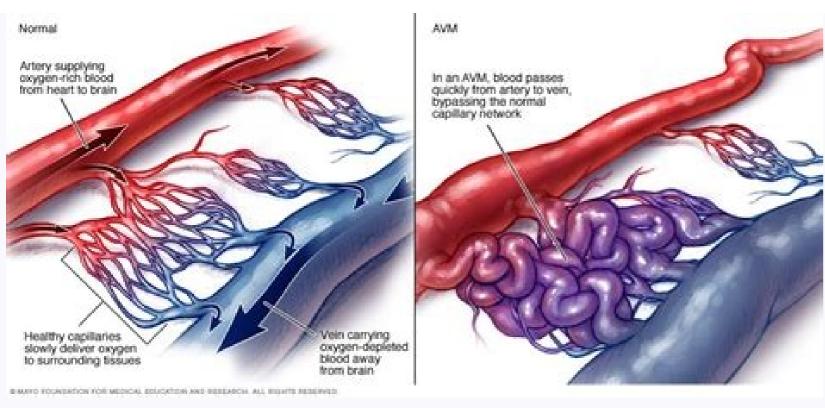
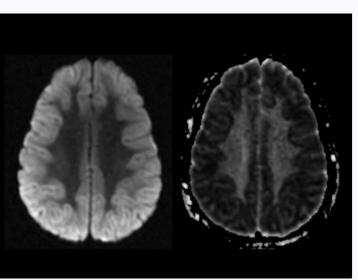
Arteriovenous malformation aneurysm causes

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Is arteriovenous malformation dangerous. Avm aneurysm symptoms. Is avm a brain aneurysm. What causes an avm rupture. Can an avm cause an aneurysm.

AbstractBackground and Purpose—Brain aneurysms and vascular malformations can cause cerebral hemorrhages, with devastating consequences for the patients and their families. Since the development of microcatheters and materials used for endovascular embolization, we have witnessed a rapid advancement in the technology and in the number or patients treated with this approach. The aim of this review is to survey recent data relevant to new technologies and emerging treatment strategies in these areas. Summary of Review— Clinical trials assessing the safety and efficacy of coil embolization for cerebral aneurysms were based on the use of bare platinum, helical coils. Since then, endovascular operators have been testing and using new materials such as bioactive coils, expandable coils, and complex-shaped coils. Based on the data so far obtained, third and fourth generation coil designs are rapidly emerging and will be ready for clinical application in the near future. Balloon- and stent-assisted coil embolization is enabling the treatment of complex, large-neck aneurysms and the vascular reconstruction of lesions previously considered not treatable. New open- and closed-cell designs allow the navigation and deployment of stents in extremely tortuous vessels. With regards to the embolization of vascular malformations, it is possible to safely navigate microcatheters and microwires through very small arteries previously considered not accessible. In addition, embolization materials such as n-butyl cyanoacrylate and ethylene-vinyl alcohol copolymer are now routinely injected to safely reduce or obliterate large and complex arteriovenous malformations and fistulae. Conclusions—Advancements in technology are rapidly improving the endovascular approach to the treatment of cerebral aneurysms and arteriovenous malformations. According to angiographic and eurysms remain undetected; however, increasing numbers of aneurysms are detected incidentally during neuroimaging studies. A minority of aneurysms are detected when they cause symptoms either by cranial nerve compression or when they rupture, causing a subarachnoid hemorrhage (SAH). Aneurysmal SAH (aSAH) can have devastating consequences, with a reported case fatality of 32% to 67% and a 10% to 20% long-term dependence in survivors.1-4The first description of the endovascular approach to embolize cerebral aneurysms was made by Fedor Serbinenko in 1971.5 The Russian neurosurgeon described the use of detachable balloons to occlude either the aneurysmal sac or the parent vessel. In 1991 Guido Guglielmi introduced the use of detachable coils for the treatment of intracranial aneurysms.6-7 As described by the authors, platinum coils were inserted into the aneurysmal sac and separated from a stainless steel introducer by electrochemical detachment. Initially, the treatment was reserved for aneurysms judged to be at too high a risk for microsurgical clipping. However, as technology and the operator's ability improved, the use of detachable coils rapidly spread. Ten years after its first description, it was estimated that 1500 patients per month were treated with endovascular embolization worldwide.8To compare the safety and efficacy of endovascular embolization versus microsurgical clipping, the International Subarachnoid Aneurysm Trial (ISAT) investigators undertook the first multicenter, randomized trial, 9 which involved 42 centers in Europe and North America. The trial enrolled 2143 patients and randomly assigned them to neurosurgical clipping (n=1070) or endovascular embolization with bare platinum coils (n=1073). The clinical outcomes were assessed at 2 months and at 1 year, with interim ascertainment of rebleeding and death. The primary outcome was a modified Rankin Scale score of 3 to 6 (dependency or death) at 1 year. Trial recruitment was stopped early by the steering committee after a planned interim analysis revealed worst outcomes in the neurosurgical group. In particular, 190 of 801 (23.7%) patients in the endovascular arm were dependent or dead at 1 year compared with 243 of 793 (30.6%) treated with microsurgical clipping (P=0.0019).9 The relative and absolute risk reductions in dependency or death after allocation to an endovascular versus surgical treatment were 22.6% (95% CI, 8.9 to 34.2) and 6.9% (2.5 to 11.3), respectively.9The data from ISAT were the subject of debate in the literature.10-11 Those in disagreement with the trial's results mainly argued that (1) the outcome of the aneurysms randomized to both treatments was biased for not being representative of the general population of patients with aSAH, (3) the long-term outcome of the endovascular treatment was unknown, and (4) the majority of patients screened were not subject to randomization. Although enrollment in ISAT was halted, the investigators continued their long-term follow-up analysis on the patients enrolled. The data published in 2005 showed that the early survival advantage of the endovascular arm. The risk of epilepsy was substantially lower in patients treated with coil embolization. However, the risk of rebleeding at follow-up (7 years) was slightly higher in the endovascular arm, 0.2% per patient-year as compared with surgical clipping, 0.1% per patient-year (log rank, P=0.22).12The Cerebral Aneurysmal Rupture After Treatment (CARAT) investigators compared rerupture rates after aSAH.13 In an ambidirectional cohort study, 9 institutions identified all ruptured saccular aneurysms treated between 1996 and 1998. A total of 1010 patients (711 surgically clipped, 299 treated with coil embolization) were contacted by written questionnaire or telephone. A neurologist, neurosurgeon, and neurointerventional radiologist independently adjudicated possible reruptures. Rerupture of the treated aneurysm after 1 year occurred in 1 patient treated with coil embolization during 904 person-years of follow-up (annual rate, 0.11%) and in no patients treated with surgical clipping during 2666 person-years (P=0.11). Aneurysm retreatment after 1 year was more frequent in patients treated with coil embolization; however, major complications were rare during retreatment. The authors concluded that late events are unlikely to overwhelm differences between the procedures at 1-year follow-up.13Van Der Shaf et al recently reviewed randomized trials comparing coiling versus microsurgical clipping in patients with aSAH using the Cochrane Stroke Group Trials Register, MEDLINE, and EMBASE.14 The analysis included 2272 patients (range per trial: 20 to 2143). At 1-year follow-up evaluation, the relative risk of poor outcome for coiling versus clipping was 0.76 (95% CI, 0.67 to 0.88). The absolute risk reduction was 7% (95% CI, 4% to 11%). For patients with SAH secondary to ruptured, anterior circulation aneurysms, the relative risk of poor outcome was 0.78 (95% CI, 0.68 to 0.90) and the absolute risk was 27% (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.68 to 0.90) and the absolute risk was 0.41 (95% CI, 0.88 to 0.90) and the absolute risk was 0.41 (95% CI, 0.88 to 0.90) and the absolute risk was 0.41 (95% CI, 0.88 to 0.90) and the absolute risk was 0.41 (95% CI, 0.88 to 0.90) and the absolute risk was 0.41 (95% CI, 0.88 to 0.90) and the absolute risk was unruptured intracranial aneurysms (UIA) is still a matter of debate 15-17 In 2000 the Stroke Council of the American Heart Association published guidelines for the treatment of UIA.16 Currently, Raymond et al are investigating these issues in a randomized trial.18 The discussion of the variables that play a role in the decision on whether to treat UIA is beyond the scope of this review. With regards to the safety and efficacy of the endovascular treatment for UIA, several authors indicate an overall 5% to 10% risk of morbidity, mostly secondary to embolic phenomena, and near 0% mortality.19-23 In the largest data set so far, Johnston et al evaluated the safety of the endovascular treatment compared with microsurgical clipping in 2069 patients treated in California hospitals for UIA from 1990 to 1998.24 In-hospital mortality and discharge to another healthcare facility rather than home were less frequent in the endovascular group (10% versus 25%; P

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