group, but consistent with other studies. In 2004, Henkes and colleagues[12] published a large series of 1811 aneurysms treated by endovascular coil occlusion with nearly identical corresponding occlusion rates.

In keeping with the angiography results, technical outcomes in ISAT clearly show that if endovascular coiling is attempted it is more likely to fail than an attempt at surgical treatment of ruptured aneurysms. This result is revealed by the 6·1% (66/1080) of patients in the endovascular group and 1·4% (14/1004) of patients in the surgery group in whom the first procedure attempted was not completed ($p<0·0001$).

Overall, rehaemorrhage rates were not significantly different between the endovascular and surgical groups. However, a larger proportion of patients randomised to surgery than to the endovascular intervention bled before the procedure. Because risk of rebleeding is greatest within the initial period after subarachnoid haemorrhage, this discrepancy could be accounted for by the significant difference in time between randomisation and the first procedure for the two treatment groups (1·1 days for endovascular and 1·7 days for surgery).[2] If patients who rebled before any intervention are then excluded, the resulting significant difference favours a reduced rate of rebleeding in surgically treated patients (p=0·004).

Despite these cautions, ISAT is a well-executed and statistically powerful study. Its randomisation and large patient population give us confidence to use some of the information to make evidence-based treatment decisions for certain ruptured intracranial aneurysms. The ongoing collection of data from ISAT will continue to yield information about the durability and long-term efficacy of endovascular coiling compared with surgical clipping. As techniques continue to improve, however, more aneurysm locations and conformations will become accessible and amenable to endovascular interventions, which will continue to raise the question of whether surgery or coiling is the best treatment for our patients.

Alim P Mitha, Christopher S Ogilvy
Massachusettes General Hospital, Boston, USA
cogilvy@partners.org

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The hippocampus and remote autobiographical memory

In Newsdesk (August, 2005),[1] new evidence for the neuroanatomy of remote memory was reported. On the basis of the findings of the US team lead by Larry Squire,[2] remote autobiographical memory was suggested to be independent of the medial temporal lobe but dependent on the neocortex. By contrast with previous hypotheses, this new proposal predicts that after damage to the medial temporal lobe only recent autobiographical memories should be impaired in neurological patients, whereas loss of both recent and old autobiographical memories implies additional damage in the neocortex. However, there is evidence not included in the Newsdesk article, that is problematic for this new prediction.

Two patients, NT and VC, were previously reported to have lesions restricted to the medial temporal lobe and exhibited loss of remote memories extending for decades. Patient NT presented with extensive and

ungraded retrograde amnesia after a right temporal lobectomy.[3] This patient had substantial difficulty recalling autobiographical memories dating to childhood. The neuropathological investigations revealed clear-cut sclerosis of the unresected left hippocampus, but all other cortical areas, including the previously removed right temporal lobe, were normal. Thus, it is tempting to conclude that her severe remote memory loss was a consequence of her bilateral hippocampus damage.

Detailed cognitive testing of the severely amnesic patient VC reinforces this conclusion.[4,5] On all retrograde memory tests, including the standard autobiographical memory interview, his results were equally poor over all periods tested: he had no autobiographical recollection from any period of his life. Qualitative MRI, MRI volumetry, voxel based morphometry, spectroscopy, and functional MRI showed that the primary abnormality was located in the hippocampus bilaterally. Only MRI volumetry identified a slight decrease of the left parahippocampal volume, but functional neuroimaging showed that this region was active in VC during memory retrieval. Therefore, investigations of VC suggest that the hippocampus is crucial for remembering one's personal past. This finding is consistent with those from other lesion and neuroimaging studies.[6]

The discrepancy in findings between patients such as NT and VC and those reported by Squire's team may depend on important differences in the patients' severity of amnesia. For example, Bayley and colleagues[2] draw attention to the test results of patients (EP and GP) with selective damage to the temporal medial lobe who obtained maximum scores on the childhood portion of the autobiographical memory interview (9/9), by contrast with the very impaired score of VC (1/9). However, EP and GP's performance on other standard memory tests was only mildly impaired, whereas on similar tests VC barely could score any points.

We suggest, therefore, that questions regarding the neuroanatomy of remote memory, and particularly the role of the medial lobe and hippocampus, are far from resolved. Further studies of amnesic patients with well-documented and restricted lesions are needed to ascertain the critical anatomical structures affected in remote memory.

Lisa Cipolotti, Morris Moscovitch
Department of Neuropsychology, National Hospital for Neurology and Neurosurgery, Queen Square, London, UK, and Dipartimento di Psicologia, University of Palermo, Palermo, Italy (LC); and Department of Psychology, University of Toronto, Toronto, ON, Canada (MM)
l.cipolotti@ion.ucl.ac.uk

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Birth order, infection in early life, and multiple sclerosis

Sadovnick and colleagues[1] did not show an association between birth order and multiple sclerosis and thus concluded that a possible protective effect of infant sibling exposure to putative environmental factors in the first 6 years of life[2] is doubtful, assuming birth order is the main partial surrogate for infant sibling exposure. Unfortunately, birth order is subject to error as a proxy measure for exposure to younger infant siblings (table), and thus the lack of association for birth order could implicate imprecision in measurement of infant sibling exposure. The Spearman correlations between birth order and various sibling measures in the Tasmanian multiple sclerosis case-control study[2] suggest that birth order could be used as a good proxy measure of older

	Patients (n=136)	Controls (n=272)
Total sibling exposure by age 6 years (cumulative days)	0·84*	0·85*
Older sibling exposure by age 6 years (cumulative days)	0·92*	0·98*
Younger sibling exposure by age 6 years (cumulative days)	−0·36*	−0·13†
Infant (restricted to siblings age <2 years) younger sibling exposure by age 6 years (cumulative days)	−0·34*	−0·15†

*p<0·001; †p<0·05.

Table: **Spearman correlations between birth order and various sibling measures[2]**