Review

Systematic Review: Repair of Unruptured Abdominal Aortic Aneurysm
Frank A. Lederle, MD; Robert L. Kane, MD; Roderick MacDonald, MS; and Timothy J. Wilt, MD, MPH

Background: Recent recommendations to screen for abdominal aortic aneurysm (AAA) in high-risk populations and the rapidly increasing use of endovascular repair have led to increased interest in evaluating the effectiveness of treatment options for patients with AAA.

Purpose: To compare the effectiveness of treatment options, including active surveillance, open repair, and endovascular repair, for unruptured AAAs.

Data Sources: The authors searched MEDLINE, the Cochrane Library, and www.ClinicalTrials.gov through December 2006 with no language restrictions, searched reference lists, and queried experts and study authors.

Study Selection: Randomized trials that compared open or endovascular AAA repair with another treatment strategy and published clinical outcomes.

Data Extraction: Data were extracted onto standardized, piloted forms and were confirmed.

Data Synthesis: Two trials compared open repair with surveillance for small AAAs (n = 2226). Repair did not improve all-cause mortality (relative risk, 1.01 [95% CI, 0.77 to 1.32]) or AAA-related mortality (relative risk, 0.78 [CI, 0.56 to 1.10]). Four trials compared open repair with endovascular repair (n = 1532). Endovascular repair reduced 30-day mortality (relative risk, 0.33 [CI, 0.17 to 0.64]) but not mid-term (up to 4 years) mortality (relative risk, 0.95 [CI, 0.76 to 1.19]). One trial compared endovascular repair with observation in 338 patients who were unfit for open repair. Endovascular repair did not reduce all-cause mortality or AAA-related mortality, but high crossover and procedural mortality rates complicate interpretation of results.

Limitations: Few trials have been published. Those published were of small to moderate size and were not U.S. trials of endovascular repair.

Conclusions: Repairing AAAs smaller than 5.5 cm has not been shown to improve survival. Endovascular repair is associated with lower operative mortality than open repair, similar mid-term mortality, and unknown long-term mortality and has not been shown to improve survival in patients unfit for open repair. Long-term trial data comparing endovascular repair with open repair are needed, as is another trial comparing endovascular repair with observation in high-risk patients.


For author affiliations, see end of text.

Abdominal aortic aneurysm (AAA) is a common condition, occurring in approximately 1 in 20 older men who have ever smoked (1). Rupture of these aneurysms has a mortality rate of 80% (2) and causes 9000 deaths per year in the United States (3). To prevent rupture, elective repair of asymptomatic AAAs is performed in nearly 40 000 patients each year in the United States. However, these procedures result in about 1500 operative deaths (4). Because most AAAs never rupture (5), deciding when to electively repair an AAA, and by what method, can be difficult. Two forms of repair are available. Standard open surgical repair has been in widespread use for more than 50 years. More recently, endovascular repair was developed to provide a less-invasive alternative and is overtaking open repair in frequency of use (4).

The rapid increase in the frequency of endovascular repair and the recommendation by the U.S. Preventive Services Task Force to screen for AAA in high-risk populations (6) have led to increased interest in evaluating the effectiveness and adverse effects of treatment options for patients with AAA. We summarize and update parts of a larger review that was done at the request of the Agency for Healthcare Research and Quality (AHRQ) and America’s Health Insurance Plans (7). That review included and updated a systematic review by the National Institute for Clinical Excellence (8). We sought to determine the comparative effectiveness of treatment options, including active surveillance, open repair, and endovascular repair, for unruptured AAAs. Cost-effectiveness is addressed in a separate study (9).

Methods

Search and Selection Processes

We searched MEDLINE from 1966 through December 2006 using the exploded term aortic aneurysm limited to the publication type randomized, controlled trial with no language restrictions. We also searched the Cochrane Library and www.ClinicalTrials.gov for clinical trials of aortic aneurysm and reviewed titles and abstracts of identified references. We used reference lists and contacted content experts to identify additional reports. Clinical experts representing areas of vascular surgery; internal medicine; AAA epidemiology, diagnosis, and treatment; and systematic review methods served as members of a technical panel who provided advice throughout the study. When necessary, we requested additional information from the original authors.

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Figure 1. Literature search for selected studies.

Table 1. Studies included in the analysis of early mortality.

<table>
<thead>
<tr>
<th>Study Design</th>
<th>Treatment</th>
<th>Primary Outcome</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Randomized</td>
<td>Repair</td>
<td>Mortality</td>
<td></td>
</tr>
<tr>
<td>Surveillance</td>
<td>Observation</td>
<td>Mortality</td>
<td></td>
</tr>
</tbody>
</table>

AAA = abdominal aortic aneurysm.

**Study Selection**

Studies were eligible if the investigators randomly assigned patients who had an unruptured AAA to open repair or endovascular repair versus another treatment strategy (for example, the other repair procedure or active surveillance) and reported clinical outcomes. We did not include trials that reported on variations in method for 1 procedure (for example, type of incision for open repair or use of cell saver).

**Data Extraction and Quality Assessment**

We used previously published methods to determine the quality of randomized, controlled trials (10). We assessed the method of randomization, allocation concealment, blinding, and intention to treat.

Trained personnel extracted all data onto standardized, piloted forms, which were subsequently confirmed by the lead author. Discrepancies were resolved through discussion. Three design categories of randomized trials were identified: immediate repair versus surveillance for small AAA, open repair versus endovascular repair, and endovascular repair versus observation in patients who were unfit for open repair. Data on study characteristics and outcomes were extracted. The primary outcome of our analysis (and of most included trials) was all-cause mortality, which was calculated for all randomly assigned patients. Additional outcomes that we examined included operative mortality (at 30 days in patients having repair), quality of life, and adverse events and complications (treatment-related mortality and morbidity and the need for additional interventions). We also examined AAA-related mortality (deaths directly or indirectly attributable to AAA rupture or repair but not to thoracic aortic aneurysm) because this outcome has been widely discussed since the publication of the endovascular repair trials. However, AAA-related mortality is a problematic outcome because of the likelihood of ascertainment bias and because it was not specified as an outcome a priori in any of the endovascular repair trials (11–14). Deaths within 30 days after repair are all considered to be AAA-related, whereas late AAA ruptures are easily missed and are difficult to confirm (15), resulting in bias that favors the less-invasive strategy.

**Data Synthesis and Analysis**

Mortality data were pooled and analyzed by using Cochrane Collaboration Review Manager software (Nordic Cochrane Centre, Copenhagen, Denmark) (16). Heterogeneity between studies was assessed by using chi-square and $I^2$ tests—a $P$ value less than 0.1 and an $I^2$ value greater than 50%, respectively, were considered high (17). Relative risks and CIs were calculated with the random-effects models (18).

**Role of the Funding Source**

The AHRQ funded this study, and an AHRQ representative served as a task-order officer during the conduct of the full evidence report and provided comments on drafts of the full evidence report. The AHRQ formulated the initial study questions but did not participate in the literature search; determination of study eligibility criteria; data analysis or interpretation; or the preparation, review, and decision to submit the manuscript for publication.

**RESULTS**

The MEDLINE search for randomized, controlled trials identified 258 abstracts. After review of the abstracts and exclusion of nontrials and trials that did not compare open repair or endovascular repair of nonruptured AAA with another treatment strategy (Figure 1), we identified 7 trials that had reported results. All were published within the past decade. The search of the Cochrane Library and www.ClinicalTrials.gov returned 445 and 27 entries, respectively, and identified several studies in progress but no additional completed studies. The Table (11, 14, 19–32) shows the quality measures for the 7 trials. Blinding is not usually feasible in trials that compare surgery with other treatments, and no trial we identified was blinded. The trials will be discussed according to design category.

**Repair versus Surveillance for Small AAA**

Two randomized trials, the United Kingdom Small Aneurysm Trial (UKSAT) (19, 20) and the Aneurysm Detection and Management (ADAM) Veterans Affairs Cooperative Study (22, 23), compared all-cause mortality after immediate elective open repair of AAA with a policy of imaging surveillance with repair of AAAs that enlarged greater than 5.5 cm or became symptomatic ($n = 2226$). Both trials were of high quality according to the factors assessed in the Table. Few women were included, which reflects the 4:1 male preponderance of the disease (22) and, in the ADAM trial, the veteran population studied.
In the chi-square analysis shown in Figure 2, neither trial achieved a statistically significant difference in all-cause mortality. The trends favored surveillance in the ADAM trial and immediate open repair in UKSAT. In the UKSAT (21) report, the hazard ratio was borderline significant (0.84 [CI, 0.70 to 1.00]; \( P = 0.05 \)), although the authors speculated that this may have resulted from different rates of smoking cessation and noted that mean survival did not statistically significantly differ (6.5 years for surveillance vs. 6.7 years for immediate repair; \( P = 0.29 \)).

Figure 2 also shows the combined effects for the trials. There was no statistically significant difference in all-cause mortality (relative risk, 1.01 [CI, 0.77 to 1.32]) or AAA-related mortality (relative risk, 0.78 [CI, 0.56 to 1.10]), with statistically significant heterogeneity between studies (\( P = 0.03; I^2 > 78.5\% \)).

In both trials, differences in quality-of-life measures, when present, were small but tended to favor immediate open repair after 1 year, even though nearly all AAAs under surveillance were asymptomatic (20, 24).

A similar trial in Canada was discontinued because of inadequate enrollment, and we were unable to obtain outcome data on the 107 randomly assigned patients. We also identified 2 ongoing industry-funded trials comparing immediate elective endovascular repair with imaging surveillance in patients with small AAAs: 1 in the United States funded by Medtronic, Santa Rosa, California (33), and 1 in Europe funded by Cook, Bloomington, Indiana (34).

### Table. Characteristics of Randomized Trials of Abdominal Aortic Aneurysm Repair*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study, Year (Reference)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basic design</td>
<td>Open repair vs. surveillance</td>
</tr>
<tr>
<td>Primary outcome</td>
<td>All-cause mortality</td>
</tr>
<tr>
<td>Adequate randomization</td>
<td>Yes</td>
</tr>
<tr>
<td>Blinding</td>
<td>No</td>
</tr>
<tr>
<td>Allocation concealed</td>
<td>Yes</td>
</tr>
<tr>
<td>Intention to treat</td>
<td>Yes</td>
</tr>
<tr>
<td>Randomly assigned patients, n</td>
<td>188 (17)</td>
</tr>
<tr>
<td>Women, n (%)</td>
<td>188 (17)</td>
</tr>
<tr>
<td>Mean age, y</td>
<td>69.2 (60-76)</td>
</tr>
<tr>
<td>Mean diameter of AAA (range), cm</td>
<td>4.6 (4.0-5.5)</td>
</tr>
<tr>
<td>Mean follow-up (range), y</td>
<td>8 (6-10)</td>
</tr>
</tbody>
</table>

* AAA = abdominal aortic aneurysm; ADAM = Aneurysm Detection and Management Veterans Affairs Cooperative Study; DREAM = Dutch Randomized Endovascular Aneurysm Management; ESPAS = Eindhoven Stent Prosthesis for Aneurysm Study; EVAR = Endovascular Aneurysm Repair trial; EVR = endovascular repair; UKSAT = United Kingdom Small Aneurysm Trial.
† Randomized in a 3:1 ratio of endovascular repair to open repair.
‡ Range or SD is presented in parentheses.
§ Median values.

### Open Repair versus Endovascular Repair

Four trials have reported outcomes of open repair versus endovascular elective repair of large AAAs (\( n = 1532 \)) in patients who were candidates for both procedures (11, 14, 25–31). Three of the trials have published mid-term follow-up results: up to 2 years for the Dutch Randomized Endovascular Aneurysm Management (DREAM) Trial (mean, 21 months) (27); up to 4 years for the Endovascular Aneurysm Repair trial 1 (EVAR-1) (median, 2.9 years) (31); and up to 4 years for a small trial from Montréal, Canada (mean, 29 months) (14). The 2 larger studies, DREAM and EVAR-1, were of high quality and contributed most of the data. All 4 trials were conducted outside of the United States and began recruitment before 2001, and many of the devices used are not currently available in the United States. Figure 3 shows that endovascular repair reduced 30-day postoperative all-cause mortality compared with open repair (1.6% vs. 4.8%; relative risk, 0.33 [CI, 0.17 to 0.64]). There was no inconsistency among studies (\( I^2 = 0 \)). Each trial also found a statistically significant reduction in initial length of hospital stay with endovascular repair (combined weighted median, 6.2 days vs. 11.5 days).

The early mortality difference disappeared before 2 years in the DREAM and EVAR-1 trials, resulting in no statistically significant difference in mid-term all-cause mortality (Figure 3). Fewer deaths related to AAAs were reported with endovascular repair at mid-term (Figure 3),
but this finding should be interpreted with caution because of the likelihood of ascertainment bias, as discussed in the Methods section. Of note, the DREAM Trial did not consider deaths possibly attributable to rupture to be AAA-related if an autopsy was not done.

The findings for all-cause and AAA-related mortality did not show statistically significant variation with age, AAA diameter, renal function, or sex, although few women were enrolled.

In the EVAR-1 trial, reinterventions were required in 3 times as many patients who had endovascular repair as in those who had open repair, exceeding 20% at 4 years. The DREAM and Montréal trials showed a similar pattern, with more than twice as many patients in the endovascular repair group requiring reintervention. Assessment of reinterventions and adverse events in these trials seemed thorough and comprehensive, but differences in types of adverse events preclude meta-analysis. In the EVAR-1 trial, postoperative complications (for example, rupture, infection, thrombosis, or additional procedures) were 5 times more frequent with endovascular repair (17.6 vs. 3.3 repairs per 100 person-years). The DREAM Trial reported all severe or moderate adverse events (rather than only complications of AAA repair) and found nearly identical rates of event-free survival at 2 years in the 2 groups (83.1% for endovascular repair vs. 80.6% for open repair for severe adverse events and 65.6% vs. 65.9%, respectively, for moderate or severe adverse events).

The DREAM Trial reported quality-of-life (Short Form-36 [SF-36] and EuroQol) and sexual functioning data from the first 153 randomly assigned patients (28, 29). Scores for these measures favored endovascular repair in the early postoperative period. At 3 months, sexual function had returned to baseline values in both groups, and at 6 months, quality of life was statistically significantly better in patients in the open repair group. In the EVAR-1 trial, SF-36 and EuroQol scores favored endovascular repair for the first 3 months, but no differences were observed thereafter (31). The small Eindhoven Stent Prosthesis for Aneurysm Study (ESPAS) saw similar differences in SF-36 and EuroQol scores at 1 month that disappeared by 3 months (25), whereas the Montréal study showed no differences in SF-36 scores (14).

We identified 2 additional ongoing trials of open repair versus endovascular repair that have not yet reported results: the Veterans Affairs Open versus Endovascular Repair (OVER) Trial for Abdominal Aortic Aneurysms and the French Anévrisme de l’aorte abdominale: Chirurgie versus Endoprothèse (ACE) trial. Another trial from Ontario, Canada, is in the planning or pilot phase.

Endovascular Repair versus Observation in Patients Who Are Unfit for Open Repair

The EVAR-2 trial was a methodologically high-quality study and was the only randomized trial that compared endovascular repair with observation (32). Eligible patients had an AAA of 5.5 cm or greater and were candidates for endovascular repair but were judged to be medically unfit...
for open repair ($n = 338$). The study reported no statistically significant difference in the primary outcome of all-cause mortality between the 2 groups: The trend favored observation (hazard ratio, 1.21 [CI, 0.87 to 1.69]). There was also no statistically significant difference in AAA-related mortality (hazard ratio, 1.01 [CI, 0.55 to 1.84]). These findings did not show a statistically significant variation according to age, sex, AAA diameter, or renal function. Several aspects of the study have led to disagreement regarding the validity of the findings, including the following: 9 AAAs ruptured while awaiting repair in the endovascular group after a median of 98 days after randomization, 30-day mortality after endovascular repair in the endovascular group was high (9%), and 27% of patients in the observation group had AAA repair.

**DISCUSSION**

Our systematic review identified 7 randomized trials that compared open repair or endovascular repair with another treatment strategy for unruptured AAAs; all 7 trials were published within the past decade. Two of the studies compared immediate open repair with surveillance for small AAAs. Despite the inconsistency that was demonstrated by standard tests for heterogeneity in these 2 studies, their conclusions were similar to each other and to the finding of our analysis—namely, there was no benefit in all-cause mortality or AAA-related mortality associated with repair of unruptured AAAs that were smaller than 5.5 cm in diameter. The question of repair of small AAAs has been reopened with the launching of 2 industry-funded trials comparing endovascular repair with surveillance, but it will be difficult for these trials to show a benefit (for example, the Cook-funded trial [34] is designed to detect a very large 47% relative reduction in all-cause mortality at 4.5 years).

In comparisons of open repair with endovascular repair, published trials showed reduced operative mortality and early improvement in quality of life with endovascular repair. 

**Figure 3.** Randomized trials comparing endovascular repair with open repair of unruptured abdominal aortic aneurysm (AAA).

<table>
<thead>
<tr>
<th>Study, Year (Reference)</th>
<th>Endovascular Repair, n/n</th>
<th>Open Repair, n/n</th>
<th>RR (Random) (95% CI)</th>
<th>RR (Random) (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-day all-cause mortality</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DREAM, 2004 (26); 2005 (27)</td>
<td>2/171</td>
<td>8/174</td>
<td>0.25 (0.05–1.18)</td>
<td></td>
</tr>
<tr>
<td>ESPAS, 2001 (11)</td>
<td>1/57</td>
<td>1/19</td>
<td>0.33 (0.02–5.07)</td>
<td></td>
</tr>
<tr>
<td>EVAR-1, 2004 (30); 2005 (31)</td>
<td>9/532</td>
<td>25/518</td>
<td>0.35 (0.17–0.74)</td>
<td></td>
</tr>
<tr>
<td>Montréal, 2005 (14)</td>
<td>0/20</td>
<td>0/20</td>
<td>Not estimable</td>
<td></td>
</tr>
<tr>
<td>Subtotal (95% CI)</td>
<td>780</td>
<td>731</td>
<td>0.33 (0.17–0.64)</td>
<td></td>
</tr>
<tr>
<td>Total events</td>
<td>12</td>
<td>34</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Test for heterogeneity: chi-square = 0.14, $P = 0.93$, $I^2 = 0$

Test for overall effect: $Z = 3.32$ ($P = 0.0009$)

Mid-term AAA-related mortality

<table>
<thead>
<tr>
<th>Study, Year (Reference)</th>
<th>Endovascular Repair, n/n</th>
<th>Open Repair, n/n</th>
<th>RR (Random) (95% CI)</th>
<th>RR (Random) (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DREAM, 2004 (26); 2005 (27)</td>
<td>2/173</td>
<td>8/178</td>
<td>0.26 (0.06–1.19)</td>
<td></td>
</tr>
<tr>
<td>EVAR-1, 2004 (30); 2005 (31)</td>
<td>19/543</td>
<td>34/539</td>
<td>0.55 (0.32–0.96)</td>
<td></td>
</tr>
<tr>
<td>Montréal, 2005 (14)</td>
<td>1/20</td>
<td>0/20</td>
<td>3.00 (0.13–69.52)</td>
<td></td>
</tr>
<tr>
<td>Subtotal (95% CI)</td>
<td>736</td>
<td>737</td>
<td>0.53 (0.31–0.92)</td>
<td></td>
</tr>
<tr>
<td>Total events</td>
<td>22</td>
<td>42</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Test for heterogeneity: chi-square = 2.05, $P = 0.36$, $I^2 = 2.3$

Test for overall effect: $Z = 2.28$ ($P = 0.02$)

Mid-term all-cause mortality

<table>
<thead>
<tr>
<th>Study, Year (Reference)</th>
<th>Endovascular Repair, n/n</th>
<th>Open Repair, n/n</th>
<th>RR (Random) (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DREAM, 2004 (26); 2005 (27)</td>
<td>20/173</td>
<td>18/178</td>
<td>1.14 (0.63–2.09)</td>
</tr>
<tr>
<td>EVAR-1, 2004 (30); 2005 (31)</td>
<td>100/543</td>
<td>109/539</td>
<td>0.91 (0.71–1.16)</td>
</tr>
<tr>
<td>Montréal, 2005 (14)</td>
<td>4/20</td>
<td>3/20</td>
<td>1.33 (0.34–5.21)</td>
</tr>
<tr>
<td>Subtotal (95% CI)</td>
<td>736</td>
<td>737</td>
<td>0.95 (0.76–1.19)</td>
</tr>
<tr>
<td>Total events</td>
<td>124</td>
<td>130</td>
<td></td>
</tr>
</tbody>
</table>

Test for heterogeneity: chi-square = 0.72, $P = 0.70$, $I^2 = 0$

Test for overall effect: $Z = 0.46$ ($P = 0.65$)
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Repair but did not find a difference in mid-term all-cause mortality or quality of life. Improvement in mid-term AAA-related mortality was observed with endovascular repair, but this outcome may be unreliable because of the difficulty in identifying late AAA rupture and because it was not specified a priori in these trials. Endovascular repair is associated with more reinterventions and requires periodic imaging for the remainder of the patient’s life. Longer-term comparisons are needed to determine the relative effects of the 2 procedures on all-cause mortality. At least 4 trials, EVAR-1, DREAM, OVER, and ACE, are ongoing and should contribute to this assessment.

In the third category of published trials, endovascular repair versus observation for patients who are unfit for open repair, the only published randomized trial did not find a survival benefit from endovascular repair. This was disappointing because the clearest indication for endovascular repair had been assumed to be patients who are at higher risk from open repair. As noted, high crossover and procedural mortality rates have led to controversy regarding the validity of the EVAR-2 trial. It is uncertain whether these rates could be improved in another trial or whether they reflect the inherent difficulties of managing these very sick patients, as argued by the EVAR-2 investigators (32). Critics of the EVAR-2 trial, including the Society for Vascular Surgery (35), have generally drawn unfavorable comparisons with selected case series, but it is difficult to determine whether these case series are truly comparable with patients in the EVAR-2 trial. The debate seems unlikely to be settled without another trial, but to our knowledge, no additional trials are being planned.

The principal limitations of our review are the small number of randomized trials, the small to moderate size of the trials, and the absence of U.S. trials of endovascular repair.

From our review, we conclude that survival has not been shown to be improved by repairing AAAs that are smaller than 5.5 cm; endovascular repair is associated with lower operative mortality, similar mid-term all-cause mortality and quality of life, more frequent reinterventions, and unknown long-term outcomes compared with open repair; and endovascular repair has not been shown to improve survival in patients who have medical contraindications to open repair. Long-term data from randomized trials comparing endovascular repair with open repair are necessary and are expected from ongoing trials. Another randomized trial comparing endovascular repair with observation would be helpful in patients who are unfit for open repair.

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References

Copenhagen: The Nordic Cochrane Centre; 2003.

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