from even low doses of anticholinergic agents.

Two points are illustrated by this unusual case. First, anticholinergic agents, which do not have demonstrable efficacy in NMS are probably contraindicated in NMS. Second, patients with NMS should be carefully monitored for signs of anticholinergic toxicity.

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Intraoperative aneurysms rupture during the prediscision stage

Much has been written about intracranial aneurysms which rupture as they are being dissected, probably because of the frequency of this event (24%, in one well known series).1 Less has been written about rupture of aneurysms during the early stage after the craniotomy is started and before the aneurysm is exposed, the prediscision stage, discussed by Batjer and Samson.2 Fortunately, this is uncommon (Yasargil 1964; Shidas G Drake, personal communications). Batjer and Samson encountered it four times in a series of 370 consecutive aneurysm operations and questioned whether the operations should have been aborted. I report an experience of rupture of an aneurysm before dissection.

A 59 year old right handed white woman experienced the acute onset of severe headache 48 hours before admission. She had no neurological symptoms and the only abnormal finding was neck stiffness. Computerised tomography (CT) of the head was normal but lumbar puncture disclosed grossly bloody fluid under an opening pressure of 210 cm H2O. Left carotid angiogram showed an anterior communicating artery aneurysm 3 mm in diameter pointing forward and down.

During opening the patient was given 50 g of mannitol, but spinal fluid was not drained. A slow drip of nitroprusside was started. As the chiasmatic cistern was opened, and before the aneurysm was displayed, the aneurysm ruptured and the brain herniated massively into the craniotomy opening. A hand held brain retractor was left in place to allow escape of blood. At the time of rupture the patient’s blood pressure was 100/80 mm Hg. Within a few minutes he was able to follow simple commands. His temperature decreased from 102°F to 99.5°F over the next hour.

The differential diagnosis of NMS includes CAS. In a recent review, Guze and Baxter state that a response to physostigmine is sufficient to differentiate between NMS and CAS.3 However, this case suggests that the two disorders can coexist in the same patient. Furthermore, since drugs that cause NMS have anticholinergic properties, cholinergic blockade by neuroleptic drugs may make patients more susceptible to CAS
Combined neuroleptic malignant syndrome and the central anticholinergic syndrome.

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*J Neurol Neurosurg Psychiatry* 1990 53: 711
doi: 10.1136/jnnp.53.8.711

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