The second case was a nine year old boy presenting with intellectual deterioration and an epileptic seizure. A month later, when admitted to Hôpital Ste-Justine, Montreal, he had moderate mental retardation, his affect was inappropriate and there was mild oorfacial ore. The left plantar was extensor and his gait ataxic. He developed generalised myoclonus with occasional opisthotonus, and showed decorticate posturing. Serum measles virus titre was 1024. CSF protein was 0·54 g/l, with no cells, and the mea
cles complement fixation titre was 128. An EEG was dominated by low voltage slow and periodic high voltage slow-wave complexes.

His condition stabilised, but he remained bed-bound and died at the age of 19 years. The brain showed generalised atrophy, with profound neuronal loss and gliosis in the cerebral cortex and hippocampus with a few microglial nodules and tangles. Some remaining neurons showed intranuclear inclusions. All central grey nuclei showed severe neuronal loss with glial and microglial re-
actions and several tangles. There was severe neuronal loss in the substantia nigra and locus 
coeeruleus with tangles and Lewy bodies (fig.). Lewy bodies were also found in the dorsal vagal nucleus, cerebral cortex, spinal cord or autonomic nervous system.

These patients with SSPE showed long survival of 13 and 10 years respectively. Tangles occur even in patients dying at a young age,1 but Lewy bodies have not been described in other cases. They were present in the substantia nigra and locus coeruleus, but not in other areas usually susceptible to Lewy bodies. We know of a third case of SSPE with onset at 21 years, and a nine year survival. In this case there were very few nerve cells in the substantia nigra, and some pale bodies, which are normally seen in Lewy body 
diseases.4

Apart from the Lewy body–Parkinson's disease spectrum Lewy bodies are confined to a small group of rare degenerative disorders.1 They may be by-products of attempted regeneration, rather than of degeneration, thus explaining their occurrence in long-
standing cases of SSPE showing periods of relative stability. SSPE is a destructive in-
flammatory process due to persistent measles infection, but the aberrant immunological mechanisms are not fully understood. Although there is no direct evidence for an infectious aetiology for Parkinson's disease, there are parallels with SSPE which results from an infectious agent acquired in early life, leading to a progressive disorder associated with disconnection in identical twins,5 and Lewy bodies pathologically.

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Combined neuroleptic malignant syn-
drome and the central anticholinergic syndrome

The neuroleptic malignant syndrome (NMS) is an idiosyncratic reaction to neuroleptic drugs characterised by encephalopathy, rigidity, dysautonomia and hyperthermia.1 Dopamine receptor blockade appears central to the pathogenesis of rigidity,1 and possibly the encephalopathy.2 Because cholinergic receptor blockade can also cause encephalopathy, the differential diagnosis of NMS includes the central anticholinergic syndrome (CAS).2,3 Infections, anticholinergic, which are used to treat Parkinsonism associated with neuroleptic use, are often used to treat the rigidity in NMS.1 However, anticholinergic could theoretically exaggerate the encephalopathy of NMS, possibly spawing the development of combined NMS and CAS. We report the first case of combined NMS and CAS.

After four months of lithium therapy, a 28 year old man with a recent affective disorder was given 15 mg of daily haloperidol. One week later he was brought to the emergency room confused, agitated and hallucinated. Neurological examination disclosed a fever of 101°F, cogwheel rigidity, resting tremor, and dysmetria. Serum glutamic oxaloacetic transaminase 128 IU/l (normal 5-35), serum glutamic pyruvate transaminase 65 IU/l (normal 5-30), lactic dehydrogenase 373 IU/l (normal 90-220) were all increased in concentration. His creatinine phosphokinase was 7700 IU/l (normal 25-145). All other laboratory studies, including computed tomography (CT) of the brain and lumbar puncture were normal. The haloperidol and lithium were stopped.

Treatment included levodopa/carbidopa 25/250, and 4 mg of intramuscular benz-

odrine over three hours after receiving the benzodrine, his mental state deteriorated from being agitated and confused to comatose, and his temperature increased from 101°F to 104°F. The rigidity was unchanged, but he developed urinary retention, decreased bowel sounds and large pupils, suggesting anticholinergic toxicity. Six mg of intravenous physostigmine was administered. Within 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.1 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 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 tox-

icity.

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Intraoperative aneurysms rupture dur-
ing 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 stage when the crani-

otomy is started and before the aneurysm is exposed, the prediscision stage, discussed by Batjer and Samson.2 Fortunately, this is uncommon. (Yasargil 1984; Chaidos G Drake.

Left carotid angiogram showed an anterior communicating artery aneurysm 3 mm in diameter pointing forward and down.

During opening the patient was given 50 mg of nitroglycerin, but spinal fluid was not drained. A slow drip of nitropresside 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 the systolic blood pressure fell to 75/55 mm Hg. Immediately after rupture the patient was given 40 mg of nitropresside and 20 mg of dexamethasone intravenously. Five minutes after rupture nitropresside was stop-
ped and a nitroglycerin drip (40 mg in 50 cc of 5% dextrose and water) was started. Within two minutes, that is seven minutes after rupture, the brain suddenly returned to its pre-rupture position and the BP fell to 60/40 mm Hg. Frontal lobe amputation, which had removed only 5 cc of brain, was terminated and the BP was allowed to rise slowly over the next 45 minutes. No further attempt to
Combined neuroleptic malignant syndrome and the central anticholinergic syndrome.

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