Eur J Vasc Endovasc Surg **25**, 408–411 (2003)
doi:10.1053/ejvs.2002.1850, available online at http://www.sciencedirect.com on SCIENCE DIRECT®

A Myth Exposed: Fast Growth in Diameter does not Justify Precocious Abdominal Aortic Aneurysm Repair

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Objectives: *fast growth of abdominal aortic aneurysm (AAA) diameter is claimed to be an indication for repair. We investigated the validity of this claim.*
Methods: *between January 1988 and October 2000, 277 patients have had duplex sonography at six-monthly intervals in our aneurysm surveillance programme. During this period fast AAA growth was not an indication for operation in our unit.*
Results: *we identified 63 patients whose aneurysms had grown 0.5 cm or more in 6 months. Thirty-one of the 63 patients had aneurysms measuring 5.5 cm or greater in anterior–posterior diameter after the fast growth and all have been operated on unless deemed not fit due to anaesthetic risk. The remaining 32 patients continued in surveillance for a total of 50 patient years and none had rupture of their aneurysm. The calculated 95% confidence interval for the risk of rupture was 0–6 per 100 patient years. Six patients, who would have been operated on if fast growth had been an indication, have been spared surgery of whom 3 died and 3 became unfit. Nine patients remained in surveillance at the end of the study.*
Conclusion: *our data support the view that rapid increase in AAA diameter is not an indication for elective AAA repair.*

Key Words: Abdominal aortic aneurysm.

Introduction

Fast growth of abdominal aortic aneurysm (AAA) diameter is claimed to be an indication for aneurysm repair.[1,2] As a consequence of this belief, which is supported by no valid evidence, elective repair of AAAs after a period of rapid growth has become established practice.[3–5]

A thoughtful and competent recommendation of elective repair of AAA is based on a knowledge of the natural history of the disease, individual patient risk factors and audited operative mortality data. A more conservative approach than previously advised is now generally advocated.[5] The rate of expansion of AAAs has been documented in surveillance programmes.[6,7] If fast growth is a valid indication for operative repair then an episode of measured fast growth should be followed by sustained rapid expansion and a high risk of rupture. Until now no data on the subsequent natural history of AAAs that have had a period of rapid growth have been available, since in all previously reported studies fast growth has been an accepted indication for elective surgery.[3–5]

Patients and Methods

Patients who had two or more six-monthly diameter measurements of their AAA were identified from our aneurysm surveillance programme data files. The surveillance programme commenced in January 1988 and is ongoing. Duplex sonography is performed at six-monthly intervals by full time, dedicated and experienced vascular technologists who record the maximum anterior–posterior diameter of the infrarenal aorta. During the study period from January 1988 to October 2000, fast growth of an AAA was not an indication for operation in our unit. The status of each patient was identified at October 2000. Patients continued in the surveillance programme until they left as a consequence of operation, death, patient choice, moving away, lack of medical fitness for elective repair, or advanced age (greater than 80 years if also infirm). Where a patient died, cause of death was deemed to be that found at autopsy or that stated on the death certificate if an autopsy was not performed. Patients who had not yet missed their next surveillance appointment were assumed to be

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1078–5884/03/050408 + 04 $35.00/0

alive. Growth rates were recorded by subtracting each pair of data points 6 months apart.

Results

We identified 277 patients with aneurysms initially less than 5.5 cm anterior–posterior diameter and they underwent 1362 scans. The median value of all size data for AAAs in the surveillance programme was 4.6 cm. There was no convincing case for an exponential regression fit of data concerning growth rate and size of aneurysm. Linear regression of growth rate and size of aneurysm was significant (ANOVA d.f. $= 1$, $p = 0.024 \times 10^{-14}$). The best fit straight line is described by the equation $Y = 0.059X - 0.11$ where Y is growth in cm per 6 months and X is the AP diameter in cm. The R value is 0.23. The median growth was 0.14 cm per 6 months (IQR 0.01–0.30).

We identified 63 patients whose aneurysms had grown 0.5 cm or more in any six-month period (Table 1).

Table 1. Anterior–posterior diameters of the 63 patients who experienced fast growth.

Patient	Less than 5.5 cm after fast growth		More than 5.5 cm after fast growth	
	Before fast growth	After fast growth	Before fast growth	After fast growth
1	3.0	3.9	4.7	5.6
2	3.4	4.0	4.8	5.5
3	3.8	4.7	4.8	5.5
4	4.0	4.6	5.0	5.8
5	4.0	4.7	5.1	5.8
6	4.0	4.7	5.1	6.0
7	4.0	4.5	5.1	5.6
8	4.1	4.7	5.2	6.0
9	4.1	4.6	5.2	5.9
10	4.2	4.7	5.2	5.8
11	4.2	4.7	5.2	5.7
12	4.3	4.9	5.3	5.8
13	4.3	4.8	5.3	5.9
14	4.3	4.8	5.4	6.1
15	4.4	5.0	5.4	6.6
16	4.4	5.2	5.4	5.9
17	4.4	5.0	5.5	6.0
18	4.4	5.1	5.6	6.5
19	4.5	5.2	5.6	6.1
20	4.5	5.0	5.6	6.2
21	4.5	5.2	5.7	6.4
22	4.6	5.1	5.7	6.2
23	4.6	5.1	5.7	6.2
24	4.6	5.2	5.7	6.2
25	4.7	5.2	5.7	6.2
26	4.7	5.3	5.9	6.6
27	4.7	5.2	6.0	6.5
28	4.8	5.3	6.3	6.9
29	4.8	5.4	6.4	7.0
30	4.9	5.4	6.6	7.3
31	4.9	5.4	7.4	8.3
32	4.9	5.4		

This represented the top 5% of all growth data. Twenty-seven per cent of patients who experienced fast growth were females, identical to the proportion of females in the whole surveillance population. Following the episode of fast growth, the growth rate regressed towards the population average rather than continuing at the same or an exponentially increasing rate (Fig. 1). Data showing subsequent growth after the episode of fast growth was available in 32 patients, with a total of 100 scans. The subsequent growth rate of this sub-group was not significantly different from our surveillance population for the equivalent size range (Z Test, $z = -1.92$, $p = 0.055$).

Thirty-one of the 63 patients had aneurysms measuring 5.5 cm or greater in anterior–posterior diameter after the fast growth. Twenty had their aneurysm surgically repaired, the remainder being deemed not suitable for elective repair due to anaesthetic risk (4) or advanced age (7). The remaining 32 patients continued in surveillance for a combined total of 50 patient years. None of these patients had rupture of their aneurysm. The calculated 95% confidence interval for risk of rupture was 0–6 per 100 patient years. At the end of the study period, the status of the 32 patients was as follows: 11 patients underwent operation because their aneurysm reached our size threshold and an additional four patients were operated on for symptoms attributable to their aneurysm, three patients were considered for operation but were deemed unfit, one patient with an eventual aneurysm diameter of 7.0 cm refused surgery, three patients died of non-aneurysm related causes, one patient left surveillance when he moved away, and nine remained under surveillance. The 30-day operative mortality of the patients who did not have surgery immediately in consequence of fast growth was 2 out of 15 and those that were operated because they reached our size threshold after an episode of fast growth was 1 out of 20. The operative mortality of these two groups of patients was not significantly different (χ^2 $p = 0.38$).

Discussion

Until the 1980s the mere diagnosis of an AAA was considered to be a sufficient indication for surgical repair.[1] The autopsy study of Darling[8] at the Massachusetts General Hospital from 1952 to 1968, reported that even small aneurysms were found ruptured. This paper is often incorrectly cited as supporting evidence for a strategy of operating on small AAAs. Nevitt *et al*.[9] challenged the validity of operative recommendations based on autopsy data because of selection bias inherent in referral for autopsy and the likely

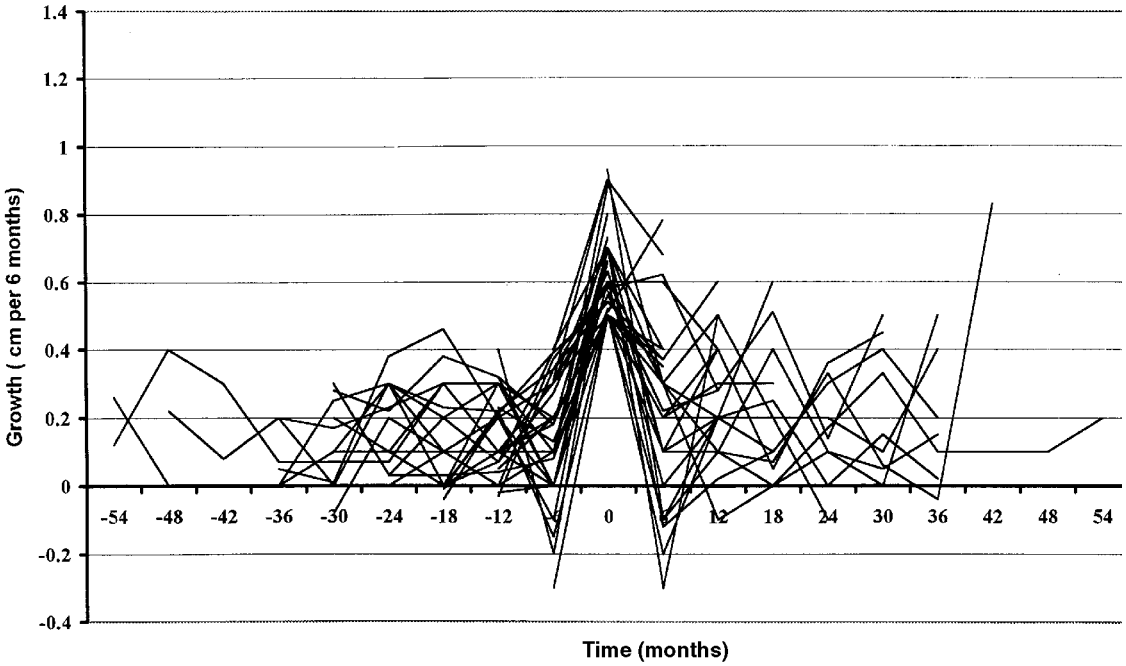

Fig. 1. Growth of aneurysm AP diameter in 63 patients with fast growth indexed by episode of first fast growth.

underestimation of *in vivo* aortic diameter from autopsy measurement.

Szilagyi *et al.*[10] showed that, in patients unfit for elective AAA repair, the cause of death was rupture in 31% of the patients who initially had aneurysms less than 6 cm in diameter and that death occurred within 2 years in the majority. Patients unfit for elective repair are, however, unrepresentative, including many with hypertension, obstructive airways disease, current smoking addiction, and other risk factors for AAA rupture.[11]

The ability to accurately measure AAA diameter with ultrasonography or computed tomography eventually provided the data informing the present more enlightened consideration of the balance of risk and benefits of elective AAA surgery.

Berstein *et al.*[12] reported ultrasonographically measured growth rates of aneurysms in patients initially deemed too unfit for elective AAA repair. His group subsequently proposed repair if an AAA expanded by more than 0.5 cm in 3 months, but it is not clear what proportion of patients they operated on for this indication.[1] Limet *et al.*[2] reported that aneurysms that went on to rupture had faster expansion rates than average for their study population of unfit patients. Rupture of the aneurysm occurred in 11 out of 114 patients under surveillance. They recommended repair for aneurysms of any size with markedly accelerated expansion rate, but did not define what this should be. Others such as Glimaker *et al.*[13] have not found an association between risk of rupture and expansion rate. Nevitt *et al.*[9] reported a study following 176 residents of Rochester with an AAA documented by ultrasonography but not immediately operated on; of these 11 patients subsequently ruptured their aneurysm but none had an aneurysm measured at less than 5 cm prior to rupture. They recommended surveillance of small aneurysms with elective repair being considered for aneurysms of 5 cm or more in diameter. The small aneurysm treatment trial[5] has subsequently confirmed that elective AAA repair is inappropriate for AAAs measuring less than 5.5 cm in anterior–posterior diameter.

Our surveillance programme is one of the largest reported in the literature from a single centre and may be the best data available on fast growth as other investigators have operated for fast growth. Our conclusion does not rely solely on the outcome of the patients in surveillance subsequent to fast growth but also on the observation that fast growth is not sustained. In our study, fast growth was sustained in only 11% (4/36) of the observations. Our study shows that after a period of fast growth (0.5 cm in 6 months) the rate of aneurysm expansion does not continue at an exponentially increasing rate or even the same rate but reverts towards the population average (Fig. 1). The most plausible explanation for our observations is likely to be the phenomenon of regression to the mean. The components involved in regression to the mean are known to be measurement errors, individual

physiological fluctuation and the expected random variance in any study population. Although formal studies of observer variability of aortic sonography have not been performed locally our technologists were experienced and dedicated vascular sonographers. Examining Figure 1, it can be seen that the time period immediately adjacent to the episode of fast growth shows a concentration of values that are negative. Given that aneurysms do not shrink this gives an indication of the maximum observer error i.e. 3 mm. This is consistent with values reported in the literature from studies of observer variability.[14] The very concept of fast growth may be challenged on the grounds that it is an artefact solely due to observer variation. The greater the observer variability the more likely it is that a diagnosis of fast growth is spurious. This does not detract from the conclusion of our paper that operating for an episode of fast growth is precocious. Awareness of regression to the mean allows the informed surgeon to predict that in any patient who has experienced a period of rapid aneurysm growth it is unlikely that it will be sustained in the next period of observation. Whatever the explanation, we have demonstrated that, following a period of rapid aneurysm growth to a maximum diameter of 5.5 cm, growth rates revert towards the average and the risk of rupture is low. Although we have data on only a small number of patients after an episode of fast growth the finding of a low risk of rupture is statistically robust provided our observation of no ruptures in the follow-up group is correct. This could be questioned as cause of death relies on death certification in these cases rather than autopsy and this may underestimate the cause of death by rupture due to misattributation.

The risk of rupture for patients who experience a period of fast AAA growth during surveillance is likely to be lower than the risk of elective surgery[5,15] provided the anterior–posterior AAA diameter measured by ultrasonography remains less than 5.5 cm. The second order question of whether repair is avoided or merely delayed in this group is interesting. Our numbers are perhaps too small to draw any valid conclusion but 19% had already become ineligible for operation by the end of the study. These patients benefited by not being exposed to the risk of an operation that they did not require. Forty-seven per cent subsequently had their aneurysms surgically repaired and for these individuals the benefit of deferring the risk of surgery is partially offset by the gradual inevitable decline in anaesthetic fitness with advancing age. The operative mortality for this group was not significantly greater for having been deferred in this small study. For the 28% who were still in surveillance at the end of the study period, it is not known what proportion would eventually come to surgery.

In our clinical practice excluding fast growth as an indication for aneurysm surgery has, to date, not resulted in the death of any patient or any emergency operation for aneurysm rupture. In conclusion, our data provide no support for the belief that rapid growth of an AAA is an indication for elective AAA repair.

References

1 Bernstein EF, Chan EL. Abdominal aortic aneurysm in high-risk patients; outcome of selective management based on size and expansion rate. *Ann Surg* 1984; **200**: 255–262.
2 Limet R, Sakalihanassan N, Albert A. Determination of the expansion rate and incidence of rupture of abdominal aortic aneurysms. *J Vasc Surg* 1991; **14**: 540–548.
3 Scott RAP, Ashton HA, Kay ND. Abdominal aortic aneurysm in 4237 screened patients: prevalence, development and management over 6 years. *Br J Surg* 1991; **78**: 1122–1125.
4 Brown PM, Pattenden R, Vernooy C, Gutelius JR. Selective management of abdominal aortic aneurysms in a prospective measurement program. *J Vasc Surg* 1996; **23**: 213–220.
5 The U.K. Small Aneurysm Trial Participants. Mortality results for randomised controlled trial of early elective surgery or ultrasonographic surveillance for small abdominal aortic aneurysms. *Lancet* 1998; **344**: 1649–1655.
6 Watson CJE, Walton J, Shaw E, Heather B, Collin J. What is the long term outcome for patients with very small abdominal aortic aneurysms? *Eur J Vasc Endovasc Surg* 1997; **14**: 299–304.
7 Lindholt JS, Vammen S, Henneberg EW, Fasting H, Juul S. Optimal interval screening and surveillance of abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg* 2000; **20**: 369–373.
8 Darling RC. Ruptured arteriosclerotic abdominal aortic aneurysms. A pathological and clinical study. *Am J Surg* 1970; **119**: 397–401.
9 Nevitt MP, Ballard DJ, Hallett JW. Prognosis of abdominal aortic aneurysms: a population-based study. *N Engl J Med* 1989; **321**: 1009–1014.
10 Szilagyi DE, Elliot JP, Smith RF. Clinical fate of the patient with asymptomatic abdominal aortic aneurysm and unfit for surgical treatment. *Arch Surg* 1972; **104**: 600–606.
11 Brown LC, Powell JT. Risk factors for aneurysm rupture in patients kept under ultrasound surveillance. *Ann Surg* 1999; **230**: 289–297.
12 Bernstein EF, Dilley RB, Goldberger LE, Gosink BB, Leopold GR. Growth rates of small abdominal aortic aneurysms. *Surgery* 1976; **80**: 765–773.
13 Glimaker H, Holmberg L, Elvin A, Nybaka O, Almgren B, Bjorck CG, Erikson I. Natural history of patients with abdominal aortic aneurysms. *Eur J Vasc Surg* 1991; **5**: 125–130.
14 Singh K, Bonaa KH, Solberg S, Sorlie S, Bjork L. Intra- and inter-observer variability in ultrasound measurements of abdominal aortic diameter. The Tromso Study. *Eur J Vasc Endovasc Surg* 1998; **15**: 497–504.
15 Bayly PJM, Matthews JNS, Dobson PM, Price ML, Thomas DG. In-hospital mortality from abdominal aortic surgery in Great Britain and Ireland: Vascular Anaesthesia Society audit. *Br J Surg* 2001; **88**: 687–692.

Accepted 18 December 2002