Acute central nervous system oxygen toxicity at normobaric pressure

That acute central nervous system oxygen toxicity may occur at normobaric pressure[1] was suggested to me by a clinical experience nearly half a century ago. A healthy, burly male in his 30s was riding home on his motorcycle late one evening when he drove into a wire that had been stretched across the road, hitting him in the neck. Despite major haemorrhage and ruptures of his larynx and oesophagus, an emergency room physician successfully intubated him.

The following morning, in the operating room (OR) he was transferred from breathing 100% oxygen (O_2) spontaneously via his endotracheal tube and an Ambu bag to 100% O_2 from an old Boyle's anaesthetic machine via a circle circuit. Over a few minutes, he became increasingly tachypnoeic and restless and then deeply cyanosed before it was realised that nitrous oxide (N_2O), not O_2, had been turned on unintentionally. He was immediately turned onto high-flow 100% O_2 and his colour pinked up within a few breaths. Shortly thereafter (perhaps 10 seconds), he had a grand mal convulsion, which was controlled by an intravenous bolus of sodium thiopentone. No subsequent complications arose from this episode of which he had no recollection and he had no further convulsions during his long hospital stay.

In the early 1970s, continuous O_2 and carbon dioxide (CO_2) monitoring and modern anaesthesia machines were things of the future. On review, the anaesthetist, working in that OR for the first time, discovered that the N_2O rotameter was mounted on the left of the rotameter block on that Boyle's machine, whereas on all other machines in his previous clinical experience, including in all the other ORs in that hospital, the O_2 rotameter had been situated on the left of the block. He had turned the rotameter knob by touch behind him whilst attending to the patient.

Whilst the causation of his convulsion remains conjecture, the two most likely mechanisms are acute hypoxic hypoxia and acute oxygen toxicity. Hypoxic hypoxia occurs in a variety of environmental (breath-hold diving, altitude) and traumatic (drowning,[2] choking, strangulation) situations and in neonatal hypoxia. Seizures are well documented following hypoxic hypoxia.[3] Multifocal myoclonic jerks have been reported after loss of consciousness from breath-hold diving.[4]

Given the chronological sequence described above, the other possible explanation is that this was an oxygen-induced convulsion since it did not occur until the patient was fully re-oxygenated. Hypoxia is known to impair cerebral autoregulation,[5] though the interactions of O_2 and CO_2 are complex. The writer suggests that the cerebral circulation would have been vasodilated from the severe acute hypoxia when it was perfused by blood with a high partial pressure of O_2 which was then imparted to the cerebral tissues before autoregulation could be restored. Since CO_2 monitoring was not available on anaesthetic machines in the early 1970s, it is unknown whether or not hypercapnia contributed to this episode, as appears to have been so with the diver in the case report.[1]

References

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F Michael Davis
Address for correspondence: *Department of Anaesthesiology, University of Auckland, Auckland, New Zealand*
simo01@xtra.co.nz

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