA.[12,14,17-19] For women, the prevalence was much lower (1%-1.5%).[20,21]

Most (90%) of the screening-detected AAAs are small and do not reach the critical cutoff diameter size of 55 mm, generally accepted as an indication for prophylactic endovascular or open surgery.[2,9-11] In the largest AAA screening trial (Multicenter Aneurysm Screening Study [MASS], 1997-1999),[10] a ≥55 mm AAA was detected in 0.6% of the 65- to 74-year-old screened men. Similarly, low percentages of >55 mm AAAs were noted in the Western Australian trial (0.4%),[11,16] in the Malmö study (0.14%),[2] in the Danish VIVA (Viborg Vascular) trial (0.3%),[14] and in the Gloucestershire Aneurysm Screening Program (GASP).[15] The AAA (≥30 mm) detection rate increased when screening elderly patients. In the Western Australian trial, the prevalence reached 10.8% for the elderly (70-83 years old) screened men.[11] In the Uppsala screening study (2006-2007), the AAA prevalence rose from 1.5% at 65 years of age to 2.4% at 70 years of age.[2] For self-referring men with a mean age of 73 years, the prevalence of AAA attained 4.1%, compared with 1.4% in the cohort of men at age 65.[22]

In the second half of the 20th century, AAA had a steady increase in prevalence and mortality. However, the prevalence of AAA has been dramatically decreasing during the past decades. In the Central Sweden screening program (2006-2015),[2] US detected AAA (≥30 mm) in only 1.5% of the screened 65-year-old men. Adding 0.5% of the invited men who had been previously operated or were already followed for an AAA results in an overall 2.2% prevalence of AAA in men aged 65 years. The prevalence of large AAAs (≥55 mm) dropped to 0.14%. In the Danish Viborg Study, ≥50 mm AAAs were detected in 0.5% of all screened men aged 64 to 73 years.[9]

This decrease in prevalence of AAA has also been reported in New Zealand (a 50% drop from 1990 to 2007) and in England (a 30% drop from 1996 to 2007).[23] In the GASP, the prevalence of US screen-detected AAA of ≥3.0 cm among men aged 65 years decreased from 5.0% in 1991 to 1.3% in 2015.[15] A possible explanation for the reduced prevalence of AAA could be an improved control of risk factors for AAA development. A steep decline in cigarette consumption per person per year has been observed in the past decades, as well as more efficient antihypertensive and lipid-lowering treatment.[2,22] Lederle[24,25] described an important increase in AAA disease between 1940 and 1975 (a period when cigarette smoking was common and fashionable), culminating in a prevalence of 4.5% in males at age 65 years. But, in 2015, the prevalence declined to 2%; this coincides with a drop of the proportion of daily 65-year-old male smokers from 32% in 1980 to 11% in 2007.

There is a strong link between smoking behavior and aneurysmal degeneration of the aorta.[25,26] Ever-smoking increases the risk of developing AAA by 3- to 4-fold. The relative risk for developing an AAA increases in function of the amount of cigarette smoking (half pack a day during 10 years: odds ratio [OR] = 2.6 and more than 1 pack a day for 35 years, OR = 12.13).[6] Changing habits among smokers (reduced cigarette consumption per day) could logically reduce their risk of AAA development and growth. This has never been analyzed in large studies. This led to a modified strategy of the US Veteran AAA Screening Program. Today, the US Preventive Services Task Force Guidelines recommend a one-time US screening for men and women aged 65 to 75 years who have ever smoked.[4,6] Another target group for selective screening is the first-degree relatives of patients with AAA. A family history of AAA increases the risk of AAA 2-fold. Female relatives are exposed to a 2.2-fold and male relatives to a 1.7-fold risk of developing an AAA, compared with the estimated sex-specific population risk.[27] For this reason, the Society for Vascular Surgery (SVS) recommend AAA screening in men and women aged 55 years with a family history of AAA.[6] The European Society for Cardiology (ESC) also advises screening of women, aged >65 years, with a history of smoking or with a family history of AAA, besides screening all men, aged 65 to 75 years.[7] On the other hand, patients with diabetes have a lower incidence of AAA (OR = 0.80-0.50), a lower growth rate (1.7 mm/yr vs 5.0 mm/yr for nondiabetics), and a reduced risk of rupture.[28-30] A recent meta-analysis of 19 studies analyzing AAA growth rate in patients with and without diabetes shed some light on the protective effect of diabetes on aneurysmal degeneration of the aorta.[28] In that study, the yearly expansion rate of AAA in diabetics was 0.5 cm slower, compared with AAAs in nondiabetic patients. A possible explanation could be the excess of vascular matrix deposition and increased collagen synthesis (aortic wall stiffness), characteristic of diabetic arteriopathy, counterbalancing the proteolytic process, and thinning of the aortic wall. Enzyme glycation causes a downregulation of matrix metalloproteinases, key enzymes in the process of aneurysmal degeneration of the aorta.[31] This makes the benefit of AAA screening among diabetic patients less evident. The increased prevalence of diabetes in the Western world could partly explain the decreased prevalence of AAA. Its impact on the reduced prevalence of AAA is however less than the impact of declined tobacco use. Lipoprotein(a) [Lp(a)] has recently been linked to aneurysmal degeneration of the aorta, with an Lp(a) level 2-fold higher in patients with an AAA, compared with controls.[32] The Lp(a) carries oxidized phospholipids, implicated in endothelial dysfunction and monocyte recruitment.[33] It could be considered as a possible marker of the risk to develop an AAA. The Lp(a)-lowering drugs are under investigation.[34] The use of statins is today recommended since it restrains the expansion rate of AAA and its rupture risk (a 25% relative risk reduction)[35-37] by attenuating oxidative stress and inflammation inside the aortic wall.[38] The role of statins in the growth rate of AAA could not be confirmed in other observational studies.[39] The role of inflammation in the pathogenesis and expansion of AAA has also been correlated with the plasma level of human C-reactive protein (CRP).[40] However, CRP lacks specificity to consider it as a valuable marker of AAA disease progression.[6]

The principle of a single one-time US screening for men who are 65 year old (NHS Abdominal Aortic Screening Program, set up in 2009 in England[3]) was based on an initial

conclusion of the Gloucestershire study in 2001. This stated that a single normal US scan at the age of 65 years rules out the risk of clinically relevant AAA disease for life.[41,42]

However, in the Chichester study, a cohort of 4308 men with an AAA was originally screened as "normal" at the age of 65 years, but 166 (3.8%) of them evolved to AAA disease over a 5-year follow-up (detected either incidentally or by rescreening).[8] Individuals screened at the age of 65 years, presenting at that time a non-AAA (diameter <30 mm), will probably live long enough to develop a "de novo" latent aneurysm as time goes by. This is also well illustrated in the MASS trial by a noticeable increase in aneurysm ruptures after 8 years among patients with a "normal" (<30 mm) screen at the age of 65 years.[43] One can conclude that a nonaneurysmal aorta at the age of 65 years does not permanently eliminate the risk of AAA rupture (rescreening at 5-8 years seems justified).[6,43]

Surveillance of Screening-Detected AAA

Once an enlarged infrarenal aorta (≥30 mm) has been detected at a screening visit, control visits are to be scheduled at regular intervals based on the initial diameter of the AAA.[1,7,18] Rescreening intervals to check for AAA expansion shorten as the AAA enlarges. According to the Gloucestershire study, a second US should be proposed at 12 months for small (30-44 mm) AAAs and for subaneurysmal aortas (2.6-2.9 cm). The delay for repeating US is reduced for larger AAAs: a control at 6 months for 45 to 49 mm AAA and at 3 months for 50 to 55 mm AAA.[15,41] Once the diameter exceeds 55 mm, elective repair should be considered, since the annual rupture risk approaches 10% and increases exponentially for larger AAAs. The ESC recommends US control every 3 years for 30 to 39 mm AAA, every 2 years for 40 to 44 mm AAA, and every year for 45 to 54 mm AAA.[7] The SVS 2018 guidelines suggest a rescreening protocol consisting in a 3-year interval for surveillance of 3.0 to 3.9 cm AAAs, 12-month interval for 4.0 to 4.9 cm AAAs, and 6-month interval for 5.0 to 5.4 AAAs.[6]

The surveillance of an AAA detected at baseline screening is not always complete, since 15% did not comply for a subsequent appointment for rescreening in the MASS study[41] and the Gloucestershire program[41] for various reasons.

The small subgroups of patients with subaneurysmal disease (25-29 mm) require a US control at 5 years.[2,8,15,17] Such moderately enlarged aortas (defined as "aneurysms in formation") concerned 2.5% of all screened men in the Chichester study,[6] 1.8% in the Uppsala county screening program,[2] and 1.5% in the GASP.[15] During the long-term follow-up (13 years) of the MASS trial, AAA rupture or repair of "subaneurysmal" aortas at initial baseline US appeared to start increasing from the 8-year follow-up.[43]

This need for surveillance of "subaneurysmal" aortas was well illustrated in the Uppsala county screening program.[2] Out of the 40 patients with a borderline "subaneurysmal" aorta (25-29 mm diameter), 53% expanded to an AAA (≥30 mm) within 5 years. For smaller aortas (<25 mm), only 0.7% progressed to AAA within 5 years. An analysis of the screening data in the GASP over the past 25 years revealed that 57.6% of the men with a subaneurysmal aorta (2.6-2.9 cm) at the age of 65 years (1.5% of all screened men) will develop an AAA of 3.0 cm or larger within 5 years and 28% will develop a large AAA of ≥5.5 cm within 15 years.[15]

Screening programs, and subsequent surveillance when indicated, can be criticized for needless disease labeling, inducing patient anxiety.[44] This suggested mental impact should be minimized. In the Viborg trial, patients found to have an AAA on screening US experienced some anxiety for a short period after screening. However, this mental effect resolved within a few months.[7] In the Danish VIVA trial,[14] only 6% of the men enrolled in follow-up for AAA had increased psychological stress. In the MASS trial, the self-related mental health status did not differ between positive and negative screened men. No adverse emotional effects were apparent.[10,43] This is in contrast to screening-detected breast cancer, in which half of the patients report psychological distress during the first year after diagnosis.[45]

Long-term follow-up revealed that half of the screening-detected AAAs among men under surveillance will not rupture nor require repair over a 13-year period, since these AAAs remained stable or did not reach the threshold diameter of 55 mm.[9,43] Another inherent risk of screening programs is the temptation of overtreatment by operating AAAs below the 55 mm threshold. Vascular surgeons should resist this temptation and remain adherent to the guidelines of scientific societies.[6,7,46] There is currently no evidence to operate (open or endovascularly) on small (40-54 mm) AAAs, as evidenced in the Aneurysm Detection and Management trial,[47] the Comparison of Surveillance versus Aortic Endografting for Small Aneurysm Repair trial (CAESAR trial),[48] and the Positive Impact of endoVascular Options for Treating Aneurysm earLy study (PIVOTAL study).[49]

Long-Term Benefit of AAA Screening

In order to analyze the potential impact of AAA screening on longevity, we refer to 4 population-based randomized controlled trials of the 90s.[18,50,51] These trials investigated the long-term outcome of 64- to 83-year-old men, invited to an US AAA screening, compared with a control group (unscreened cohort) who received standard care[33,34]: MASS (1997), Chichester trial (1988), Viborg County-Denmark (1994) trial, and Western Australia (1996) trial.

All these trials demonstrated that US AAA screening results in a reduction of AAA-related mortality in men aged 65 to 74 years (OR = 0.66).[18,50] The only screening trial conducted in women was the Chichester trial,[16] which could not demonstrate a reduction in AAA rupture among women after either 5 or 10 years of follow-up.

The largest trial with the longest follow-up is the MASS trial, carried out in the United Kingdom from 1997 to 1999.[10,43] In 2012, the 13-year follow-up revealed an increased rate of elective AAA repair in men subjected to screening. Out of 33 883 screened men, aged 65 to 74 years, 1334 (4.9%) had

an AAA (≥30 mm), resulting in 600 elective repairs over a 13-year follow-up. This exceeds the elective repair (n = 277) of incidentally identified AAA in the unscreened group (33 887). In the Western Australian trial[11] and the Danish VIVA trial,[14] twice as many of the screened men underwent elective surgery at 43 months of follow-up, compared with the control group. In the Uppsala county screening program[2] and the Danish VIVA trial,[14] 50% of screening-detected aneurysms (≥3.0 cm) were electively repaired during a 5-year follow-up. In the MASS trial[43] and Viborg trial,[7] this percentage reached 25%.

On the other hand, this increased rate of elective AAA repair resulted in a lower rate of emergency repair for ruptured AAA.[17] In the MASS trial, 80 of the 33 883 screened men (0.24%) underwent repair for ruptured AAA versus 166 ruptured AAAs in the nonscreened control group (0.48%). This means a relative risk reduction of AAA rupture of 57%, corresponding to an absolute risk reduction of 6 of 10^4 screened men, compared with the nonscreened group.[51] Similar outcomes have been reported in the Viborg study with fewer emergent procedures (hazard ratio [HR]: 0.44) and more timely elective repairs (HR: 2.0) in the screened group.[9]

The operative mortality rate of AAA repair is reduced by a factor of 10 when performed as an elective rather than as an emergency procedure.[52] This should be taken into account when estimating the beneficial impact of screening on AAA-related mortality. The beneficial effect of AAA screening in terms of reduction in AAA-related mortality supposes a low operative mortality of elective AAA repair in the referral hospital (<5% for open repair and <2% for EVAR).[6,47] Diabetic patients are exposed to an increased operative risk (OR = 1.32) due to comorbidities. This renders the beneficial effect of AAA screening among diabetic patients less evident. For diabetic patients with a 50 to 55 mm AAA, a surveillance approach would be preferable to an aggressive treatment strategy. There are arguments to upgrade the threshold for AAA repair to 60 mm in diabetic patients.[28-30]

The MASS screening program led to a significant decline in AAA-related mortality (including fatal rupture and 30-day operative mortality of elective or emergent AAA repair) by 42% (relative risk reduction).[43] At the end of the 13-year follow-up, only 0.66% of the screened men died from AAA (rupture or procedural death) compared with 1.12% of the control group (a 0.46% absolute risk reduction). A similar and substantial benefit was obtained in the Danish Viborg study with a 66% relative risk reduction of AAA-related mortality at 13-year follow-up.[9] In contrast, in the Western Australia screening study, only an 8% decline in AAA-related mortality was observed over a 13-year follow-up period (0.46% AAA-related mortality for screened men [65-74 years] vs 0.51% for controls).[53] This moderate effect could be explained by the fact that the Western Australian trial enrolled men up to 83 years old. The benefit of screening decreases with aging and is less evident for frail high-risk patients with severe comorbidities and higher operative mortality.[11,18,53] This negatively influences the expected positive effect of a screening program in elderly patients.

Over time, the benefit of an AAA screening by a single US performed at the age of 65 years becomes more evident. Screening and surveillance reduce AAA rupture, since more AAAs are operated earlier and electively. According to the 4-year follow-up of the MASS trial, 710 men need to be screened to prevent one AAA-related death.[10] At 13-year follow-up, the beneficial impact on reducing AAA-related mortality improved, with only 216 men (65-74 years old) to be screened to prevent one AAA-related death.[43] In a Canadian screening program, the number needed to screen (NNS) was 625.[54] Combined screening for AAA, peripheral arterial disease, and hypertension (Danish VIVA trial)[14] resulted in an even more beneficial effect: The NSS was 169 to save one life. These NNS compare favorably to the NNS of 1887 in breast cancer screening programs.[55] Less convincing results were obtained in the Western Australian trial with a nonsignificant difference in AAA-related mortality at 13-year follow-up for both screened and control groups (0.46% and 0.51%, respectively). This means that 3290 men (65-74 years old) needed to be screened to prevent a single AAA-related death.[53]

One should not forget that the majority (60%-90%) of men with a screening-detected AAA will ultimately die from an unrelated cause.[2,11,43] Most studies could not demonstrate a significant difference in all-cause mortality between screened and nonscreened men.[20,43] A recent meta-analysis of the long-term results of 4 randomized trials evidenced a moderate 3% relative reduction (HR = 0.97) of all-cause mortality in screened patients after a follow-up of 15 years.[50] Combined screening for AAA, peripheral arterial disease, and hypertension (Danish VIVA trial)[11,14] resulted in a 5.5% relative risk reduction of all-cause mortality after 4.4-year follow-up, mainly due to risk factor management and adequate treatment. Attenders (participants who accepted the invitation for AAA screening) had the additional benefit of adequate counseling on lifestyle, such as risk factor control, medication, and health management, all explaining in part the moderate reduction of all-cause mortality.

The smaller Chichester trial had some contradictory outcome results.[17] Over time, the benefit of AAA screening fell from a 42% risk reduction in AAA-related death at a 5-year follow-up, respectively, to a 21% and 11% relative risk reduction at 10- and 15-year follow-up. Enrollment up to 80 years of age in the Chichester trial partially explains the vanishing long-term benefit, with aneurysmal degeneration of aortas that were nonaneurysmal at the initial screening echography. In addition, these elderly patients under surveillance for AAA are occasionally classified as unfit for surgery. For these elderly frail patients (≥75 years old), the screening program could not impede AAA-related mortality. These negative results raised some concern about the merit of mass screening for AAA among elderly men.

The American Heart Association (AHA) Guidelines recommend against screening among women, elderly men (aged ≥75 years), or men aged 65 years who never have smoked.[56] Regarding women, the low prevalence of AAA and the higher operative mortality rate outweigh any benefit of screening.[20,21]

The 2014 Guidelines of the ESC advise to include female relatives of patients with AAA and female 65-year-old smokers in the targeted screening programs.[7] Analogous recommendations were published in 2018 by the SVS.[6] It is well known that AAA in women are more prone to rupture and that the threshold to operate AAA in women is lower than for men.[56,57] Such a more selective targeted screening program could be more beneficial in terms of cost-effectiveness and expected gain in quality-adjusted life years (QALY). The ESC, the SVS, and the AHA recommend systematic AAA screening during routine transthoracic cardiac echography, hoping to increase cardiologists' awareness of the prevalence of AAA in the sixth decade of life.[6,7,58]

The final question is the cost-effectiveness of a nationwide screening program. What is the estimated cost per extra gained QALY? In the MASS trial, AAA-related death was halved in the screened group at a cost of 130 euros for every men screened.[43] In the Viborg County trial,[9] screened men obtained a 0.07 QALY gain after a 14-year follow-up at a cost of 179 euros/person/yr. Analogous data were obtained in the recent Malmö screening program with a cost of 169 euros/person/yr and a 0.011 QALY gain for screened patients.[59] The cost per QALY gained was estimated at 15 710 euros. In the MASS study, screening for AAA added a QALY at a cost of 42 600 euros at 4-year follow-up (costs included initial screening, surveillance US, elective or emergency AAA repair).[60] At 10-year follow-up, the cost per life year gained decreased to 11 400 euros. The more favorable cost-effectiveness of screening over the long term can be explained by the fact that the positive impact of screening at the age of 65 years continues to generate additional life years as time proceeds.

Cost analyses are complex. Should the screening cost be limited to the act of US (currently 61.20 euros in Belgium) or should secretarial workload, rent for office, depreciation of the technical equipment, surveillance US, and additional elective repair of screening-detected AAA also be considered as cost drivers?

The closer surveillance of screened patients, once an AAA (>30 mm) is identified, could result in a better control of cardiovascular risk factors, improved disease-modifying behavior (such as smoking cessation, weight control, hypertension control), and optimized medical treatment (statins and aspirin), contributing to the beneficial impact of screening on longevity.[14]

During the past decade, the absolute screening benefit in reducing AAA-related mortality has become less evident than what was estimated in the randomized trials of the 90s. The lower detection rate of AAA in recent screening programs raises doubts on the utility and cost-effectiveness of mass screening nowadays.[12,44] In New Zealand, the risk of dying from a ruptured AAA for a 65-year-old man fell by 28% (from 1.9% to 1.3%) between 1992 and 2007.[23] In 2015, according to the data obtained from the Belgian Ministry of Public Health (office of national statistics), only 0.32% of all deaths of citizens aged 65 years or older was registered as "aneurysm-related."[16] The same percentage (0.33%) for aneurysm-related mortality has been reported for the unscreened control group in the MASS trial.[43] This is probably an underestimation of the real AAA-related mortality since miscoding of causes of death is inevitable. Death certificates can be criticized for their inaccuracy. Sudden death by hypovolemic shock as a consequence of a ruptured AAA is often inaccurately categorized on the death certificate as cardiac death, and operative mortality following AAA repair is not always reported as "AAA-related death."

Conclusions

Nowadays, the implementation of an AAA screening program continues to be debated. Cost containment in public health care demands an appropriate selection of public spending. The AAA screening competes with other prevention priorities in primary care, such as early detection of cancer. Today, the Belgian ministry of public health maintains some reservations toward mass screening for AAA. Until now, nationwide AAA screening is not organized nor government sponsored in Belgium.

Currently, there is a willingness to pay up to 40 000 euros per QALY (generally considered as the cost-effectiveness cut-off).[59] Considering this criterion, AAA screening among men aged 65 years can be evaluated as cost-effective since its cost is estimated at 11 400 euros per QALY gained.[61] Screening for AAA is expected to be worthwhile especially in countries where smoking rates are high. Screening provides borderline or no benefit for women (except women with a family history of AAA or women who ever smoked) and for >75-year-old men.[53,62] Surveillance is not only mandatory for screening-detected AAAs ($\geq$30 mm) but should also include subaneurysmal aortas (25-29 mm) at large intervals. Finally, AAA screening offers an opportunity to start lipid-lowering and antiplatelet therapy as well as smoking cessation counseling. An update on the usefulness of AAA screening was recently published and approaches our critical analysis.[63,64]

This theoretical approach on the merit of AAA mass screening should be attenuated since the real gain in survival obtained by screening men aged 65 to 74 years is only some months (0.07 QALY) in the Viborg trial at 14-year follow-up.[9] There are arguments to change from mass screening toward targeted screening for AAA in patients at risk (smoking history, family history, cardiovascular occlusive disease).[65]

Authors' Note

All authors made (1) substantial contributions to conception and design, or acquisition of data, or analysis and interpretation of data; (2) drafting the article or revising it critically for important intellectual content; and (3) final approval of the version to be published.

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