hyperalgesia. Furthermore, they suggested that remifentanil in association with ketamine was useful in patients pretreated with opioids.

One possibility for this patient’s pain in the postoperative period – and in our view the most likely one – however is not discussed, namely opioid-withdrawal hyperalgesia. Postoperative analgesia in patients who receive opioids for chronic pain is undoubtedly a challenge for anaesthesiologists and pain therapists. In clinical practice, chronic pain patients receiving strong opioids preoperatively show high inter-individual variability and sometimes extremely high postoperative demand for opioids. Depending on the chronic opioid dose, total cumulative doses of 30–45 mg piritramide within the first 1–2 h are common in these patients. These observations are in accordance with Rapp and colleagues [2], who found a more than three-fold (135.8 vs. 42.8 mg) increase in opioid demand in the first 24 h after surgery in patients with preoperative opioid consumption as compared with opioid-naïve patients.

In addition, we feel that postoperative analgesia is much more difficult to handle in chronic pain patients receiving remifentanil as sole opioid intraoperatively as compared with those receiving long-acting μ-opioid agonists like fentanyl or sufentanil. Irrespective of the fact whether this patient’s fentanyl patch was removed before surgery or not, we feel that the administration of 10 mg piritramide and 4 mg morphine for postoperative analgesia in this patient was simply not enough to provide sufficient analgesia.

It has been shown that enhanced pain sensations after cessation of a remifentanil infusion are due to an acute withdrawal response, which cannot be modulated by N-methyl-D-aspartic acid receptor antagonists [3,4]. Therefore, we think that the therapeutic effect of ketamine observed by Dumont and colleagues is most likely due to its direct analgesic or hypnotic effect and not based on the reversal of pronociceptive mechanisms induced by remifentanil. The increasing evidence for opioid-induced hyperalgesia should not lead to a restricted use of opioids in the perioperative period, especially not in patients who have a history of chronic opioid administration.

A. Z. Tzabazis, W. Koppert
Anaesthesiologische Klinik
Universitätsklinikum Erlangen
Germany

References
Conditions involving release of pro-inflammatory cytokines predispose to ARDS
doi: 10.1017/S026502150700049X

EDITOR:
Sadis and colleagues [1] investigated risk factors for the development of ARDS in patients receiving multiple transfusions and found that it was not the number of transfusions but thoracic trauma and hypoxia that were associated with the subsequent development of ARDS. Patients who developed ARDS received significantly more fresh frozen plasma. Previous studies showed that septicaemia is an additional predisposing factor for transfusion-related ARDS [2]. Another condition commonly associated with pulmonary oedema during infusion of large amounts of intravenous fluids is diabetic ketoacidosis [3]. All these conditions with their different pathophysiology have in common the release of large amounts of cytokines including tumour necrosis factor (TNF) and interleukin-1 (IL-1). Transfusion of an anti-CD28 monoclonal antibody into human volunteers stimulated T-cells to release large amounts of these two cytokines and led to pulmonary oedema in all subjects of this trial [4]. The mechanism by which these cytokines lead to or predispose to pulmonary oedema has recently been clarified: Alveolar epithelial fluid clearance in pulmonary oedema is dependent on pulmonary epithelial sodium and chloride transport through the apical alveolar epithelial sodium channel and the cystic fibrosis transmembrane conductance...