Unruptured intracranial aneurysms: benign curiosity or ticking bomb?

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15 years ago, the treatment of incidentally discovered intracranial aneurysms was straightforward with a good evidence base behind it. When intracranial aneurysms were identified, people were referred to neurosurgeons who would offer surgical repair if the patient was in reasonable health and had a good life expectancy. Since that time, several studies have given contradictory evidence for what should be done with these lesions, and a new technique for the repair of aneurysms, endovascular coil embolisation, has been developed. Here we review the research and make several recommendations. First, incidentally discovered aneurysms in the anterior circulation less than 7 mm in size in people with no personal or family history of subarachnoid haemorrhage should be left untreated. Second, people with remaining life expectancy of less than 20 years or so (ie, those over age 60 years) should be informed that from a statistical point of view the benefits of treatment do not outweigh the risks. Third, in all other cases treatment with surgical clipping or coil embolisation should be advised. And finally, if surgical treatment is not feasible then medical hypotensive treatment may be a viable alternative.

Data on prevalence, risk, and treatment outcome of an unruptured intracranial aneurysm (UIA) depend on the context in which they were gathered. From the mid 1970s to the 1990s most UIAs were diagnosed as “additional” to ruptured aneurysms found in patients presenting with subarachnoid haemorrhage (SAH) and surgical treatment was recommended. Several studies supported this advice but recent years have seen a shift in the range of UIAs diagnosed owing to changing patterns of investigation. This shift has brought more truly incidental UIAs—those without associated pathology—to light. The initial approach to these was to extrapolate recommendations from “additional” to “incidental” aneurysms. Whether such extrapolations are justifiable is at the centre of current debate and has been addressed by the recently published results of the International Study of Unruptured Intracranial Aneurysms (ISUIA).1,2

Imaging and diagnosis

Angiography

Cerebral angiography was introduced by Egas Moniz in 1927.3,4 It became a key investigation for various disorders, not all of them vascular. The technique is sensitive to intracranial aneurysms and the number of UIAs discovered increased with its use. CT replaced angiography and ventriculography as the main diagnostic tool of neuroradiology in the 1970s, but angiography was still a principal investigation of neurovascular diagnostics and the number of UIAs discovered continued to grow but with a bias towards people being investigated for vascular disorders, most notably other aneurysms. The exact sensitivity and specificity of angiography for aneurysms cannot be given because it is the standard against which other tests are judged, but both are thought to be high. Whatever method is used to detect an aneurysm, if interventional treatment is considered confirmatory angiography is likely to be needed to best define its anatomy.

Computed tomography

CT was developed by Hounsfield and colleagues at EMI Ltd in 1972.1 It was insensitive to aneurysms, especially with earlier scanners. As the focus of routine neuroradiological investigation shifted from angiography to CT scanning, fewer intracranial aneurysms were discovered incidentally to the investigation of non-vascular disease. The improved spatial resolution of more recent scanners and the technique of CT angiography have substantially improved sensitivity to aneurysms,4 and this trend is set to increase the number of truly incidentally diagnosed aneurysms in the future.

Magnetic resonance imaging

MRI was introduced to clinical practice in the mid 1970s. It did not achieve high penetration routine neuroradiological investigation until the 1980s in the USA and 1990s in the UK. Standard magnetic resonance sequences are more sensitive to aneurysms than conventional CT, though still not as sensitive as angiography.5,6 The specialised technique of magnetic resonance angiography is more sensitive than standard MRI but like conventional angiography is generally only done when vascular disease is being investigated.

Improvements in CT and MRI over the past 15 years have led to a large increase in the detection of specifically incidental aneurysms. The trends implied by this changing technology are amplified by the steady increase in the volume of neuroradiological investigations that has occurred.
Epidemiology
The collection of data on the prevalence of UIAs has reflected the development of neuroimaging. The three main sources of such data are post-mortem studies,\textsuperscript{9,10} angiography,\textsuperscript{10,11} and MRL.\textsuperscript{12,13} One angiographic study in particular illustrates the situation in the CT era. To estimate the prevalence of UIAs, Atkinson\textsuperscript{10} studied 9295 angiograms; after the elimination of all those where there was an association between the indication for the angiogram and aneurysms he was left with 278. His estimation of prevalence of around 1% remains one of the best available. A review found prevalences ranging from as high as 11% for some series to under 0·5% for others.\textsuperscript{14} The prevalence in Finland\textsuperscript{15} and Japan\textsuperscript{16} is consistently higher than in other parts of the world.

Prevalence reports based on magnetic resonance angiography have found unsuspected aneurysms in 2·8%\textsuperscript{13} and 2·9%\textsuperscript{17} of patients imaged but the patients in these studies were clinically selected for the procedure. The high sensitivity of magnetic resonance techniques to aneurysms together with its broad range of indications is likely to give better data on prevalence in the future.

The other epidemiological issue is the association of SAH with UIAs. The general incidence of SAH is around 10 per 100 000 people per year in developed countries. If the prevalence of UIAs is taken to be 1%, the risk of SAH for a person with a UIA can be calculated as 1% per year.\textsuperscript{18} This commonly cited calculation has one crucial weakness: it depends on the rupture rate of aneurysms not changing with time from their formation and this is contentious.\textsuperscript{19,20}

Direct data from follow-up studies published since the 1970s\textsuperscript{21–24} broadly support the 1% risk of rupture per year calculated above. These studies began when angiography was increasingly restricted to the diagnosis of vascular disorders and the largest group of patients entered were those with multiple aneurysms, one of which had bled. The significance of this was not appreciated until some years later but a clue came in 1987 when Wiebers and colleagues\textsuperscript{25} published data on small aneurysms in people with SAH. This distinction between the aneurysms in people with SAH and those in people with SAH. This distinction between the aneurysms in people with SAH and those in people with SAH. This distinction between the aneurysms in people with SAH and those in people with SAH.

The trend has been away from a conservative towards a more interventional approach. Early in the development of aneurysm surgery, techniques to reduce local blood pressure, such as Huntarian ligation of the carotid artery, were common but did not provide complete protection at preventing rehaemorrhage.\textsuperscript{26–28} Direct surgical approaches to intracranial aneurysms were first tried in the 1930s\textsuperscript{29} but the morbidity from early attempts at aneurysm ligation or clipping was high.\textsuperscript{30} Before 1974, it was unclear whether surgical treatment of ruptured aneurysm was of any benefit at all.\textsuperscript{31} Indeed trials in the 1960s that compared surgical treatment with conservative treatment had not shown a significant difference between the two.\textsuperscript{32–34} It was the results of the cooperative aneurysm study published between the mid 1960s and the early 1980s, that put surgical clipping of ruptured aneurysms on a firm scientific footing.\textsuperscript{23–25} This large four-arm international trial randomly assigned 972 cases of aneurysmal SAH to regulated bed rest (hospitalisation and bed rest for 3 weeks), regulated bed rest plus pharmacological hypotension, Huntarian ligation, or intracranial surgery. On an intention-to-treat basis, intracranial surgery was best with a 6·5 year total mortality of 37% compared with 55% for regulated bed rest, 46·3% for regulated bed rest with hypotension, and 39·6% with carotid ligation.\textsuperscript{23,24,36–38}

Clipping
While there was uncertainty about the surgical treatment of ruptured aneurysms, there was little enthusiasm for intervention in unruptured aneurysms in people who were well. The possibility began to receive serious attention in the 1970s when the benefits of treatment for ruptured aneurysms had been proven. At the time, little was known about the behaviour of UIAs. It was clear that aneurysms caused most SAHs and it was presumed that before aneurysms ruptured they existed as UIAs, although for how long was unknown. The mortality and serious morbidity from SAH may be as high as 70% and 10%, respectively.\textsuperscript{40} A 1% chance of a SAH every year adds up to a large risk of death or disability over the remaining lifespan of a young adult. Thus the argument for surgery seemed a strong one.

Surgeons began to report their results from clipping unruptured aneurysms in the late 1970s and they looked quite favourable.\textsuperscript{41–44} Various actuarial-style analyses were done to compare the lifetime risks of harbouring unruptured aneurysms with those of surgical intervention.\textsuperscript{45–48} The net gain from surgery is strongly dependent on the patient’s current age. These analyses all concluded that surgery brought substantial benefits well into middle age. By the early 1990s the situation seemed clear. Unruptured aneurysms were perceived as very dangerous and should be repaired. The only available means of doing this was intracranial surgery (figure).

Endovascular embolisation (coiling)
Finely coiled tungsten wire was found to be suited to aneurysm embolisation.\textsuperscript{49} Tungsten was attractive because of its high thrombogenicity,\textsuperscript{50} but it was soon discovered that it dissolved in vivo.\textsuperscript{51} Platinum coils were equally effective, did not dissolve, and were dense or opaque on radiography. A system of deployment and detachment by electrolysis of a fusible metal was developed by Guglielmi in 1989.\textsuperscript{52} The technique was adopted increasingly for ruptured aneurysms as the 1990s progressed. It appeared well tolerated and effective at preventing rehaemorrhage.\textsuperscript{53,54} Technical improvements\textsuperscript{55} have extended the range of treatable aneurysms so that now it is feasible to manage 80–90% in this way. The results of an international randomised trial comparing coiling and clipping of ruptured aneurysms (International Subarachnoid Aneurysm Trial [ISAT]) were published in 2002.\textsuperscript{56} Rates of death and serious morbidity at 1 year were 23·6% for coiling compared with 30·6% for surgical clipping. The management strategy for ruptured aneurysms has evolved into one of giving

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neuroradiologists first refusal. It is in this context that the treatment of UIAs has also shifted towards coiling, although the two situations are not directly comparable. Non-randomised data indicate that coiling is better tolerated than surgery in UIAs, 64–66 or vice versa. 67 New keyhole surgical techniques are reported to be better tolerated than conventional approaches.68 However the short-term results compare, the long-term protection from SAH conferred by coiling is what really matters, and this is unknown.

The ISUIA
This international study, launched in the mid 1990s, is a rather complicated observational study of the natural history of UIAs and the results of both clipping and endovascular embolisation.

Interim results with retrospective natural history data published in 19981 restarted the debate on how to deal with UIAs. Incidental aneurysms smaller than 1 cm were reported to have an extremely low rupture rate (<0·05% per year). Larger incidental aneurysms and all additional aneurysms had higher rupture rates of between 0·5% and 2·6% per year. This amounted to saying that there are two phenotypes of aneurysms, with low and high risk. The group of patients with additional aneurysm had a disproportionate number of high-risk phenotypes because they were selected on the basis of a previous SAH.

The outcomes of treatment (principally clipping) were worse than was suggested by previous large meta-analyses69 but not by much.70 Rates of mortality and major disability were 6·5% in those aged under 45 years, 14·4% for those between 45 years and 65 years, and 32·0% in those over 65 years. Actuarial calculations based on these results suggest that the risks of intervention outweigh the benefits in anybody with an incidental aneurysm under 1 cm and in anybody else over the age of 50 years.40

These results provoked protest. Some of the issues raised were not without merit but economic motivations could be discerned in the debate. The acceptance of the results by salaried surgeons was substantially greater than of those working in a fee-for-treatment environment, an example of a recognised phenomenon.71 Reports consistent with the ISUIA results72 and of much better surgical results appeared soon after its publication.73,74 Further results from the ISUIA were published in July 2003 (table).2 The size bands of aneurysms were changed to <7 mm, 7–12 mm, 13–24 mm, and >24 mm and the dependence of operative risk on age, aneurysm size, and aneurysm location was clarified. Incidental aneurysms of the anterior circulation (with the possible exception of posterior communicating artery aneurysms) under 7 mm are low risk and the risks of treating them outweigh the benefits (table). For other groups, the risk of haemorrhage increases with size and location in the posterior circulation as does the risk of treatment. Life year analysis taking account of group-specific treatment and haemorrhage risks suggests benefits from treatment up until around the age of 60 years.

Although the more recent results are broadly in agreement with those of the first ISUIA publication, questions remain. The earlier finding that incidental aneurysms under 10 mm in diameter are safe7 has not been borne out, as haemorrhages in the 7–9 mm range have been noted.7 Consequentially the safe size limit has been revised to 6 mm. It is paradoxical that, whereas the small incidental aneurysms seem substantially safer than small additional aneurysms, for larger aneurysms the reverse was true. Also significant is that incidental aneurysms of one of the commonest locations in the anterior circulation (the posterior communicating artery) were reported to have a higher risk of rupture. Doubt exists as to whether this effect is real.

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<th>Aneurysm size range (mm)</th>
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Aneurysm subtypes

**Familial aneurysms**

The evidence base behind recommendations for the treatment of incidental UIAs cannot be applied directly to the issue of familial aneurysms. Knowledge about familial UIAs is based on studies of families with one or more members affected by ruptured aneurysms. This point could prove to be important. There may be families with inherited UIAs in which the risks are very low, whereas current studies have selected only families with high-risk aneurysms.

Evidence specific to familial aneurysms suggests that they are distinct from other aneurysms. Within families, aneurysms tend to be at the same site and haemorrhages tend to occur within the same decade of life. The similarities are even greater among identical twins, who, if both are affected by aneurysmal SAH, tend to have the bleed within 5 years of each other. Familial aneurysms have been reported to affect particularly the middle cerebral complex, but this could be because the few families studied happened to display clustering at that site. Familial aneurysms tend to rupture when smaller and in younger people than non-familial aneurysms.

**Prevalence**

Several studies have reported that people with first degree relatives affected by SAH are more likely than the rest of the population to have UIAs. Reports of prevalence in such relatives varies from 4% to 14%. Higher prevalences have been reported in families from Finland and Japan where intracranial aneurysms are particularly common. They are more common in women, as is the case generally for aneurysms. It has also been shown that the risk increases with the number of first degree relatives affected: someone with two affected relatives is roughly twice as likely to have a UIA than someone with one affected relative. For most developed countries the risk of someone harbouring a UIA is estimated to be 4–5% if they have had one first degree relative affected by SAH and 8–10% if they have two or more affected relatives.

**SAH incidence rates**

Studies on the rates of SAH affecting the families of index patients have the problems of uncertainty about the causes of death of family members. Several population-based studies have been reported, all of which show a high risk in first-degree relatives of people who have SAH. The proportion of first degree relatives affected ranges from 1% to 11%. The evidence for an increased risk of aneurysm formation and SAH in people with second or third degree relatives affected is less persuasive. As any index case will tend to have a much larger number of second or third degree relatives than first degree relatives, coincidental associations due to chance alone are to be expected.

**Symptomatic aneurysms**

Attempts have been made to separate truly asymptomatic incidental aneurysms from those that have not ruptured but have caused symptoms of pain from mass effect or emboli. It is currently accepted that aneurysms presenting with an acute onset of symptoms should be treated. There is thus little opportunity to collect observational data on untreated ones. When aneurysms are discovered in the investigation of neurological symptoms there is commonly uncertainty about whether the aneurysm is causing the symptoms. In follow up series the break down of bleeds into symptomatic and asymptomatic aneurysm may not be reported. Different symptoms may denote different risks. Aneurysms associated with transient ischaemic episodes are reported to present a low risk of rupture whereas symptomatic aneurysms generally appear to have a higher risk. These factors combine to mean that the difference in behaviour between asymptomatic and symptomatic unruptured aneurysm is poorly defined but symptomatic aneurysms seem to be more dangerous with a reported relative risk of 8.3.

There is some credibility to the early high-risk hypothesis, which, together with such data as exists from direct observation, suggests that aneurysms presenting with an acute onset of symptoms should be treated. These include posterior communicating artery aneurysms presenting with acute painful third nerve palsy. It is interesting that palsy of the third nerve commonly recovers after coiling. For those aneurysms presenting with longer-standing symptoms, particularly embolic phenomena, the risk of rupture may be low but there is a possibility of successfully controlling the symptoms which adds another argument in favour of treatment.

**Giant aneurysms**

No firm definition exists for when a large aneurysm becomes a giant one, but a diameter of 25 mm or over, as adopted in the ISUIA, is a workable definition. Data on the behaviour and treatment risks associated with such aneurysms are sparse because of their relative rarity. Such data as exists cannot be confidently applied to individual cases because of the large variability in the clinical and anatomical features of the condition. Management decisions thus remain largely a matter of clinical judgement. In general, giant aneurysms pose greater risks and are more dangerous to treat than smaller ones, effects which cancel to some extent when considering the benefits of treatment. Our analysis of the available data suggests that offering reparative treatment to people with a life expectancy of 20 or more years is a reasonable starting point. Medical hypertensive treatment may be a particularly attractive option in this group.

**Risk factors**

Risk factors for SAH are fairly well-defined and include female sex, smoking, heavy drinking, hypertension, family history, ischaemic heart disease, uncustomed physical exertion, high cholesterol, autosomal polycystic kidney disease (ADPKD) and use of oral contraceptives. By inference, these are also implicated in the risks of UIAs, but direct evidence is scarce. There are two issues involved: the risk of developing an unruptured aneurysm and the risk that it will rupture once formed. Data on risk factors for aneurysms come from patients under surveillance with regular angiography for other aneurysms.
Female sex and smoking have been identified as risk factors. The reported risk factors for rupture of formed aneurysms include, age (inversely), smoking, and hypertension.

**Associated disorders**

Several disorders are known or suspected of being associated with intracranial aneurysms. These include type III collagen deficiency (Type IV Ehlers-Danlos), pituitary tumours, and aortic coarctation. Graves’ disease may also be associated, and Marfan’s syndrome has been implicated, but this link has been persuasively contested as has the link to neurofibromatosis I. The best known and best characterised association is with ADPKD. Association with intracranial aneurysm occurs in both type 1 and the less severe type 2 ADPKD. The prevalence of aneurysm in these patients is higher than in the general population, although by how much varies between reports. The prevalence of aneurysms in patients with ADPKD was 41% in one study, although 10–20% is more typical. ADPKD and a family history of aneurysms seem to be independently associated with intracranial aneurysms, their prevalence among people with both being over 20%. Some researchers report a high rate of new aneurysm formation in ADPKD, although others have not found this. There are no specific data on the risk posed by UIAs in ADPKD.

**Screening**

The screening of people at risk of SAH because of a strong family history (two or more affected first degree relatives), or an associated disorder, has been recommended in the past. As enthusiasm for repairing incidental UIAs dimmed after the 1998 publication of ISUIA results, so it did for screening. Although analyses published since 1998 have come down against screening, uncertainties remain.

Familial aneurysms seem to behave more like additional than incidental aneurysms. Screening analyses assume aneurysms are incidental because the people screened have no history of SAH. Affected relatives generally do have such a history and the use of incidental UIA data may give the natural history an inappropriately benign bias. Another argument for screening is that policy can be tailored according to a particular radiologist or surgeon’s results. If a radiologist or surgeon has very good results screening may be advised, whereas if they are poorer it may not. This would mean that aneurysms would only present to operatives with good results, making averaged operative results from sources like ISUIA inappropriately poor.

Screening has been said to have less average benefit per patient than does treatment of known lesions, because it carries the additional costs of clinical and psychological risks. These costs include the investigation of many healthy people with tests that may have their own dangers. Also, screening is not 100% specific and there are risks of subsequent treatment for mistakenly identified lesions. With this background screening can only be justified if the benefits of treatment are clear and substantial. Another argument against screening concerns the lifespan of aneurysms before rupture. This lifespan is unknown, and formation and rupture of new aneurysms have been reported after screening. Some estimates suggest that aneurysms bleed within a few weeks of formation, which, if true, would mean that screening would catch few aneurysms before they bleed.

With the current state of evidence, whether or not to screen is an open question. In cases where there is a strong family history with clear age clustering and where local operative results are good, screening of family members at high risk within a decade of the age at which the family SAHs cluster may be justified. However, in low-risk families, in a family member well away from the dangerous age, or where local operative results are unknown or poor, screening may be best avoided. In cases there is concern about a substantial risk of SAH, but screening is not justified, deliberate pharmacological hypotension as used by Slosberg could be considered together with avoidance of smoking.

**Unresolved issues**

That coils, where possible, is better tolerated than clipping for unruptured aneurysms seems reasonable from the current standpoint, but there remain some gaps in the evidence. The reduced morbidity and mortality offered by clipping in this situation is supported by observational data but remains partially an extrapolation, albeit a reasonable one, from ruptured aneurysms. A more nagging uncertainty is the long-term performance of coiling. Untreated ruptured aneurysms have a very high re-bleed rate within a year of the first haemorrhage, after which it falls. The exact magnitude of these risks is not well defined because the principal studies on the natural history of aneurysmal SAH date from the 1960s and 1970s when confirmation of causes of death was more difficult than today. The death rate after conservative management of ruptured aneurysms was 2–3%. How many of these deaths are caused by SAH is unknown. The re-bleed rate does fall substantially over the first few months. There is little doubt that coiling effectively prevents early re-bleeds, but its good performance compared with clipping could be explained by this alone and tells us nothing about long-term efficacy. The long-term efficacy of treatment is particularly important for UIAs, because the short-term risks are much less than after SAH, and long-term protection is the point of treatment. In fairness, clipping is not a guaranteed fix either but it gives better protection in the short term and intuitively it seems more secure.

**Medical treatment**

Little consideration has been given to the possibility of medical treatment. Two manageable risk factors are clearly associated with aneurysmal disease: hypertension and smoking. Slosberg has collected a series of patients referred after SAHs because they had aneurysms that were not surgically repairable. His strategy for reducing the risk of future SAH consisted of deliberate and aggressive pharmacological lowering of systemic blood pressure to the limit that patients could tolerate without developing...
symptoms. Most patients came to Slosberg a few weeks after the SAH, and the early very high-risk phase had passed. Nevertheless the risk of aneurysms in this group of patients was high. Surgically unmanageable aneurysms tend to be large and involve multiple cerebral vessels. It has long been suspected, and now proved,1,2 that such aneurysms carry a high risk of rupture; however, over many years of careful follow-up, Slosberg reported a low rate of SAH.1,2,10,11,12 This finding raises the obvious question: would simple antihypertensive medication be a better treatment for unruptured aneurysms than either surgical or endovascular intervention? To date, this question is unanswered. Cigarette smoking is known to be strongly associated with the rupture of aneurysms.2,3 Hypotensive medication and counselling on smoking could be highly efficacious at reducing risk.

**Posterior communicating artery aneurysms**

There is an issue with the anatomical classification of aneurysms in the recent ISUIA publication. Aneurysms of the posterior (vertebrobasilar) circulation are comparatively rare, comprising around 10% of the total.14 That they carry a higher risk of haemorrhage than those of the anterior circulation has been reported several times.15 No consistent differences in risk between the three main anterior sites (anterior cerebral, internal carotid, and middle cerebral arteries) have previously been observed, but the authors of the ISUIA classified posterior communicating artery aneurysms (the commonest of the internal carotid aneurysms) in the verteobasilar rather than anterior group.1 This was because all of the bleeds that occurred in the small anterior incidental aneurysms occurred in posterior communicating aneurysms. The number of such bleeds was not given, but can be estimated by interpolation to be two. With so few observed bleeds their coincidence in the posterior communicating group could easily be due to chance alone. If small incidental posterior communicating aneurysms are grouped with the anterior circulation the bleed rate is around 0·1% per year, too low to merit interventional treatment. If, however, they are grouped with the verteobasilar circulation their rate becomes about 0·5% per year, which justifies intervention. This would be a minor matter if small incidental posterior communicating aneurysms were not one of the commonest types. The hypothesis that posterior communicating aneurysms are more dangerous than others in the anterior circulation needs to be corroborated.

**Risk changes over time**

The most significant unresolved issue is how the risk posed by an untreated unruptured aneurysm varies with time. The competing theories are the “constant risk” and the “early high risk”. The constant risk theory holds that aneurysms, once formed, pose a constant risk of rupture that does not substantially change as the years go by. The alternative early high risk theory holds that aneurysms pose a high risk immediately after they form, but that this risk becomes very low after a few weeks or months either because of some healing process within the aneurysm wall or because all the weakest ones are selected out by bleeding early, leaving the strong ones to survive as unruptured aneurysms.

There is evidence to support both theories. Heiskanen and Juvela in Finland have been following up a cohort of patients with unruptured aneurysms for more than 40 years and have found a nearly constant rupture rate.2,12,13 However, recently released data from the ISUIA study may show that rupture rates in the first 5 years after diagnosis are substantially higher than those after this period; longer term follow up of the ISUIA study population will be required to confirm that this observation is not an artefact of the follow-up methods used.7 A more complex argument for the early high-risk-theory comes from epidemiological data. Most people presenting with SAHs have small aneurysms and no previous history and would have fallen into the safe incidental class before the bleed. In this class there is a massive discrepancy between the observed rupture rate and that calculated from incidence and prevalence data. This discrepancy could be accounted for if the early high-risk theory were true. Data on the incidence of SAH, the prevalence of aneurysms, and the behaviour of unruptured aneurysms can be used to calculate that the high-risk period is probably less than about 40 weeks.19

If the early high-risk theory is true, the observed risks will settle over a short time to a much lower total level. Moreover, the rupture risks themselves may be incorrect if many of the recorded SAHs in the various studies were actually from new aneurysms and not the ones that were supposed to be under observation. Another implication of the early high-risk theory concerns people at high risk of SAH such as those with a strong family history. They could not be fully protected by screening angiography because it would be impractical to screen frequently enough.

**Management recommendations**

In very-low-risk aneurysms the risks of treatment outweigh the benefits at all ages. These are incidental anterior circulation aneurysms under 7 mm in patients with no history of SAH. We include posterior communicating aneurysms in this group pending further data on their risks. All other types carry significant risks and merit consideration of treatment. We have done risk benefit analyses based on 1998 and 2003 ISUIA data (unpublished). The benefit in life years saved falls and passes from positive to negative as the person gets older. The amount of benefit in younger people is highly sensitive to changes in aneurysm risks and operative morbidity but the age beyond which gain turns to loss is not. This age is around 60 years. Though easy to apply, this can be misleading as it assumes that everyone of age 60 years has the same life expectancy, which is clearly not true. More accurate but less easy to apply is to say that gain turns to loss when remaining life expectancy falls below 20 years. Endovascular coiling is better tolerated than surgical clipping and would be the treatment of first choice if long-term efficacy were proved; until it is, both surgical clipping and coiling should be regarded as reasonable options. The issue of screening for occult aneurysms in people at risk is unresolved.
Conclusions
Incidentally discovered aneurysms of the anterior cerebral circulation under 7 mm in size in people who do not have a personal or family history of SAH should be left untreated. The benefits of clipping or coiling are strongly dependent on life expectancy, and the risks probably outweigh the benefits for people with a remaining life expectancy of less than 20 years or so (eg, someone 60 years old in a developed country). In all other cases the risks associated with aneurysms justify the risks of treatment. The best-tolerated treatment at the moment seems to be endovascular embolisation, but data on long-term efficacy are awaited and until this is confirmed surgical clipping is an equally reasonable option. If neither is feasible then medical hypotensive treatment is a viable alternative.

Authors’ contributions
PM did the literature review and wrote the epidemiological parts of the review, ADM wrote parts of the review from a surgical perspective; AG until this is confirmed surgical clipping is an equally

References
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