wash-out of helium from the alveolar compartment, to one more akin to steady-state (albeit drifting) alveolar elimination of anaesthetic agent delivered to the lung from the tissues via the blood. This more resembles the process of normal CO₂ elimination than helium wash-out. Clearly increasing the VT:FRC ratio has no effect on CO₂ elimination, which is dependent merely on the alveolar ventilation rate.

Finally, the authors ascribe the observed effects to the differences in the blood-gas partition coefficient between the two agents. Whilst this is certainly a dominant factor (particularly in wash-in and wash-out after a short exposure) it does not wholly explain exposure-time dependency (particularly as this characteristic is proposed to differ between small and larger children, yet the blood-gas solubilities do not differ between these groups).

There are two distinct processes underway during emergence: one is elimination of vapour from the alveolar compartment and the other is elution of agent from the tissues and subsequent transfer to the alveolar compartment. The dominance (and rate-limiting effect) of one process over the other varies, and is key to overall elimination. Following prolonged anaesthesia, the elimination characteristics move away from the ‘non-steady-state helium wash-out’ type of kinetics to the ‘drifting-steady-state’ type, where alveolar elimination equals (or just exceeds) tissue elution rate. The effect of blood-gas solubility is less dominant here.

Mapleson’s 1973 model² of volatile agent kinetics is complex, but looked at very simply; there is a large reservoir of volatile agent; notably in fat. The capacity of this reservoir for an agent depends not on the agent’s blood-gas solubility, but on its lipid solubility. The rate-constant for the wash-out (by the blood) of agent from the reservoir is a function of the volume of the reservoir, and the ratio of the ‘blood-gas’ to ‘tissue-gas solubility’. Desflurane’s blood-gas solubility is 3-fold less than isoflurane, but its oil-gas solubility is 5-fold less than isoflurane. Hence the reservoir’s wash-out rate-constant is greater for desflurane than for isoflurane.

So in summary, during emergence from desflurane anaesthesia, the lack of ‘exposure-time’ dependency is as much (or more) a function of its low potency (i.e. low lipid solubility) as its low blood-gas solubility.

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Reference

Supraglottic oedema and cocaine crack abuse

Editor—Many serious medical complications¹ ² and anaesthetic considerations³ have been described in cocaine abuse. Several cases of burn injuries of the larynx have been reported, presenting usually with acute laryngeal symptoms.⁴ We report a case of supraglottic oedema without the specific symptoms of acute laryngeal distress, discovered incidentally in a crack cocaine addict patient during...
assessed for general anaesthesia before lung surgery.

A 70-yr-old man was admitted with a 2 week history of haemoptysis, without dyspnoea or stridor. He was an alcoholic and a heavy smoker with overt signs of chronic obstructive pulmonary disease. Investigations revealed a right lung carcinoma. During bronchoscopy, a supraglottic oedema was noted. The patient was undergoing right pulmonary resection. During the preoperative anaesthesia assessment, he admitted smoking and sniffing large quantities of crack cocaine for the past 18 yr, most recently 2 days ago. He denied having any history of pharyngeal or laryngeal symptoms. He noted having a few episodes of chest pain, within hours of a large dose of crack, which resolved after sublingual nitroglycerin. He never had general anaesthesia before. His physical examination revealed no evidence of respiratory distress, pain, or injury to the tongue, palate or oral surfaces. Preoperative investigations were all within the normal range. The supraglottic oedema prompted us to perform a fibreoptic laryngoscopy immediately before induction of anaesthesia. It revealed atrophy of the nasal mucosa and perforation of the nasal septum. The oral mucosa appeared normal. A supraglottic oedema with mucosal thickening was noted, involving the epiglottis as well as the aryepiglottic, arytenoid and false vocal folds, causing marked impairment of the mobility of both vocal cords. The otolaryngologist was confident however, that a tracheal tube could be inserted. After i.v. administration of propofol 200 mg, fentanyl 100 μg and suxamethonium 100 mg, the trachea was smoothly intubated under direct laryngoscopy with a 6.0 mm cuffed microlaryngeal tube. The supraglottic area was exposed again for biopsies and bacteriology swabs. Frozen section revealed oedematous changes without evidence of malignancy. We decided that a tracheostomy should be done before lung surgery because: (i) the insertion of a double-lumen endobronchial tube (DLET) in an oedematous larynx could be traumatic; (ii) the airway may be compromised following extubation; and (iii) impaired mobility of the vocal cords could interfere with coughing and clearing of bronchial secretions after operation. Following insertion of a short left DLET, right lung resection was performed with one lung ventilation, with invasive monitoring, under general anaesthesia and thoracic epidural analgesia. Antibiotics and i.v. steroids were given. The perioperative period was uneventful. Pathology of the laryngeal biopsies revealed fibrosis with non-specific oedematous changes. Fibreoptic laryngoscopy 4 days later showed marked improvement of the upper airway oedema allowing successful removal of the tracheostomy tube.

To our knowledge, this is the first incidentally discovered case of cocaine-related supraglottic oedema in a crack addict patient. Cocaine burns of the upper airway have been reported in 22 cases, with various and sometimes puzzling clinical manifestations, but with related symptoms such as hoarseness, dysphonia, odynophagia, dysphagia or stridor.4,5 In our opinion, the anaesthetic management helped in the uneventful course of the surgery. Recommendations concerning acute laryngeal oedema are controversial and range from observation in the ICU, with or without local anaesthetics, steroids and antibiotics, to intubation or tracheostomy.6,7

The fortuitous discovery of this patient’s laryngitis adds another insidious clinical presentation that anaesthetists should be aware of in any patient with a history of drug abuse. Even in the absence of evident laryngeal symptoms or respiratory distress, we recommend careful history and discussion between the anaesthetist and otolaryngologist for adequate airway management before surgery.

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doi:10.1093/bja/ael304

Pneumorrhachis presenting as quadriplegia following surgery in the prone position

Editor—Pneumorrhachis, or air within the spinal canal, although a recognized complication, is a very rare occurrence. This term was first used by Newbold and colleagues to define their case with air in subarachnoid space at the cervical level.1 It is also referred to as aerorachia, intraspinal pneumocele, pneumosaccus, pneumomyelogram or simply intraspinal air.2 Cervical pneumorrhachis occurs mostly