Objective: To determine whether the diameters of treated abdominal aortic aneurysms (AAAs) have changed during the last 10 years.

Design, Setting, and Patients: A retrospective record review was completed on all patients undergoing any AAA repair from January 1, 2000, through December 31, 2009, at a single high-volume institution. All cases of repaired AAAs that had computed tomographic scans within 3 months of surgery were included. The mean and median maximal diameters of AAAs were noted. Correlation and regression analyses were used.

Main Outcome Measures: The mean and median maximal diameters of AAAs.

Results: Of 360 patients with treated AAAs, 339 met the inclusion criteria. The mean (SD) diameter of repaired AAAs decreased from 6.49 (1.46) cm (median, 6.40 cm) in 2000 to 5.83 (1.23) cm (median, 5.60 cm) in 2009. Correlation analysis confirmed a decrease in diameter across years (Pearson $R = -0.141; P = .01$). A fitted regression line also showed a decreasing trend (slope $=-0.059$ cm per year; $P = .01$).

Conclusions: The diameters of repaired AAAs at our hospital have decreased progressively during the last decade. This observation is consistent with a reduction in the expansion rates of AAAs and may account for the progressive decreases in the aneurysm rupture rate in the United States.
Inclusion of Diseases, Ninth Revision (ICD-9) was used for tabulations of both diagnoses and procedures. The ICD-9 diagnosis code 441.3 was referenced for ruptured AAA and 441.4 was used for AAA without rupture. The ICD-9 procedure codes 38.34, 38.44, and 38.64 were used to capture all patients who underwent open AAA repair. The ICD-9 code 39.71 was used to determine the number of AAAs treated by endovascular aneurysm repair.

All patients undergoing AAA repair with computed tomographic scans within 3 months of surgery were included in the analysis. Those undergoing emergent repair without imaging studies or patients with computed tomographic scans older than 3 months were excluded. The size of the aneurysm was rechecked on the original images against the initial radiographic report; there was no discordance between reported maximal diameters and remeasured sizes. Relevant demographic data such as age, race, and sex were collected. Numerical variables were summarized by mean, standard deviation, and median for each year. Categorical variables were summarized by frequency and percentage.

Analysis of variance, Pearson correlation, and linear regression were used to assess changes in numerical variables across the years. The Fisher exact test and the Cochran-Armitage trend test were used to assess changes in binary variables across the years. A significance level of .05 was used throughout. We used SAS version 9.1.3 statistical software (SAS Institute, Inc) for statistical calculations.

RESULTS

Of 360 patients with treated AAAs, 339 met the inclusion criteria. The demographic data and number of AAAs treated per year are reported in the Table. Twenty-one patients were excluded from the study owing to lack of a proximate imaging study. The majority (14 patients [67%]) were in the first 5 years of the study. Excluded patients included 7 with ruptured AAAs; 5 other patients with rupture had proximate imaging studies and were included. There were no significant demographic differences between the small excluded group and the patients included in the study.

In the study group, the primary method of treatment changed from a predominance of open repair in 2000 (92%) to endovascular repair in 2009 (87%). No other significant demographic changes (age, sex, and race) were observed.

The mean (SD) diameter of repaired AAAs decreased from 6.49 (1.46) cm (median, 6.40 cm) in 2000 to 5.83 (1.23) cm (median, 5.60 cm) in 2009. Correlation analysis confirmed a decrease in diameter across years (Pearson $R = -0.141; P = .01$) (Table). A fitted regression line also showed a decreasing trend (slope $= -0.059$ cm per year; $P = .01$) (Figure). The prevalence of the largest AAAs (defined as $\geq 7$ cm) also decreased over time. For example, in 2000, 10 of 27 AAAs (37%) had diameters of 7 cm or larger, while in 2009, only 6 of 37 (16%) were this large. The Cochran-Armitage test (1-sided) confirmed a statistically significant decrease in large AAAs over time ($P = .007$). Comparing just 2000 with 2009 shows the shift to smaller mean and median sizes as well as the reduction in large AAAs overall.

COMMENT

This study demonstrates that the diameters of repaired AAAs in 1 high-volume medical center are decreasing. This change in the mean and median size of repaired AAAs in our hospital occurred during a decade in which the numbers of new AAAs diagnosed (about 53 000 yearly) and treated (about 42 000 yearly) were stable across the United States. Furthermore, the current clinical guidelines regarding the size threshold for aneurysm repair has, if anything, been revised upward from 4 cm to 5 or 5.5 cm based on a number of well-accepted studies of small aneurysm growth patterns and consensus guidelines from the Society for Vascular Surgery. This change in practice pattern would tend to promote larger AAAs in any surgical series, exactly the opposite of what we observed.

This reduction could be caused by a change in patient population owing to altered catchment area or referral patterns, but there is no evidence for either. Alternatively, the population of AAAs may simply be skewed toward smaller AAAs owing to reduced expansion rates. Such a trend could partially explain the 35% decrease in the rupture rate seen across the United States during the last decade.

The most likely explanation for this apparent change in the natural history of aneurysmal disease is risk fac-
tor modification, including decreased tobacco use and the use of statins and other lipid-decreasing regimens. For example, use of pharmacological lipid-decreasing treatment increased from 11.7% between 1988 and 1994 to 40.8% between 1999 and 2004; age-adjusted mean total cholesterol concentrations in men and women also declined from 212 and 208 mg/dL, respectively, to 199 and 197 mg/dL, respectively (to convert to millimoles per liter, multiply by 0.0259). A recent meta-analysis including nearly 700 patients from 5 studies showed that statin therapy was associated with lower expansion rates in small aneurysms. Others have documented changes in the specific metalloproteinases implicated in AAA rupture in the arterial walls of patients treated with statins. Recently, Karrowni et al demonstrated decreases in the AAA expansion rates in patients treated with statins. They focused on 211 patients in a Veterans Affairs hospital who had undergone serial imaging surveillance. Patients receiving statins had a decreased aneurysm growth rate compared with control subjects (0.9 vs 3.2 mm/y, respectively; \( P < .0001 \)).

There is evidence that other medications may contribute to slower expansion and/or AAA wall stabilization. A human case-control study by Hackam et al demonstrated that angiotensin-converting enzyme inhibitors are associated with a reduced risk of ruptured AAA. This effect was not seen in patients treated with other antihypertensive agents. In an animal model, treatment with angiotensin-converting enzyme inhibitors suppressed the development of elastase-induced AAAs in the rat. This effect was distinct from hemodynamic alterations and was attributed to the preservation of medial elastin.

In conclusion, the small but statistically significant reduction in the sizes of treated AAAs in our practice environment correlates with the reduction in AAA rupture rates seen in larger population-based studies during the same period. Acknowledging the limitations of a retrospective study in which definitive risk factor assessments and AAA expansion rates could not be determined, our study is consistent with the hypothesis that the natural history of aneurysmal disease has changed. Prospective validation and a larger sample will be required to confirm this observation.

Accepted for Publication: April 18, 2012.
Published Online: September 17, 2012. doi:10.1001/archsurg.2012.1151
Correspondence: Bruce L. Gewertz, MD, Department of Surgery, Cedars-Sinai Medical Center, 8700 Beverly Blvd, Ste 8215N, Los Angeles, CA 90048 (bruce.gewertz@cshs.org).

Author Contributions: Dr Gewertz had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Cossman and Gewertz. Acquisition of data: Hadjibashi. Analysis and interpretation of data: Mirocha and Gewertz. Drafting of the manuscript: Hadjibashi, Cossman, and Gewertz. Critical revision of the manuscript for important intellectual content: Mirocha and Gewertz. Statistical analysis: Mirocha. Obtained funding: Gewertz. Administrative, technical, and material support: Gewertz. Study supervision: Gewertz.

Financial Disclosure: None reported.

Additional Contributions: Ms Sherri-Marie Robinson helped in coordination between research members.

REFERENCES


