*European Journal of Anaesthesiology* 2003; **20:** 753–759

ISSN 0265-0215

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# Subcutaneous emphysema, pneumomediastinum and pneumothorax after laryngeal laser surgery

EDITOR:

A 59-yr-old male underwent laryngeal microsurgery with a carbon dioxide laser for extirpation of a polyp in the anterior commissure of the vocal cord and Reinke oedema in the right vocal cord. The patient had chronic bronchitis, arterial hypertension and obesity (body mass index 31.5 $kg\,m^{-2}$); he had suffered periorbital emphysema following facial trauma some years before. On three occasions between 1987 and 1988, he had undergone resection for Reinke oedema. The electrocardiogram, non-invasive arterial pressure and pulse oximetry were monitored in the operating room. General anaesthesia was induced with propofol 2 $mg\,kg^{-1}$, fentanyl 5 $\mu kg^{-1}$ and rocuronium 0.6 $mg\,kg^{-1}$. Laryngoscopy and intubation were carried out with a 7 mm orotracheal tube (Laser-Trach®; Kendall, Sheridan, Mansfield, USA) and the cuff was then filled with saline. The patient presented as grade II on the Cormack–Lehane scale and the trachea was intubated with no difficulty without the need for a rigid guide. The lungs were artificially ventilated conventionally with anaesthesia being maintained with sevoflurane 2% in oxygen and air ($FiO_2 = 0.4$); end-tidal carbon dioxide concentration was measured, and muscular relaxation was monitored by accelerometry (Tof-Guard®; Organon Técnica, Boxtel, The Netherlands).

Correct placement of the suspension laryngoscopy was achieved on the fourth attempt by the surgeon. Moistened swabs were placed around the endotracheal tube cuff to shield it from the laser's energy and to protect the subglottic tissue. Excision of the anterior commissure polyp followed by elimination of the Reinke oedema in the right vocal cord was performed with a carbon dioxide laser. The procedure lasted 90 min. High pressures were not required during artificial ventilation and no untoward incidents were observed. On completion of surgery, the endotracheal tube was removed uneventfully and the patient recovered effective spontaneous ventilation. During the immediate postoperative period, the appearance of slight subcutaneous emphysema was observed in the anterior side of the neck progressing towards the eyelids. Respiration was normal, maintaining oxygen saturation of 97–98% with added oxygen via nasal spectacles at 21 breaths $min^{-1}$. The emphysema progressively increased extending to all of the face, neck, thorax and extremities. Three hours after the endotracheal tube was removed, respiratory difficulty was reported, although oxygen saturation was maintained at 96% with a facemask. A chest radiograph showed large areas of subcutaneous emphysema, pneumomediastinum and a pneumothorax. The patient was sedated with propofol 2 $mg\,kg^{-1}$ and a conventional laryngoscopy was performed before the administration of succinylcholine 1 $mg\,kg^{-1}$ for endotracheal intubation with an 8 mm orotracheal tube. The intubation was without difficulty despite the cervical and facial emphysema. Bilateral pleural drains were inserted. After intubation, arterial blood-gas analysis showed a respiratory acidosis. After sedation, the patient developed a tendency to hypotension, which was controlled by a dopamine infusion 5 $\mu g\,kg^{-1}\,min^{-1}$. The patient was transferred to the intensive care unit where artificial ventilation of the lungs was instituted. His airway was explored via a fibreoptic-bronchoscopy, which showed laceration towards the front of the trachea in the mucosa of the subglottic region at the level of the cricothyroid membrane. Artificial ventilation was discontinued and the tracheal tube was removed after 3 days; the generalized emphysema gradually reduced over the next few days; the pleural drains were removed 4 days after their insertion and the patient was discharged from hospital after 11 days.

The differential diagnosis of the subcutaneous emphysema included four possible causes: (a) alteration of the cutaneous barrier (in surgical procedures such as tracheostomy, dissection of the neck or external trauma), (b) alterations of the mucosal or cartilaginous barrier (intubation, endoscopic procedures, dental surgery, fracture of facial bones, thoracic drains,

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Accepted for publication October 2002 EJA 1239

foreign bodies or neoplasms), (c) barotrauma (positive pressure ventilation, Valsalva manoeuvre, bronchial asthma, labour) or (d) bacterial infections with gas producing micro-organisms [1–7].

A possible aetiology of the emphysema in this case was the traumatic lesion of the mucosal barrier in some part of the upper airway. A suspicion of an alveolar lesion by barotrauma was discarded because artificial ventilation was uneventful during the intraoperative period.

Rupture of the mucosa may occur in the larynx, pharynx or oesophagus. We consider that in this case, the rupture was produced at the laryngeal level since laser surgery was performed at this site. Fibreoptic bronchoscopy showed an area of subglottic erosion, and during hospitalization in the intensive care unit no signs of mediastinitis (frequent in cases of perforation of pharyngeal or oesophageal mucosa [1–4]) were reported. The sequence of events points to a laryngeal lesion since emphysema was not observed until the patient was extubated, at which time the lesion behaved as a flap valve, so that air was gradually trapped in the subcutaneous tissue [3]. Until then, the air leakage through the laryngeal lesion was not manifest since the airway above the cuff of the endotracheal tube was not pressurized. The origin of the laryngeal lesion may have occurred during intubation [1,7] or during surgery [2]; we believe it was most likely produced during surgery since endotracheal intubation was performed without difficulty. On the one hand, placement of the laryngoscope was not achieved until the fourth attempt; however, it is unlikely that a subglottic lesion would be caused by this manoeuvre. On the other hand, complications have been reported with the use of a laser in the airway, including the appearance of subcutaneous emphysema and pneumothorax [2]. These lesions may be avoided by protecting the subglottic tissue – in this case, moistened swabs were placed in the subglottic region around the cuff, but it may not have provided sufficient protection. In addition, if surgery is prolonged, as in this case, the swabs need to be checked regularly in case they dry out. Drying out not only increases the risk of airway fires, but also of soft tissue injury. These lesions include perforation of the cricothyroid membrane [1] or the cartilaginous laryngeal tissue with dissection of the tissue being produced by the turbulent airflow circulating through the upper airway and becoming manifest when the tracheal tube is removed. The picture is aggravated if the patient coughs or makes inspiratory efforts.

The appearance of subcutaneous emphysema during the postoperative period should be considered a severe complication, as the airway can be compromised [1–7]. As soon as this complication is detected, the patient requires surveillance (electrocardiogram, blood pressure, oximetry). The appearance of tension pneumomediastinum or pneumothorax requires emergency decompression by placement of pleural drains since, in addition to ventilatory compromise, haemodynamic failure may occur secondarily to the restriction in venous return [1]. Intubation in these patients may add further difficulties because of the cervicofacial distortion produced by the emphysema [6]. On stabilization of the patient, localization of the lesion by fibreoptic bronchoscopy or oesophagoscopy allows the aetiology to be established and surgical correction of the lesion to be performed. Early detection of an airway lesion caused during laser surgery is important; however, it seems unlikely that early detection before extubation will be possible in most cases unless fibreoptic bronchoscopy is performed routinely before extubation. Close observation for signs of subcutaneous emphysema is likely to be the only option in most cases. Endotracheal intubation should be maintained so that the upper airway 'rests' until scar formation of the lesion is initiated. This implies that the lesion needs regular assessment by fibreoptic endoscopy, and if the endotracheal tube is removed, close surveillance of the patient is vital.

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# Subacute epidural abscess after spinal cord stimulator implantation

EDITOR:

Electrical stimulation of the nervous system with the aim of treating chronic pain is performed at different levels of the neuraxis, e.g. the peripheral nerve, trigeminal ganglion and/or root, spinal cord, thalamus and motor cortex. Shealey and colleagues first described the use of spinal cord stimulation in 1967 [1]. We report on a case of subacute epidural abscess 1 yr after spinal cord stimulator implantation and explain the clinical features in order to highlight the significance of the radiological diagnosis.

A 59-yr-old male had a fall and was admitted to hospital because of unrelieved sciatic pain. Magnetic resonance imaging (MRI) revealed an L5–S1 disc herniation as the source of pain and lumbosacral spinal surgery was performed. Surgery consisted of an L5–S1 decompression (lumbar discectomy). A few weeks later, he developed intense low back pain with right episodic sciatica. A computed tomographic scan, MRI and myelography showed periradicular fibrosis and arachnoiditis with right-sided predominance.

Two years later, the patient consulted the pain clinic complaining of chronic low back pain resulting from 'failed back surgery syndrome'. The pain was controlled by medical treatment (analgesics, opioids, adjuncts), epidural sessions (local anaesthetics, corticosteroids) and transcutaneous electrical nerve stimulation (TENS).

Another 2 yr later, the low back pain became uncontrollable and it was decided by anaesthesiologists and neurosurgeons to perform an L4–L5–S1 stabilization procedure. The immediate postoperative course was good. The low back pain improved, but still the episodic sciatica and radicular pain resulting from paraesthesia in the right foot and numbness in the heel persisted. The pain was successfully controlled with TENS and medical treatment. A few months later his situation worsened with invalidating low back pain again, dysesthesia in the left leg and gemellus neurological amyotrophy. Later, and with clear evidence of failed back surgery syndrome, it was decided to implant a T10–T11 spinal cord stimulator. Before the operation, the patient underwent a baseline quantitative evaluation of functional capacities by a psychologist. The system was implanted through a standard laminectomy, and placing a temporary electrode under fluoroscopic control for a routine 7 day trial. The patient reported an estimated 80% relief of pain (medication was maintained) and his physical activity improved. In a second operation, the definitive implant (Medtronic-Itrel 3®; Medtronic ibérica, Barcelona, Spain) was introduced through the laminectomy. The procedures were performed by an experienced consultant under aseptic conditions and with antibiotic prophylaxis (cefotaxime 1 g intravenously (i.v.)). The implant was placed with particular care in order to prevent paraparesis and paralysis (Fig. 1).

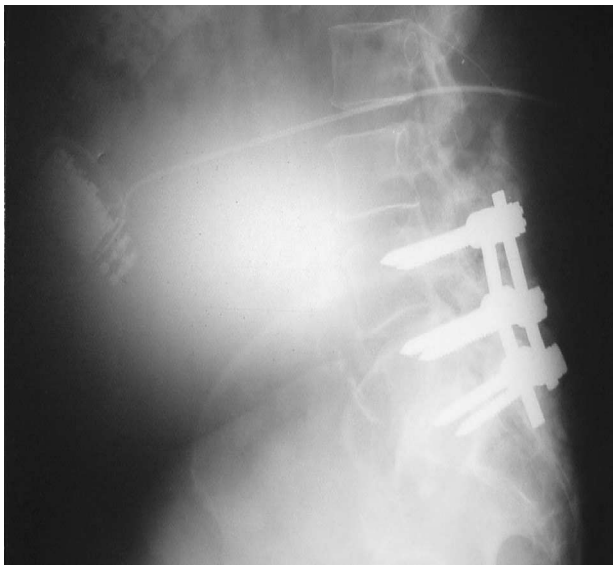

**Figure 1.**
*L4–L5–S1 stabilization procedure with a T10–T11 spinal cord stimulator. The system was implanted through a standard laminectomy and, under fluoroscopic control, a temporary electrode was introduced for a routine 7 day trial.*

One month later, the spinal cord stimulation was ineffective owing to tolerance and it was necessary to increase the medication. The patient described severe pain in the site of the pulse generator with hyperalgesia and allodynia and without inflammatory signs, and he wanted the implant to be removed. On removal, the site was surgically explored and wound infection was observed, and septic material sent for cultures was negative. After placing a Penrose-type drainage system in the wound, ciprofloxacine 750 mg i.v. was administered every 8 h for 1 week. Given that the patient had responded well to spinal cord stimulation, and with the agreement of the patient and accepting

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Accepted for publication October 2002 EJA 1311

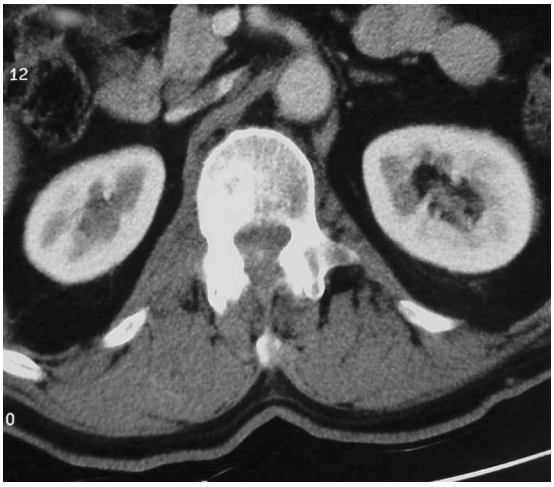


**Figure 2.**
*Computed tomographic scan showing an increased density in the posterior epidural space (T12) extending into the paravertebral space.*

the risk of infection, it was decided to remove the generator and the wire and leave the epidural electrode in place.

Six months later the patient consulted again for progressive leg weakness (paraparesis), functional impotence of the right psoas muscle, dysesthesia in the right leg and physical disability. He had apyrexia and no sphincteral dysfunction. He was unable to flex his right hip in the physical examination. The cardiopulmonary and abdomen examinations were unremarkable. The laboratory studies showed a white blood cell count of $10.8 \times 10^9\,L^{-1}$ and a C-reactive protein of $4.8\,mg\,dL^{-1}$. Chest and abdominal radiographs were normal. The computed tomographic scan showed a density increase in the epidural posterior space (T12) extending into the paravertebral space (Fig. 2). MRI showed an inflammatory process at the right psoas with muscular atrophy and did not detect an intraspinal space-occupying lesion. Electromyography showed L5–S1 irritation and denervation. Electromyography showed L5–S1 irritation and denervation confirming the existence of myeloradicular compression. As an empyema was suspected, a decompressive T11–T12 laminectomy was performed, and when the electrodes were removed, pus and fibrotic material were revealed. *Staphylococcus aureus* resistant to methicillin and *Pseudomonas aeruginosa* were found after being isolated from the tissue and cultured. The therapy was changed to ciprofloxacine 750 mg i.v. 12 hourly. The patient slowly recovered from surgery, showed improvement in the clinical symptoms and was discharged after 15 days of antibiotic therapy with a residual paresis of the right psoas muscle.

The indications for spinal cord stimulation include syndromes causing neuropathic pain in the extremities, axial pain related to mechanical factors such as spinal stenosis, mixed pain conditions (e.g. failed back surgery syndrome), intractable angina pectoris and peripheral vascular disease [2]. The pain resulting from failed back surgery syndrome is difficult to treat. Although some patients develop fibrosis in the operated nerve root after disc herniation, there is not always an association between epidural fibrosis and sciatica in the lumbar postdiscectomy syndrome. Spinal cord stimulation can be considered if the pain persists [3] despite conservative treatment and there are no significant psychological antecedents and co-morbidity. Complications associated with column stimulator implants may be technical or functional. Many authors state that the technique does not share the risks of surgery; however, this procedure is not risk free and can occasionally be life threatening [4]. Technical and functional complications after the implantation of spinal cord stimulation devices have been reported [5]. Technical complications are defined as those occurring in the immediate postoperative period due to problems associated with the surgical procedure (e.g. transitory or severe pain, seromas or leakage of cerebrospinal fluid). These are mainly difficulties with the hardware such as a displaced electrode, treated by reoperation and repositioning, or hardware malfunction. Fortunately, the possibility of changing electrode combinations non-invasively to optimize the topography or stimulation reduces the number of displacements requiring further procedures. Other complications recorded are superficial infections (successfully treated by antibiotics or removal), subcutaneous haematomas, cerebrospinal fluid leakage (spontaneously resolved) and psoas abscess. Serious neurological injuries requiring surgical intervention have been reported but never as a subacute event. Previously reported functional complications are mostly based on stimulation failures.

The correct implantation of the spinal cord stimulation device is a prerequisite for a successful outcome. The implantation of the electrode should be performed percutaneously or through a small laminectomy under fluoroscopy in strictly aseptic conditions. Antibiotics should be given before, during and after operation to reduce the risk of wound infection and meningitis. The clinical symptoms of spinal epidural abscess are pain, fever and rapidly progressing weakness. Pain is usually present either along the spine or is radicular. The duration of pain before the appearance of symptoms is generally 2 weeks or less, but in some chronic cases, it may be several months or longer. Risk factors include impaired immune status (e.g. diabetes mellitus, renal failure, alcoholism, malignancy), intravenous drug abuse and infections of the skin or other tissues. Two-thirds of epidural infections are the result of haematogenous spread from the

skin, soft tissue or deep viscera. One-third results from the direct extension of a local infection to the subdural space. Most cases are due to *S. aureus*, although Gram-negative bacilli, *Streptococcus*, anaerobes and fungi can also cause epidural abscesses.

In the present case, the patient had no risk factors and the epidural abscess developed as a subacute process 1 yr after spinal cord stimulator implantation. Perhaps the original operative cultures were falsely negative. The presence of a foreign body and the growth of *S. aureus* (resistant to methicillin) in purulent material cultures suggest that the contaminating agent was introduced by the catheter from the skin. It is not known whether the skin was the original focus of the infection, which would seem unlikely 1 yr after the last surgical procedure, or the result of a haematogenous dissemination from other tissue. Screening for methicillin-resistant *S. aureus* colonization should be considered in patients at high risk of this infection because the result could affect the choice of antimicrobial prophylaxis and empirical therapy. We took a calculated risk and decided not to remove the epidural electrode in the hope of avoiding another invasive procedure and possibly attaching a new generator.

In conclusion, even when a spinal cord stimulation lumbar catheter is implanted following all the appropriate steps, it is important to consider the possibility of subacute epidural abscess as a complication.

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# Retropharyngeal or cervicomediastinal haematomas following stellate ganglion block

EDITOR:

Stellate ganglion block has been used for the diagnosis and treatment of conditions involving chronic pain. The incidence of various complications during or after stellate ganglion block has been recognized and the commonest of these are brachial plexus, phrenic nerve or recurrent laryngeal nerve block, intra-arterial or intravenous injection, and pneumothorax and subarachnoid injection [1]. Airway obstruction due to retropharyngeal or cervicomediastinal haematomas is a rare complication following stellate ganglion block and is often not mentioned in textbooks. Only our previous case report [2] has been indexed in the English language literature. However, another 21 reported cases of this complication that have been reported in the Japanese literature have not been indexed in MEDLINE. We report another similar case and seek to increase the awareness of the life-threatening complication of stellate ganglion block.

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Accepted for publication November 2002 EJA 1327

Our patient was a 60-yr-old male who presented with neck–shoulder pain. His medical history included diabetes mellitus and obesity. He had received nine sequential stellate ganglion blocks, without complications, and his pain was improving. The tenth block was performed using an anterior paratracheal approach. The needle (23 G, 2.5 cm) was inserted until the tip reached the transverse process of the seventh cervical vertebra. After negative aspiration, mepivacaine 1% 8 mL was injected, the needle was withdrawn and digital pressure was applied to the insertion point for 5 min. One hour later the only complication was Horner's syndrome. The patient was then permitted to leave the outpatient clinic to go home. However, 2.5 h after the stellate ganglion block, the patient became hoarse and after another 1 h had to lie down complaining of dyspnoea and neck pain. His wife then brought him to the Emergency Department of our hospital. On examination, he was afebrile, with a blood pressure of 153/100 mmHg, a heart rate of 70 beats $min^{-1}$ and a respiratory rate of 25 breaths $min^{-1}$; electrocardiography revealed no abnormalities. There was no evidence of any pneumothorax. The swelling and tenderness in the anterior

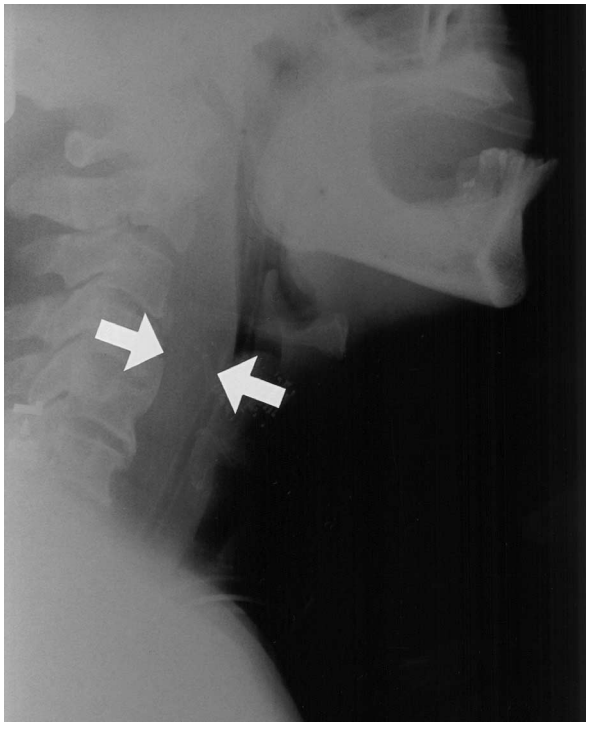

**Figure 1.**
*Lateral radiograph of the neck showing widening of the retropharyngeal space (white arrows).*

region of the neck increased. An otorhinolaryngologist examined the patient, but was unable to make a diagnosis. Since the patient's wife mentioned that her husband had undergone a stellate ganglion block that morning, the anaesthetist was called and by the time he arrived the patient's dyspnoea was quite severe. Endotracheal intubation was attempted but the usual anatomical landmarks could not be seen at laryngoscopy; the soft tissues were grossly oedematous and intubation proved impossible. The otorhinolaryngologist performed an emergency tracheotomy that relieved the dyspnoea significantly. Lateral neck radiography subsequently showed a large haematoma in the neck (Fig. 1). Laboratory data revealed an association between the haematoma and a decrease in haemoglobin concentration ($10.8\,g\,dL^{-1}$). Thus, the cause of the patient's difficulties was a haematoma resulting from the stellate ganglion block. Although surgery under general anaesthesia was undertaken to evacuate the haematoma, little blood could be evacuated and precise identification of the vessels responsible was not possible. The stoma was closed 37 days after the tracheotomy. The patient was discharged from the hospital with no further complications.

When a vessel in the neck is injured as a result of stellate ganglion block, sequelae are not typical. Minor haematomas may develop, but usually they resolve spontaneously [3]. Development of a large haematoma in the neck to the point airway obstruction occurs is exceedingly uncommon. The exact mechanism of severe dyspnoea due to the haematoma remains unclear. It was thought that the needle might have ruptured a small retropharyngeal blood vessel, so accounting for the substantial duration for the blood to collect and form a space-occupying mass of significant size. This would explain the delay in the onset of symptoms following stellate ganglion block. The primary cause of airway obstruction may have been laryngeal oedema secondary to venous and lymphatic obstruction by the haematoma [4]. Direct compression of the trachea by the haematoma is unlikely to be a feasible mechanism of airway obstruction because a normal trachea is rigid and largely resistant to compression.

This complication may give rise to difficulties in diagnosis. A history of recent stellate ganglion block is therefore extremely useful information. Symptoms can vary with the degree of bleeding and may initially present as neck pain, swelling in the neck, dyspnoea and pharyngeal discomfort. However, early symptoms are frequently non-specific [2]. Computed tomography and lateral neck radiography represent useful investigations in differential diagnosis of dyspnoea as a complication of stellate ganglion block.

If the patient displays any adverse signs following stellate ganglion block, close monitoring should be maintained in hospital until they resolve. When the patient presents with mild dyspnoea, the airway must be monitored and preparations made for possible endotracheal intubation or tracheotomy. Endotracheal intubation may be sufficient in less severe cases, but in severe cases the trachea is extremely difficult to intubate even using small endotracheal tubes. Tracheostomy should be performed when endotracheal intubation is unsuccessful or considered dangerous.

Unfortunately, we were unable to identify useful precautions for the prevention for this complication. Fortunately, our patients undergo stellate ganglion block without complications and rarely display adverse symptoms immediately following the procedure. The onset of initial signs following stellate ganglion block can range in time from 2 to 3 h, which means that diagnosis and treatment are often delayed. It is therefore important that not only physicians, but also patients undergoing stellate ganglion block should be informed about this complication.

In conclusion, retropharyngeal or cervicomediastinal haematomas following stellate ganglion block are a rare but life-threatening complication. Prompt recognition and treatment is therefore vital.

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# Breathing or ventilation via a mouth mask for rhinoplasty operations in the early postanaesthetic period

EDITOR:

Nasal packs are usually used to maintain haemostasis and stabilization of the reconstructed nose after rhinoplasty. Since the nostrils are completely blocked by the packs, breathing is only possible through the mouth once endotracheal extubation has taken place at the end of the operation. Compression of the fragile nose by a standard facemask could spoil the surgical result. Furthermore, immediately the endotracheal tube has been removed, breathing through a standard facemask is extremely difficult because the usual adult facemask is difficult to seal on the face due to the plaster splint and dressing.

Nagaro and colleagues developed a method to permit fibreoptic tracheal intubation in anaesthetized patients with ventilation performed only through the mouth [1]. They applied a child's mask over the mouth (mouth mask) instead of a standard anaesthesia adult facemask. In their method, an oral airway was usually inserted to overcome any airway obstruction and the nostrils were plugged with cotton if a leak through the nares occurred. Arai and colleagues reported that fibreoptic tracheal intubation could be achieved using a mouth mask in cases of difficult tracheal intubation [2]. They used the same method as Nagaro and colleagues [1], but used an infant's or child's mask, or else a specially constructed device, as a mouth mask. Arai and colleagues concluded that the mouth mask method for fibreoptic tracheal intubation was safe, useful and practical in instances of difficult intubation. After rhinoplasty operations, in the early postanaesthetic period, we have used a silicone child's mask through which the patient is permitted to breathe only through the mouth. We prefer a child's silicone facemask (No. 2 Silicone Child Mask, GaleMed Corp., I-Lan Hsien, Taiwan) as a mouth mask because of its elasticity and pliability over the mouth. The mask is applied only over the mouth; an oral airway is inserted to overcome any upper airway obstruction (Fig. 1). Patients breathed spontaneously through the child's mask in the operating room until they regained consciousness and their cognitive functions; standard monitoring, including pulse oximetry, was maintained in the recovery room. The same child's mask was used if supplemental oxygen was necessary. In our experience, breathing is much facilitated with a mouth mask compared with a standard anaesthesia mask in these patients; we have found it helpful on occasions to assist breathing. We have been using this method after rhinoplastic surgery for 1 yr and have found it easy and safe to use.

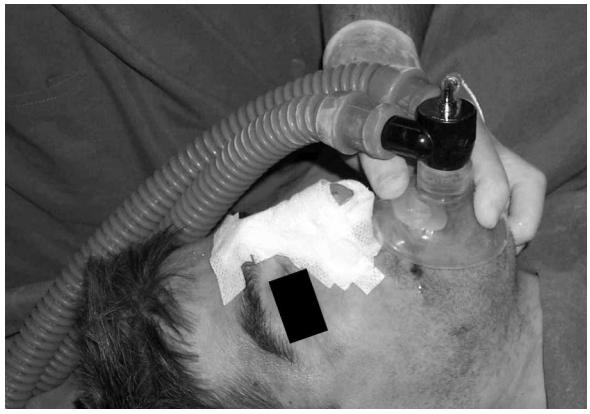

**Figure 1.**
*Application of a mouth mask after rhinoplasty surgery.*


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Accepted for publication November 2002 EJA 1340