assessment for general anaesthesia before lung cancer 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 fibreoptic bronchoscopy, a supraglottic oedema was noted. The patient was undergoing right pulmonary resection. During the preoperative anaesthesia assessment, he admitted smoking and snifffing 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. The supraglottic oedema 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. The supraglottic oedema 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. The supraglottic oedema 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.

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. 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.

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. It is also referred to as aerorachia, intraspinal pneumocele, pneumosaccus, pneumomyelogram or simply intraspinal air. Cervical pneumorrhachis occurs mostly
after traumatic episode or secondary to infection. To the best of our knowledge, it has never been reported after an elective cranial surgery. We report a case of cervical pneumorrhachis following an elective posterior fossa surgery performed in prone position.

A 63-yr-old male diagnosed with left cerebellar meningioma was planned for sub-occipital craniotomy in prone position. Preoperative evaluation of the patient was unremarkable for any medical illness. All routine investigations were within normal limits. The surgery lasted 4 h and was uneventful. After surgery, the patient was turned supine and anaesthesia discontinued. Neuromuscular block was adequately reversed. Though the patient was breathing adequately and regularly and was responding to verbal commands, he was unable to move any of his limbs. At this juncture the operating surgeon suspected cervical cord injury during positioning as the likely cause of quadriplegia. Methylprednisolone was administered in recommended dose. The trachea was not extubated and the patient was immediately taken for postoperative computed tomography (CT) scan. The CT scan revealed presence of air not only in the operative cavity, but also in the subdural space of posterior fossa and spinal canal (Fig. 1), with normal cervical cord. The patient was then shifted to neurosurgical intensive care unit and mechanically ventilated with 100% oxygen. Methylprednisolone infusion was later discontinued. Four hours after surgery, the patient began to show some movements in his upper limbs and 7 h later he regained normal tone and power in all four limbs. The patient was successfully extubated the next morning. Five days later, the patient was discharged neurologically intact.

In our case the CT scan showed pneumorrhachis with no obvious spinal cord changes. We believe that this could be due to the compression caused by the accumulated air in the posterior fossa and spinal canal. The most likely explanation of cervical pneumorrhachis could be the entrapment of air in the posterior fossa at the time of dural closure. As the patient had his head down, this could have favoured air entry through the foramen magnum and into the cervical region. Cayli and colleagues also proposed that facedown position allowed air to pass through foramen magnum, in the case they reported. It is a routine practice to flush out air from the surgical cavity using normal saline, prior to closing the dura mater. It is likely that flushing out of air in our case was incomplete. The fact that our patient recovered spontaneously following ventilation with 100% oxygen suggests gradual resolution of air from the site. Administration of 100% oxygen aids in absorption of entrapped air from body cavity. As there were no cord changes in the CT scan, the role of methylprednisolone in this case is of doubtful value. Many causes of pneumorrhachis have been reported in the literature, and these can be broadly classified into iatrogenic, non-traumatic and traumatic. Iatrogenic causes are the most common. Sustained neurological deficits as a result of pneumorrhachis have been reported after epidural analgesia.

Spontaneous resolution occurs in most cases of epidural space pneumorrhachis, which permits conservative management. To prevent accumulation of the intracranial air efforts must be directed to minimize cerebrospinal fluid loss, maintain hydration for proper cerebral perfusion and also allow the brain to regain its normal contour by bringing the end tidal carbon dioxide to normal levels. Subdural injection of saline to displace the residual air may also be useful. Similarly, a reverse Trendelenberg position or an elevated head may prevent air from entering the cervical spinal canal. We suggest that pneumorrhachis, like pneumocephalus, be recognized as yet another complication of prone position.

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Fig 1 Postoperative computed tomography scan showing air within the cervical spinal canal.
Massive haemorrhage during anterior cervical fusion attributable to a tear at the junction of the subclavian and internal jugular veins

Editor—Injury to major blood vessels during anterior cervical fusion is rare but could be catastrophic. We report a case of massive haemorrhage during cervical fusion as a result of a tear at the junction of subclavian and internal jugular vein. A 150 cm, 84 kg, 46-yr-old female with radi- cular pain in the right upper arm as a result of disc prolapse at C 5-6 level underwent anterior cervical fusion under general anaesthesia. One hour after the start of surgery, massive haemorrhage was noted. The surgeons immediately suspected a tear at the junction of internal jugular vein and subclavian vein possibly caused during retraction. We immediately secured i.v. lines in both the ankles with 16 G cannulae and started rapid infusion of crystalloids, followed by 1000 ml of colloid. We also ordered for 5 units of packed red blood cells (PRBCs) and 3 units of fresh frozen plasma (FFP). There was a delay in securing haemostasis because of profuse bleeding and difficult access to the bleeding site which was behind the clavicle and not exposed by the surgical incision. During this period systolic arterial pressure decreased to 90 mm Hg. Invasive arterial pressure monitoring was started after cannulating the dorsalis pedis artery. As the bleeding could not be stopped, a cardio-thoracic surgeon was called for and he had to cut open the clavicle to repair the venous tear. While the vascular repair proceeded, we transfused 9 units of PRBCs, 6 units of FFPs and 3 units of platelet rich plasma. Total estimated blood loss was about 8 litre. Throughout the procedure systolic arterial pressure was maintained between 80 and 100 mm Hg without using ino-tropes or vasopressors. In view of massive blood transfusion, anticipated obstruction of internal jugular vein (secondary to vascular repair) and possibility of airway oedema, we electively ventilated the lungs for 24 h in the ICU. Postoperative recovery was uneventful apart from a right-sided phrenic nerve palsy.

Vertebral artery injury during anterior cervical fusion is reported in the literature. However, during this procedure, there was massive haemorrhage as a result of a tear at the junction of the subclavian vein and internal jugular veins. This site of bleeding was not accessible for compression of the vessels for haemostasis and was not exposed by the surgical incision for anterior cervical fusion. Possible cause of vascular injury in this case is unduly forceful retraction of tissues attributable to limited exposure of the surgical area.

The anaesthetic implications of such an event include the danger of concealed bleeding presenting as haemodynamic collapse, and as bleeding cannot be controlled till the clavicle is split to obtain access to the injured site, the anaesthetist has to maintain the patient haemodynamically stable for a prolonged period.

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doi:10.1093/bja/ael306