(CMV), Epstein-Barr virus, HIV and human T-cell lymphotrophic virus DNA in skin biopsy specimens in 29 vitiligo patients and 22 control subjects and only CMV-DNA was identified in 38% of the patients, whereas all control subjects were negative. Intercellular adhesion molecule-1 (ICAM-1) on the surface of epidermal keratinocytes and melanocytes is likely to greatly influence cytotoxic damage of these cells in diseases like photosensitive lupus erythematosus, lichen planus, erythema multiforme, and vitiligo. It is proposed that disease-specific induction of ICAM-1 by factors such as ultraviolet radiation and herpesvirus infection, is an important determinant in triggering these skin diseases and in determining the pattern of disease.[7] Our results also comply with the immunologic proposal of vitiligo as herpesviruses may trigger ICAM-1 expression on melanocytes which may activate autoimmune destruction of these cells.

In conclusion, HHV-6 IgG seropositivity showing past HHV-6 infection is related to vitiligo. C-reactive protein, which is elevated in acute or chronic inflammation and tissue damage, is related neither to vitiligo nor to HHV-6. Human herpesvirus 6 infection in a genetically susceptible host could potentially mediate the destruction of melanocytes by induction of aberrant humoral and cell-mediated immunological responses eventually causing vitiligo. Nevertheless, advanced immunologic and pathologic proofs of viral infection in skin is needed to confirm vitiligo-virus relationship.

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From the Departmentof Microbiology and Clinical Microbiology (Adiloglu, Can) and the Department of Dermatology (Basak, Akkaya), Suleyman Demirel University Medical School, Isparta, Turkey. Address correspondence and reprint requests to Dr. Ali K. Adiloglu, Posta Kutusu 61, 32100, Isparta, Turkey. Tel. +90 (246) 2324811. Fax. +90 (312) 4250760. E-mail: aadiloglu@yahoo.com

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Use of laryngeal mask airway for the care of rhinoplasty

Jamil S. Anwari*, MBBS, FFARCS,* ***Javaid Bhatti****, MBBS.*

Some operations on the face, such as rhinoplasty, require care to preserve the delicate surgical work. In anesthetized patient after extubation, firm application of face mask (such as Rush™) is required for adequate spontaneous or manual ventilation. Pressure exerted on the nose by the face mask may change the shape of newly reconstructed nose. Subcutaneous emphysema has been reported after rhinoplasty where air was pumped through the lateral osteotomy incision.[1,2] The risk of emphysema would probably increase if patient receives positive pressure ventilation via face mask. Awake extubation is often associated with straining or bucking increasing the nasal bleeding, which is undesirable in this situation. To avoid both tracheal extubation response and face mask ventilation, the trachea can be extubated during deep anesthesia and ventilation can be maintained with laryngeal mask airway (LMA). As compared to Guedal airway, LMA provides easier airway maintenance.[3] Use of LMA after extubation during emergence from anesthesia compared with awake extubation or extubation in anesthetized without LMA, has been associated with less respiratory complications during recovery period.[4] Laryngeal tube has been used during emergence from anesthesia in a patient with an unstable neck.[5]

We present results on 15 cases of rhinoplasties where at the end of the operation patients were extubated during deep anesthesia and ventilation was maintained through LMA until patients regained consciousness. Mean age of patients were 32 years [standard deviation (SD) of 5] and mean body weight is 68 kg (SD = 8.7), and they were either American Society of Anesthesiologist class I or II. All our patients received oral pre-medication of diazepam and metoclopramide 10 mg each orally 2 hours before the operation. After establishing IV line and standard monitoring, general anesthesia was induced with fentanyl (2 μg/kg), thiopentone (3-5 mg/kg) and cisatracurium (0.5 mg/kg). Patients were ventilated with face mask until all 4 twitches of train of 4 disappeared, and were then intubated. Anesthesia was maintained with sevoflurane, nitrous oxide and oxygen. Additional narcotic (morphine 1.5 mg/kg, intramuscularly), nonnarcotic

analgesics (diclofenac 100 mg; rectally) and dexamethasone 8 mg were given before the start of the operation. Average duration of operation was 125 minutes (SD = 17). After the operation, throat was cleared and when the fourth twitch of train of 4 was detected, residual effect of neuromuscular blocking drug was reversed with neostigmine (2.5 mg) and glycopyrrolate (0.4 mg). Anesthesia was maintained with sevoflurane with 100% oxygen and carbon dioxide was allowed to rise (end-tidal CO_2 between 40-60 Kpas). Patients were extubated when bleeding from the naso-pharynx appeared to have stopped and LMA was inserted along with a roll of gauze between the teeth. Sevoflurane was then switched off and patients were either breathing spontaneously or ventilated manually receiving 100% oxygen. Adequate placement of LMA was judged with: a) the ability to hand ventilate easily and b) after disconnecting the breathing circuit, clear breath sounds heard at the tubal end of LMA. To stimulate spontaneous ventilation, small doses of doxapram (20 - 60 mg) were used in 60% of the patients, and in addition 2 out of 15 patients required nalaxone (80 μg). With adequate spontaneous breathing through LMA, patients were transferred to post anesthesia care unit where oxygen was given through LMA. When patients regained consciousness, LMA was removed, throat was cleared and oxygen was given through Hudson mask. Good perioperative analgesia also helped in smooth recovery of all except one patient.

We concluded that after rhinoplasty, LMA after extubation avoids the needs of application of face mask, provides adequate ventilation and smooth recovery of patients. This technique may also be useful in other conditions, which require the avoidance of firm application of the face mask (after delicate facial surgery) or endo-tracheal extubation response (such as hypertension, straining, bucking and coughing).

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From the Department of Anesthesia and Intensive Care, Armed Forces Hospital, Riyadh, Kingdom of Saudi Arabia. Address correspondence and reprint requests to Dr. Jamil S. Anwari Consultant Anesthetist, Department of Anesthesia and Intensive Care, Armed Forces Hospital, PO Box 7897, Riyadh 11159, Kingdom of Saudi Arabia. Tel. +966 (1) 4791000 ext. 5288. Bleep 0725. E-mail: janwari@ hotmail.com

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Breathlessness and respiratory failure in myasthenia gravis patient

***Shahid Barlas**, FRCP(Edin), FACG,*
***Brian F. Tregaskis**, MBBS, FRCP(Edin),*
***Aileen Coupe**, MBBS.*

Myasthenia gravis is an autoimmune disorder due to antibody mediated immune attack directed against acetylcholine receptors at neuromuscular junction. Clinically, it presents with muscular weakness and fatigability. In majority of patients initial presentation is due to the involvement of extraocular muscles. With the progression of the disease facial, bulbar, proximal limb muscles, neck extensors and diaphragm get involved.

An 80-year-old lady was admitted twice with history of progressively increasing breathlessness and respiratory failure. On first admission the diagnosis was elusive. During the second admission when she developed 'head drop', only then we realized the exact nature of her illness. Previously, she was in good health and did not experience any muscular weakness or fatigability, suggestive of myasthenia gravis. First admission was with 4 weeks history of progressively increasing breathlessness on exertion. There were no other associated symptoms and she was a nonsmoker. She suffered from mild hypertension which was well controlled with quinapril. Physical examination did not demonstrate any abnormality. Oxygen saturation was 91% on room air. Full blood count, urea, creatinine, electrolytes, electrocardiogram (ECG) and echocardiogram were all normal. Chest x-ray was normal except for small, ill defined shadow at left costophrenic angle. We were unable to point the cause of her breathlessness. She improved symptomatically and was discharged home for follow up in the out patient clinic after pulmonary function testing and high resolution computed tomography (CT) of the chest. Four weeks later, she was readmitted due to worsening of breathlessness. She was now in atrial fibrillation with a ventricular rate of approximately 120 per minute, blood pressure 120/80, respiratory rate 30 per minute,