Correspondence

The incidence of adult respiratory distress syndrome in patients undergoing off-pump coronary artery bypass grafting surgery

EDITOR:

The reported incidence of adult respiratory distress syndrome (ARDS) in patients undergoing open heart surgery with cardiopulmonary bypass (CPB) is 0.4–2.5% and this is associated with a high mortality rate (up to 68.4%) [1–4]. In the present observational study we examined the incidence of ARDS in 1157 adult patients of mean ± SD age 64.8 ± 9.7 yr who underwent off-pump myocardial revascularization during a 3½-yr period (February 2000–August 2004) in Henry Dunant Hospital, Athens, Greece. The majority of patients (86%) were males with a mean preoperative left ventricular ejection fraction 45 ± 9%. Ninety-four per cent of the studied patients underwent elective surgery.

Patients were defined to have ARDS if they had all of the following: (a) severe hypoxaemia, as defined by a PaO₂/FiO₂ ratio <200 mmHg; (b) presence of bilateral lung infiltrates on the chest radiograph; (c) pulmonary arterial wedge pressure of 18 mmHg or no clinical evidence of heart failure or increased left atrial pressure if no wedge pressure measurements were available; (d) absence of chronic obstructive pulmonary disease or other chronic pulmonary disorders and (e) invasive mechanical ventilation [5,6].

The incidence of ARDS during hospitalization was 2/1157 (0.17%). The two patients who developed ARDS were males. One developed this complication during multiple organ failure and septic shock and finally died, while the other developed ARDS during the first postoperative hours with no evident predisposing factors and was ultimately discharged from the intensive care unit (ICU) and hospital in good condition.

In conclusion, we found a lower incidence of ARDS in patients undergoing off-pump myocardial revascularization compared to historical controls of patients undergoing open heart surgery by means of CPB circuit. Our results need to be verified by studies using comparative methodology.

A. Michalopoulos
Intensive Care Unit
Henry Dunant Hospital
Athens, Greece

S. Prapas
Department of Cardiac Surgery
Henry Dunant Hospital
Athens, Greece

M. E. Falagas
Infectious Diseases Clinic
Henry Dunant Hospital
Athens, Greece

References

Lethal adverse reaction during anaesthetic induction

EDITOR:
The risk of an adverse reaction is an inevitable consequence of drug administration. The anaesthetic induction is an unusual period because several drugs are administered over a short time. We present a case of unpredictable adverse drug reaction developing during the anaesthetic induction. The patient suffered profound shock and subsequent death.

A man of 30 yr and weight 73 kg was anaesthetized for an S1 laminectomy. He smoked 2.5 packs of cigarettes a day, he ingested alcohol in a moderate fashion and he was a sporadic consumer of cocaine. The patient had undergone a knee arthroscopy 8 yr before under regional anaesthesia without any complication. Due to his back pain and the motor deficit of his left foot, he was receiving oral dexamethasone 4 mg 6 hourly, diazepam 5 mg 12 hourly, diclofenac 50 mg 8 hourly and tramadol 50 mg 8 hourly respectively. Physical examination, laboratory evaluation, electrocardiograph (ECG) and chest X-ray were normal, so we considered him as ASA II.

On arrival at the operating theatre, the patient was monitored as usual, obtaining these baseline values: non-invasive blood pressure 122/75 mmHg, heart rate (HR) 50 beats min \(^{-1}\) and \(S\text{PO}_2\) (oxygen saturation of haemoglobin by pulse oximetry): 98%. A venous cannula was inserted and lactated Ringer’s solution was administered. After the preoxygenation was accomplished with 100% oxygen for 3 min general anaesthesia was induced with midazolam (2 mg), fentanyl (200 µg), atracurium (5 mg), followed immediately by propofol (plus lidocaine 20 mg) to abolish the eyelash reflex. A further 30 mg of atracurium was administered to facilitate endotracheal intubation. During the anaesthetic induction, coinciding with the propofol administration, the patient developed jerky movements and coughing, that disappeared when a total propofol dose of 200 mg was reached. When assisted ventilation was initiated a supraventricular arrhythmia occurred: wide QRS complexes. He became pale with cyanosis and the \(S\text{PO}_2\) decreased to 80%. Immediately the sevoflurane was discontinued, using only 100% oxygen, and atropine 1 mg was administered. The HR increased to 78 beats min \(^{-1}\), and the recorded blood pressure (BP) was 148/95 mmHg, although the wide ECG complexes remained. Premature ventricular beats began to appear which degenerated to ventricular fibrillation. As the first defibrillation was carried out, electromechanical dissociation was observed. External chest compression was commenced together with epinephrine administration. We attempted to insert an arterial catheter and obtained blood samples in order to investigate a possibility of drug abuse, the presence of an allergic reaction, or to guide drug therapy (the urine samples were obtained previously).

The first laboratory values showed: haemoglobin 9.8 g dL \(^{-1}\); haematocrit 30.2%; white cell count 16 600; platelets 74 000; sodium 136.5 mmol L \(^{-1}\); potassium 3.5 mmol L \(^{-1}\); chloride 117.2 mmol L \(^{-1}\); pH 7.08; \(PCO_2\) 25.4 mmHg; \(PO_2\) 317.5 mmHg; bicarbonate 9.1; base excess \(-20.7\). A sodium bicarbonate infusion was initiated. During the resuscitation efforts, we observed several episodes of isolated ventricular beats without pulse and repeated ventricular fibrillations, but the numerous defibrillations were useless to restore the cardiac output. Despite all attempts we could maintain acceptable BP and \(S\text{PO}_2\) during 1 h of resuscitation. The presence of a tension pneumothorax was discounted by direct observation under fluoroscopy, we also placed an external pacemaker, which was ineffective. After the completion of the arterial and venous femoral lines placement, the resuscitation team evaluated the haemodynamics by invasive technique and the cardiologists put an internal pacemaker through the left femoral vein. This also was ineffective so another pacemaker electrode was inserted through a right internal jugular line but no myocardial depolarization took place (the performance of the generators was guaranteed). At that time, it was possible to achieve an echocardiograph, that demonstrated the absence of mechanical systole after the pacemaker stimulation. The capnography always showed an end-tidal CO\(_2\) oscillating in a range between 10 and 25 mmHg, a fact related to the low flow state. The last haemoglobin and the arterial gas values were similar to those obtained at the beginning of
the cardiovascular collapse. After an unsuccessful resuscitation procedure, which lasted 2 h and 15 min, the patient died.

During the cardiopulmonary resuscitation procedure, we delivered 15 defibrillations, and administered these total doses of the following drugs and fluids: epinephrine (22 mg), isoproterenol (0.4 mg), phenylephrine (1 mg), atropine (3 mg), bicarbonate (320 mEq), hydrocortisone (500 mg), calcium gluconate (4.7 mEq), colloids (hydroxyethyl starch; 1000 mL) and crystalloids (0.9% saline; 3000 mL).

The autopsy revealed several injuries related to a low perfusion state, such as a massive haemorrhage of the gastrointestinal mucosa, a congestive liver with a widespread vacuolation of the hepatocytes and acute tubular necrosis. The study of the heart showed plaques that narrowed the diameter of both coronary arteries by 30–40%; the myocardium, valves and nodes were not damaged. The histopathology demonstrated a scattered necrosis in contraction zone of the myocardial muscle cells and greater number of eosinophils than usual inside the cardiac capillaries. On the other hand, the laboratory analysis showed a serum tryptase concentration of 285 µg L⁻¹ (reference: <13.5 µg L⁻¹) and in regard to the presence of drug abuse, only benzodiazepines were detected. The investigation about specific immunoglobulin E (IgE) to: latex, lidocaine, succinylcholine and egg white was negative. It was not possible to test any other drug used in the induction.

Discussion

We believe that the diagnosis of this unfortunate case, is anaphylaxis due to the fact that the clinical manifestations were unexpected and extremely severe and because the laboratory analysis demonstrated a raised tryptase which indicates the involvement of mast-cell activation [1,2]. The finding of a higher number of eosinophils within the myocardial vessels could be related to the fact that the mast cells and basophils synthesize eosinophilic chemotactic factors to recruit eosinophils to accumulate at sites of allergic reactions [3]. An allergic reaction to latex or to lidocaine have been discarded; several authors have demonstrate that during anaesthesia, the neuromuscular blocking drugs are the most common cause of the anaphylactic reactions with a range of 50–70% [4]. In Catalunya (Spain), a prospective multicentre study showed that the neuromuscular blocking drugs alone or associated with hypnotics were responsible for 34.3% of perioperative allergic reactions [5]. In this way, we tested IgE to succinylcholine in an attempt to detect a cross-reactivity with the atracurium, and the IgE to egg white because it is a component of propofol, but none of them gave any clue about the agent(s) involved in the present case. In addition, the opioid analgesics and benzodiazepines drugs are not exempt from allergic reactions [6].

On the other hand, although the study of the heart showed necrosis of the myocardial muscle cells, a fact that could be related to the resuscitation manoeuvres and an accelerated atherosclerosis, no morphological injuries that can justify the death of the patient were found. Thus, the high level of tryptase, is the only evidence that allows us to establish the diagnosis of anaphylaxis, the most life-threatening form of an adverse reaction.

S. Fernandez-Galinski, S. Pacrev, E. Vela  
Department of Anaesthesiology  
Hospital Universitario del Mar UAB  
Barcelona, Spain

M. A. Munne  
Department of Pathology  
Hospital Universitario del Mar UAB  
Barcelona, Spain

F. Escolano  
Department of Anaesthesiology  
Hospital Universitario del Mar UAB  
Barcelona, Spain

References

Acute bilateral submandibular swelling following surgery in prone position

EDITOR:
Acute swelling of salivary glands is a clinical entity which may affect both the parotid and submandibular glands and may present as a postoperative complication after general or even spinal anaesthesia. Postoperative parotitis has been described after abdominal, orthopaedic and neurological surgery [1–3]. Acute submandibular swelling has been reported in one case after abdominal hysterectomy [4] and in one case following surgery for bilateral optic nerve meningioma [5].

We report the case of a 44-yr old, ASA I patient, admitted to the neurological department for brain tumour surgery. He had neither medical nor surgical history. A few days before admission, he complained to a neurologist of vision problems and cognitive dysfunction. The physical examination was otherwise unremarkable. The brain computerized tomography (CT) scan revealed an intraventricular tumour located in the left occipital region. Treatment consisting in carbamazepine 200 mg twice a day was started 48 h before hospital admission. The patient was premedicated with alprazolam 0.5 mg and atropine 0.5 mg given orally 1 h before surgery. Anaesthesia was induced intravenously with remifentanil infused at 0.5 µg kg⁻¹ min⁻¹ and a bolus of 2 mg kg⁻¹ propofol. After an uneventful intubation facilitated with rocuronium 0.6 mg kg⁻¹, the patient was ventilated with an air/oxygen mixture (F iO₂ 0.5). Anaesthesia was maintained in normocapnic and normothermic conditions with remifentanil infusion (0.25 µg kg⁻¹ min⁻¹) and 1 minimal alveolar concentration (MAC) sevoflurane. The patient was placed in prone position with the head maintained in the Mayfield holder and 15° tilted to the left side. During surgery, the patient received mannitol 20% 200 mL and diuresis was compensated with crystalloids and colloids. At the end of the 5 h procedure, the patient was awaken and extubated in the operating room. At that time, neurological examination did not reveal any neurological deficit. The patient was then transferred to the intensive care unit (ICU). Within the first hours following admission to the ICU, he complained of abundant saliva secretions and developed a bilateral painful swelling of the anterior submandibular region, predominant on the left side. He presented some degree of dysphonia and had difficulties to swallow but could breathe normally. The serum amylase level measured at that time was 189 U L⁻¹ (normal value < 115 U L⁻¹) and the serum C-reactive protein (CRP) value was 1.7 mg dL⁻¹. Ultrasonography and CT scan examinations revealed a bilateral swelling of the submandibular glands predominant on the left side with inflammatory signs, and an enlargement of the salivary channels without evidence of obstruction (Fig. 1). The enlargement extended to the para-laryngeal area on the left side. The parotid glands were normal. Clinical examination performed by an ENT specialist confirmed the diagnosis of anterior submandibular sialoadenitis without evidence of pus at the orifice of the salivary ducts. No culture of salivary secretions was performed. The patient was treated with steroids and non-steroidal anti-inflammatory drugs. The second day after surgery, the serum amylase level was 187 U L⁻¹. The CRP was 9.5 mg dL⁻¹ and was associated with increased white blood cell count (22 000 L⁻¹) and hyperthermia. The patient was then additionally treated with antibiotics (clarithromycin 500 mg i.v. twice a day). On day 3, the swelling of the submandibular region started to decrease. The symptomatology resolved within 6 days except for a mild degree of dysphonia. The patient was discharged from the hospital 1 week after surgery.

Correspondence to: Pol Hans, University Department of Anaesthesia and Intensive Care Medicine, CHR de la Citadelle, 4000 Liege, Belgium. E-mail: pol.hans@chu.ulg.ac.be; Tel: +32 4 225 64 70; Fax: +32 4 225 73 08

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Figure 1.
Correspondence

Inflammation or infection of the salivary glands is an uncommon although already reported complication of surgery [1–5]. This complication usually presents a number of characteristics. It much more frequently affects the parotid than the submandibular glands [1]. It has been mainly reported after major abdominal and orthopaedic surgery [1] but has also been documented after long lasting neurosurgical procedures in patients operated in the sitting position [3]. It occurs within a variable delay after surgery, ranging from the first postoperative hours to 15 weeks [3], with a peak incidence between postoperative days 5 and 7 [6,7]. It may be suppurative or not, and associated or not to sialolithiasis [1,5]. The reported factors that could favour the occurrence of this complication include the position of the head of the patient, a luxation of the temporomandibular joint during intubation, medications such as antihypertensives, antihistamines and antidepressants, dehydration whatever the mechanism, malnutrition and a poor oral hygiene [2,3,5].

When compared to the above-mentioned considerations, the present report refers to a bilateral, predominantly left sided swelling of the anterior submandibular glands without parotitis, that occurred within the first postoperative hours following a 5 h duration neurosurgical procedure in the prone position. Cases reported in the neurosurgical literature developed within a delay of 48–72 h after surgery in patients operated in the semi-sitting position [3]. Those cases mainly affect the parotid gland except for one case of submandibular swelling described after surgery for optic nerve meningioma [5]. Our patient was positioned with the head slightly tilted to the left and the lesion developed predominantly on the left side [3]. In the literature, parotitis that develop in patients whose head is tilted to one side usually affect the gland located on the opposite side. However, one can reasonably assume in this case that the slight rotation of the head during a long duration surgery can be accounted for stretching and dilation of salivary ducts, salivary stasis and development of acute submandibular swelling. Our patient received carbamazepine as the sole preoperative medication and had no sign of preoperative systemic dehydration. He was given 200 mL 20% mannitol for brain relaxation during surgery but was kept normovolaemic with i.v. crystalloids and colloids. As far as no pus was seen at the orifice of the salivary channels, the patient was first treated with anti-inflammatory drugs. Thereafter, he also received clarithromycin 500 mg twice a day because of persisting symptoms associated to an increase in CRP with raised white count. According to literature reports, failure of response to therapy within 48 h is an indication for intravenous antibiotics [3].

In summary, we report an acute bilateral submandibular swelling in a healthy patient after a 5 h neurosurgical procedure in the prone position. This swelling, evidenced by ultrasonography and CT scanning, was responsible for excessive saliva secretion, dysphonia and painful dysphagia. It resolved within 1 week after treatment with anti-inflammatory drugs and antibiotics. This complication quite markedly differs from those usually reported in the literature regarding the main gland affected, the delay after surgery and the potential role of contributing factors.

P. Hans, J. Demoitié
University Department of Anaesthesia
and Intensive Care Medicine
CHR de Citadelle
Liege, Belgium

L. Collignon
Department of Radiology
CHR de Citadelle
Liege, Belgium

V. Bex
Department of Neurosurgery
CHR de Citadelle
Liege, Belgium

V. Bonhomme
University Department of Anaesthesia
and Intensive Care Medicine
CHR de Citadelle
Liege, Belgium

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Bronchospasm caused by neostigmine

EDITOR:
We present a case of a patient who developed acute bronchospasm 10 s after the administration of neostigmine 0.05 mg kg\(^{-1}\) and atropine sulfate 0.015 mg kg\(^{-1}\) intravenously.

A 19-year-old male with known asthma was admitted to the hospital for repair of nasal fracture and nasal septum deviation. He had a history of allergic rhinitis, sinusitis, allergies to penicillin and streptomycin, hay fever and asthma. There was no family history of allergies. The patient denied any history of smoking, alcohol, drugs and food allergy. Monitors in the operating room included non-invasive blood pressure, pulse oximeter, precordial stethoscope and electrocardiogram (ECG). The patient was given 100% oxygen by mask for 10 min. General anaesthesia was induced with ketamine 2 mg kg\(^{-1}\), fentanyl 200 µg and pancuronium bromide 0.1 mg kg\(^{-1}\) intravenously and maintained with halothane 2% in oxygen. A 7.5 mm cuffed oral endotracheal tube wasatraumatically inserted under direct laryngoscopic vision. Anaesthesia was maintained with oxygen, halothane and fentanyl and controlled ventilation. Bilateral breath sounds were normal on auscultation.

At the end of the procedure, neostigmine 0.05 mg kg\(^{-1}\) and atropine sulfate 0.015 mg kg\(^{-1}\) were given intravenously to reverse the muscle relaxant. Ten seconds later the peak inspiratory pressure increased from 20 to 40 cm H\(_2\)O and acute bronchospasm was diagnosed. The patient was treated immediately with halothane 2%, oxygen 100%, ketamine 150 mg and dexamethasone phosphate 8 mg intravenously. An epinephrine infusion was started at 0.02 µg kg\(^{-1}\) min\(^{-1}\) and this was given for 10 min.

Four minutes after the neostigmine and atropine had been given, the patient’s oxygen saturation (\(S_pO_2\)) diminished to 81%, heart rate increased to 137 min\(^{-1}\) (there were no dysrhythmias) and the BP decreased to 75/30 mmHg. He was given ephedrine 10 mg intravenously which resulted in an increase blood pressure to 95/40 mmHg. Ten minutes after the initial episode, the patient’s \(S_pO_2\) had increased to 92% but bilateral wheezy breath sounds were heard on auscultation and he was given aminophylline 5.5 mg kg\(^{-1}\) intravenously over 30 min. The patient was stable and was extubated successfully 45 min after this episode.

Hypersensitivity is unique to an individual and is usually manifest on secondary contact with a particular antigen although it is apparently possible on first contact. In our case it was the first time that the patient had received ketamine, fentanyl, pancuronium, neostigmine and atropine. No test indicating anaphylactic reaction has been performed. We nevertheless believe that intravenous administration of neostigmine 0.05 mg kg\(^{-1}\) and atropine sulfate 0.015 mg kg\(^{-1}\) caused bronchospasm and that this was not an anaphylactic reaction. Other causes of intraoperative bronchospasm include mechanical obstruction of the tracheal tube, inadequate depth of anaesthesia, endobronchial intubation, pulmonary aspiration, pulmonary oedema, pulmonary embolus, pneumothorax and acute asthmatic attack [1]. Pulmonary oedema is unlikely in this patient since he had only received 2000 mL of fluid, had passed 900 mL of urine and a chest radiograph showed no evidence of oedema. The chest radiograph proved the lack of pneumothorax and bilateral breath sounds were heard on auscultation. No pulmonary aspiration had occurred.

Patients with asthma who require general anaesthesia and tracheal intubation are at increased risk for the development of bronchospasm during anaesthesia. Halothane is a potent bronchodilator and has been used during status asthmaticus [2]. Ketamine has been reported to produce bronchodilatation in patients suffering from asthma. Aminophylline has anti-inflammatory, bronchoprotective, and immunomodulatory effects [3] and is widely used for the treatment of acute asthma.

It seems most likely that the bronchospasm was the result of the known parasympathomimetic action of neostigmine and this case underscores the advice that neostigmine should be used with caution in patients with asthma even when given in conjunction with atropine [4].

A. Hazizaj, A. Hatija
Anesthesia and Intensive Care Service
University Hospital Center “Mother Tereza”
Tirana, Albania

References
Supraventricular tachycardia and ST segment depression after intravenous administration of tropisetron

EDITOR:
The 5-HT₃ antagonist tropisetron is widely used for prophylaxis and treatment of postoperative nausea and vomiting (PONV). Adverse events have been reported from other 5-HT₃ antagonists, but are extremely rare. We report a case of supraventricular tachycardia and ST depression in a young patient after a gynaecologic laparoscopic procedure.

A 31-yr-old female (56 kg, 167 cm, ASA II) was scheduled for laparoscopic removal of an ovarian cyst. She was a heavy smoker with symptoms of mild chronic obstructive airways disease. She was premedicated with midazolam 5 mg orally on the general ward 2 h preoperatively. After arrival in the operating room and application of standard monitoring, general anaesthesia was induced with propofol 2 mg kg⁻¹ and fentanyl 200 µg. Endotracheal intubation was facilitated with rocuronium 25 mg. Anaesthesia was maintained with propofol 8 mg kg⁻¹ h⁻¹ and the course was uneventful. After 40 min the surgical procedure was finished and the neuromuscular block had already recovered to a train-of-four (TOF) of 82% and so was not antagonized. The patient was extubated without any problems.

Upon arrival in the postoperative anaesthesia care unit (PACU) the patient was pain free, fully alert, and comfortable. After 1 h on the PACU she complained of an episode of nausea. Tropisetron 5 mg was diluted with 10 mL NaCl and slowly injected intravenously over 3 min. Ten minutes later she complained of anxiousness, shortness of breath and substernal chest pain. The electrocardiogram (ECG) revealed supraventricular tachycardia at 178 beats min⁻¹ and hypertension with blood pressures ranging between 150/90 and 170/95 mmHg. Her oxygen saturation was 96% on room air and respiratory rate was 24–28 min⁻¹. The ECG showed ST segment depression suggesting myocardial ischaemia. Carotid massage proved ineffective and sublingual administration of two doses of nitroglycerin converted the rhythm to sinus tachycardia of 125 beats min⁻¹. Over the next 25 min the heart rate (HR) decreased from 125 to 75 beats min⁻¹ and remained stable thereafter. The ischaemic symptoms (chest pain and ST segment depression) which had lasted for approximately 10 min resolved without further intervention. The serum biochemistry showed normal levels and serial serum creatinine phosphokinase isoenzyme and troponin I levels were normal. A careful follow-up cardiac evaluation on the next day did not reveal any abnormality.

Our patient showed supraventricular tachycardia and ST segment depression shortly after the administration of 5 mg tropisetron. In this otherwise healthy patient, tropisetron might have triggered these cardiac complications. There are large number of factors that can cause postoperative rhythm disturbances, including pain, anxiety, sub-clinical coronary artery disease, electrolyte imbalance, hypoxia and hyperventilation. However, none of these factors was present in our patient as judged by clinical criteria. Therefore the most likely explanation for the dysrhythmia and the myocardial ischaemic episode would seem to be an effect of tropisetron. This, to the best of our knowledge has not been reported before. Although both Baguley and colleagues [1] and Bosek and colleagues [2] reported three observations with similar symptoms after the administration of ondansetron.

Serotonin stimulates 5-HT₃ and 5-HT₄ receptors in the brain and the cardiovascular system, and causes nausea and vomiting. Additionally, it modulates the activated Bezold–Jarisch reflex evoked by bradycardia due to alteration of sympathetic activity. This reflex can be elicited through chemical or mechanical stimulation of vagal afferents of the cardio-pulmonary system and causes bradycardia and hypotension. Recently, Bosek postulated that 5-HT₃ antagonists suppress the von Bezold–Jarisch reflex, which can lead to tachycardia [2]. Blockade of 5-HT receptors is found to decrease neurotransmission in the presynaptic terminals of the autonomic nervous system [3]. In return, bradycardia can cause cardiac stimulation via 5-HT₃ receptors.

Correspondence to: Gottfried Mitterschiffthaler, Department of Anaesthesiology and General Intensive Care, University Hospital, Anichstrasse 35, A-6020 Innsbruck, Austria. E-mail: gottfried.mitterschiffthaler@uibk.ac.at

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leading to tachycardia and hypertension. Saxema reported that this complex pattern of bradycardia and tachycardia can even lead to coronary vasodilation or vasoconstriction [4,5].

We believe that our patient’s short episode of supraventricular tachycardia and ST segment depression was caused by inhibition of the Bezold–Jarisch reflex by suppression of the 5-HT₃ cardiac receptors.

G. Mitterschiffthaler, G. Putz
Department of Anaesthesiology and General Intensive Care
Medical University of Innsbruck
Innsbruck, Austria

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