Blood products into the affected tissue.\[5\] In the brain the most susceptible regions to ischemic injury are the hippocampus, Purkinje cell layer in the cerebellum, basal ganglia and thalamus. These areas have been previously described as developing HT.\[6\] Previous reports have described HT in the brainstem, however, extension beyond the pons, including into the ventricular system has not been described other than in hemorrhages from AVM or hypertension.\[7\] Though most brainstem hemorrhages are primary hypertensive hemorrhages, the differential diagnosis should include the possibility of HT. The patient's initial symptoms were likely related to brainstem ischemia with subsequent HT as there is no other clear explanation for the interval appearance of the patient's ICH. Spontaneous HT is a rare, but potential complication of vertebro-basilar territory ischemia. Clinicians should suspect it when a patient with an ischemic infarct in this region has a sudden deterioration.

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References

Sudden appearance of idioventricular rhythm during inhalational induction with halothane in a child with congenital cataract

Sir,
An 11-year-old American Society of Anesthesiologists I female child with bilateral congenital cataract was scheduled for lens extraction under general anesthesia. Inhalational induction was done with halothane, oxygen, nitrous oxide using a semiclosed circle system (fresh gas flows of 6L/min). The child was asked to take deep breaths and halothane was
stepped up in increments of 0.5% every two to three breaths. As the anesthetic depth increased her ventilation was gently assisted, at an inspired halothane concentration of 2.0%, an accelerated idioventricular rhythm at a rate of 80 beats/min was observed on the monitor at end-tidal carbon dioxide (ETCO₂) concentration of 33 mm Hg [Figure 1]. Normal sinus rhythm reappeared when the child was awakened using 100% oxygen. Detailed history and re-examination including a 12 lead EKG and echocardiography did not reveal any cardiac abnormality. It was then noticed that she had shortened metatarsals in both feet [Figure 2] and a short stature (123 cm). This led to the presumptive diagnosis of Albright’s hereditary osteodystrophy (pseudohypoparathyroidism) or Turner’s syndrome. On biochemical investigation the serum calcium levels (8.5 mg %), phosphate (4.8 mg %), alkaline phosphatase (594 IU/L), follicular stimulating hormone and parathormone levels were normal. As she was terrified of needles and sevoflurane was unavailable, subsequent anesthetic induction was also with halothane. The idioventricular rhythm reappeared on spontaneous ventilation (ETCO₂ 35 mm Hg) at an inspired halothane concentration of 2.5% but on administration of atropine (0.1 mg IV), sinus tachycardia occurred. A day later intraocular lens displacement necessitated re-surgery. As an intravenous cannula was in situ, anesthesia was induced using thiopentone, pethidine and vecuronium. Maintenance with oxygen, nitrous oxide, isoflurane was not accompanied by any dysrhythmia though the heart rate decreased to 64 beats/min.

Presence of anatomic features of pseudohypoparathyroidism with normal parathormone and calcium levels is termed pseudo-pseudohypoparathyroidism. Transient decrease in ionized calcium without change in total serum calcium has been observed in these patients and may be caused by hyperventilation during induction resulting in respiratory alkalosis.

Depolarization in the slow response fibers of the sino atrial (SA) and atrio ventricular (AV) nodes occurs due to the inward calcium current. Halothane inhibits this calcium influx, decelerates SA node discharge and prolongs AV node conduction which may predispose patients to develop sinus bradycardia, nodal rhythm, supraventricular or ventricular ectopies. The simultaneous decrease in ionized calcium could have precipitated the idioventricular rhythm in our patient. Isoflurane has a less depressant effect on slow inward calcium current and intracellular calcium accumulation than halothane. This may explain the absence of dysrhythmia with it.

Accelerated idioventricular rhythm or slow ventricular tachycardia (VT) consists of uniform widened QRS complexes at 60-100 beats/min caused by abnormal automaticity of a supraventricular or ventricular focus. It has been observed following acute myocardial infarction, with digitalis toxicity, during cardiac surgery, in patients with rheumatic heart disease or cardiomyopathy but rarely progresses to rapid VT or ventricular fibrillation. Though usually not associated with hemodynamic instability in the absence of left ventricular dysfunction the sudden appearance of this dysrhythmia in a normal child required workup. Atropine administration resulted in accelerating sinoatral (SA) discharge suppressing this arrhythmia. Lignocaine may prolong atrioventricular (AV) conduction and perpetuate the rhythm and is therefore not recommended.

On using Naranjo’s adverse drug reaction probability scale we obtained a score of 10 which makes halothane a definite cause for the event. The dysrhythmia occurred after halothane administration (+2), once halothane was washed out using 100% oxygen normal sinus rhythm reappeared (+1). The idioventricular rhythm (IVR) appeared on first and subsequent induction with halothane (+1), (+2). The IVR did not occur with intravenous induction and isoflurane administration (+1). Though alternative causes of IVR are known e.g. myocardial infarction they were ruled out in this 11-year-old child (+2). The dysrhythmia was objectively recorded on an EKG trace (+1).

The diagnostic features of Albright’s hereditary osteodystrophy are shortened third and fourth metacarpals or metatarsals (92%), short stature (76%), round facies (71%) and obesity (61%) and the presence of these features should alert anesthesiologist about the presence of this syndrome even in the absence of hypocalcemia. In conclusion we report the occurrence of an
episodes were unpredictable and lasted for 10-15 min each. 

sweating, intense fear and sensation of ‘impending doom’. The 
in the outpatient department (OPD) with a seven-day history of 
the patient discharged thereafter. Two weeks later, he presented 
06.11.2006, the second between 02.12.2006 to 04.12.2006, and 
chemotherapy. The first cycle was given between 04.11.2006 to 
3) and cisplatin (50 mg IV on Days 1, 2 and 3) combination 
ifosfamide (2 g IV on Days 1 and 2; followed by 1 g IV on Day 
of the hard palate and positive neck nodes was started on 
A 47-year-old man with arsenic-induced carcinoma 

aneurysms are yet to be documented. We report two cases of 
anxiety is known to be part of ifosfamide-induced neurotoxicity, 
even neurotoxic deaths have been recorded. 

psychopathological symptoms, irreversible encephalopathy, and 
acute onset and subside within 48-72 h, although persistent 
coma.[2,3] These neuropsychiatric complications are usually of 
delusions, hallucinations and psychosis to even seizures and 

The anticancer drug ifosfamide is associated with 5-30%

dependence, although relatively lower doses of 3 g/m 2/cycle 
Ifosfamide neurotoxicity is known to exhibit some dose-

are also not likely to cause panic attacks. Neither subject received 
dexamethasone (8 mg IV) and ondansetron (12 mg IV). These 
(400 mg of mesna IV/g of ifosfamide) and premedication with 
anticancer drugs, both patients received mesna uroprotection 

optic neuritis but not as neuropsychiatric symptoms. Besides the 
manifests as peripheral neuropathy, sensorineural deafness or 

neuropsychiatric symptoms (including hallucinations) were 

category. Interestingly, apart from the panic features, other 

Adverse Events Version 3, the severity of both cases qualifies 

National Cancer Institute Common Terminology Criteria for 

sensorium was clear during the witnessed attacks. As per US 

four or more out of 13 listed symptoms develop abruptly 

in a child with suspected AHO.

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References

1. Doyle DA, Di George AM. Pseudohypoparathyroidism (Albright 
Hereditary Osteodystrophy). In: Behrman RE, Kliegman RM, Jenson 
HB, editors. Nelson’s Textbook of Paediatrics. 17th ed. Philadelphia: 

2. Sunder RA, Singh M. Pseudohypoparathyroidism: A series of three 
cases and an unusual presentation of ocular tetany. Anaesthesia 

3. Atlee JL, Bosnjak ZJ. Mechanism for cardiac dysrhythmias during 

4. Atlee JL. Perioperative cardiac dysrhythmias: Diagnosis and 

a method for estimating the probability of adverse drug reactions. 