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).

Received 21st October 2004. Accepted for publication in final form 12th December 2004.

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

References

1. Dubost J, Koflan F, Roullit S, Brenton P, Bansillon V. Emphysèma sous-cutané geant, pnemomédiastin et pneumothrax bilatéral dans les suites d'une rhinpsephoplastie. ***Cah Anesthesiol*** 1986; 34: 161-162.
2. Celebioglu S, Keser A, Ortak T. An unusual complication of rhinoplasty: subcutaneous emphysema. ***Br J Plast Surg*** 1998; 51: 266-267.
3. Dob DP, Shannon CN, Balley PM. Efficacy and safety of the laryngeal mask airway vs Guedel airway following tracheal extubation. ***Can J Anaesth*** 1999; 46: 179-181.
4. Koga K, Asai T, Vaughan RS, Latto IP. Respiratory complications associated with tracheal extubation. Timing of tracheal extubation and use of the laryngeal mask during emergence from anaesthesia. ***Anaesthesia*** 1998; 53: 540-544.
5. Asai T, Shingu K. Use of the laryngeal tube during emergence from anaesthesia in a patient with an unstable neck. ***Anaesthesia*** 2004; 59: 300.

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,

oxygen saturation was 84% on room air and bibasal crepitations, more at right base posteriorly. Electrocardiogram confirmed atrial fibrillation without ischemic changes. Chest x-ray differed little from previous films aside from some consolidation in the right lower zone. High resolution chest CT showed atelectasis at both bases and some pneumonic consolidation of posterior segment of right lower lobe. There was no evidence of interstitial lung disease or pulmonary embolism. Her arterial blood gas was consistent with type II respiratory failure (pH 7.37, pO_2 6.9, pCO_2 9.1, HCO_3 38.6). We considered the possibility of left sided heart failure and pneumonia but were unsure on the cause of her respiratory failure. Despite treatment with digoxin, frusemide and antibiotics there was no improvement. Over the next few days, she had developed a 'head drop' being unable to hold her head upright. At this point it became clear to us that we were dealing with a case of neuromuscular junction disorder. She denied any previous history of diplopia, dysphagia or muscular weakness and fatigability. Tensilon test was positive with dramatic improvement in the power of her neck extensor muscles and oxygen saturation rising from 88-93% on room air. We were unable to check arterial blood gas as she had high international normalized ratio being on warfarin for atrial fibrillation. Auto antibodies screening was negative. Muscle enzymes, serum calcium, magnesium, electrolytes and thyroid function tests were all normal. Anti acetylcholine receptor antibodies assay titer was positive at 222 x 10^ 10M (normal range <5 x 10^ 10M). She responded very well to prednisolone and pyridostigmine and was discharged home. She lived a normal life until recently when she was admitted under surgical services with severe acute pancreatitis and unfortunately succumbed to death.

Myasthenia gravis classically affects young patients in their fourth and fifth decades. Incidence of myasthenia gravis is increasing and this may be explained by the aging population, improvement in prognosis and higher detection rates of patients with milder symptoms.[1] Due to late onset occurrences, myasthenia gravis is still substantially under diagnosed in older people.[2] The majority of patients initially present with ptosis and diplopia and later on develops weakness of proximal limb muscles and respiratory muscles. Approximately 15% of patients have weakness, remained confined to extraocular muscles.[3] Myasthenia gravis has been reported in the elderly patients presenting with falls and bulbar symptoms[4,5] and children presenting with respiratory failure. However, respiratory failure as the first manifestation is unusual and not reported before in the elderly. We wish to recommend that myasthenia gravis should be considered in elderly with breathlessness and respiratory failure of obscure origin.

Received 24th August 2004. Accepted for publication in final form 23rd November 2004.

From the Department of Medicine, Belford Hospital, Fort William, Inverness-Shire, PH33 6BS, United Kingdom. Address correspondence and reprint requests to Dr. Shahid Barlas, Caberfeidh, Spean Bridge, Inverness-Shire, PH33 6BS, United Kingdom. Tel. +44 (1397) 702481. Fax. +44 (1397) 702772. E-mail: shahidbarlas@btopenworld.com

References

1. Aragones JM, Bolibar I, Bonfill X, Bufill E, Mummany A, Alonso F, et al. Myasthenia gravis: a higher than expected incidence in the elderly. ***Neurology*** 2003; 60:1024-1026.
2. Vincent A, Clover L, Buckley C, Grimley Evans J, Rothwell PM. UK Myasthenia Gravis Survey. Evidence of under diagnosis of myasthenia gravis in older people. ***J Neurol Neurosurg Psychiatry*** 2003; 74: 1105-1108.
3. Philips LH 2nd, Tomer JC. Epidemiological evidence of a changing natural history of Myasthenia Gravis. ***Neurology*** 1996; 47: 1233-1238.
4. Saraux-Bonneton A, Raffin B, Vautier F, Laudet J. Myasthenia gravis and elderly persons. A case study. ***Rev Med Interne*** 2001; 22: 984-987.
5. Sharp HR, Degrip A, Mitchell DB, Heller A. Bulbar presentations of myasthenia gravis in the elderly patient. ***J Laryngol Otol*** 2001; 115: 1-3.

Preliminary results of muscle diseases prevalence in patients from Jordan

Younes Abu-Ghalyun*, MSc, PhD,*
Hussam Abu Farsakh*, MD.*

We reported 123 patients affected with a remarkable change in muscle strength and muscle weakness of variable clinical severity. The number of patients seen in all clinics during the months of July 2000 to September 2004 is shown in **Table 1**. The most common disease entities were muscular dystrophies (46 cases, 37.4%) followed by myositis diseases (25 cases, 20.3%) most of them were due to autoimmune diseases. Data demonstrated that motor neuron diseases represented the third common group of muscle diseases in this study (24 cases, 19.5%) whereas, mitochondrial myopathies were the fourth common group of muscle diseases (11 cases, 8.9%). On the other hand, type II atrophy which accounted for the fifth group was 8 cases (6.5%). However, metabolic myopathies (5 cases, 4.1%) included lipid storage diseases (2 cases, 1.6%) and glycogen storage diseases (3 cases, 2.4%). Furthermore, our data showed other rare causes of muscle diseases. Congenital muscular dystrophy (2 cases, 1.6%), end