

Currently, this treatment option is only available in a small number of centres worldwide and only at our unit in the UK. Increasing interest suggests that in future more anaesthetists may be asked to provide anaesthesia for patients receiving intralesional bleomycin treatment.

Bleomycin chemotherapy is a recognized cause of pulmonary pathology.³ The risk of developing bleomycin-related pulmonary injury is known to be increased by alveolar hyperoxia even in cases where there has been an interval of 6–12 months between bleomycin exposure and hyperoxic anaesthesia.⁴ Animal studies have shown that the risk of developing bleomycin-related pulmonary pathology is increased if bleomycin is administered concurrently with the alveolar hyperoxia.^{5,6} This has implications for the anaesthetic management of our patients where, despite a lower dose of bleomycin required than that used in chemotherapy, the drug is administered during general anaesthesia.

In our unit, we use the following protocol. Before admission, adult patients are referred to a respiratory physician for assessment by history, examination, baseline spirometry, transfer factor (DLCO), and a chest radiograph. Children are assessed by a paediatrician with a special interest in respiratory disease; baseline respiratory function tests being obtained where possible. Patients are reviewed by the respiratory team midway through a course of treatments and after completion of treatment.

The aim is to provide safe anaesthesia while avoiding alveolar hyperoxia. Pre-oxygenation is avoided and supplemental oxygen is restricted, aiming for a normal end-tidal oxygen concentration and a target minimum Sa_{O_2} of 94%. Ventilation is assisted to prevent hypoxaemia resulting from alveolar hypoventilation. Most cases can be managed with a laryngeal mask airway or oropharyngeal airway, thus avoiding any desaturation associated with extubation. The procedure is performed in the anaesthetic room to avoid hypoxic events upon transfer into theatre. If difficulties arise, the anaesthetist is encouraged to use oxygen supplementation as necessary until problems are resolved. Before transfer to recovery, the patient should have resumed satisfactory spontaneous respiration on air. During recovery, no supplemental oxygen is prescribed unless the oxygen saturation decreases below 94% and, if required, the lowest effective supplementation is used.

We have reviewed our first 3 yr experience of providing general anaesthesia for these patients. Forty-nine patients received a total of 187 general anaesthetics. Nineteen (42.9%) of the patients were children, with seven (14.3%) under 1 yr old. The majority (65.3%) of procedures involved lesions on the face; a further 12.2% involved the head and neck. The median $F_{I_{O_2}}$ during treatment was 0.21. About 66.5% of patients had an $F_{I_{O_2}}$ of 0.25 or less, 86.9% had an $F_{I_{O_2}}$ of 0.3 or less, and 91.7% of patients had an $F_{I_{O_2}}$ of 0.35 or below (Table 1). There were only two (1.1%) procedures where oxygen saturation levels below 90% were recorded. Thirty-three (17.6%)

Table 1 $F_{I_{O_2}}$ administered during procedure ($n = 167$)

$F_{I_{O_2}}$	<i>n</i>	%
0.21–0.25	111	66.5
0.26–0.30	34	20.4
0.31–0.35	8	4.8
0.36–0.40	3	1.8
0.41–0.45	2	1.2
0.46–0.50	8	4.8
>0.51	1	0.6

procedures required the patient to receive oxygen in recovery. There were no cases of bleomycin-related pulmonary disease. Two patients reported bleomycin-related skin reactions.

With careful respiratory assessment and monitoring, and using an anaesthetic technique that attempts to avoid alveolar hyperoxia, intralesional bleomycin therapy of vascular malformations under general anaesthesia has not been associated with the development of pulmonary complications in patients treated at our unit.

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Target controlled infusion of opioids for bariatric surgery and morphine loading dose

Editor—I read with interest the useful study by De Baerdemaeker and colleagues,¹ but wish to raise some concerns about the paper. First, the authors did not declare

their sample size calculation and how these 40 patients were selected. Secondly, I am concerned that morphine was not given i.v. in a timely fashion to aid analgesia in post-anaesthesia care unit (PACU), as morphine peak effect is relatively late, and I believe delaying giving these patients (especially the remifentanil group) morphine is unethical. Thirdly, in our hospital, we do not routinely prescribe i.v. patient-controlled analgesia (PCIA) to laparoscopic banding patients after operation, and most patients do well and leave hospital after their gastrographin swallow test next morning on simple oral analgesia.

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Editor—We thank Dr Al-Tamimi for his comments and his interest in our paper and for the opportunity to reply to him. We will address his points in turn. First, details of the sample size calculation and patient selection can be found in our Methods section. Since the primary endpoint of the study was the postoperative morphine consumption, power analysis was based on a similar protocol performed in non-obese patients.² Group allocation was at random with blinded envelopes.

Secondly, we would like to point out that the patients in both study groups received acetaminophen 2 g i.v. and diclofenac 150 mg i.v. at the beginning of surgery. Thus, the patients in the remifentanil group received non-opioid transitional postoperative analgesia. The initial mean (SD) visual analogue scale (VAS) score in the remifentanil group on admission in the PACU was 4.3 (1.7) and this reflects suboptimal analgesia, but to our opinion not in the range of unethical clinical practice. In non-obese patients, most clinicians will use an intraoperative i.v. morphine dose of 0.1–0.25 mg kg⁻¹ administered 30–60 min before the end of surgery for the immediate postoperative analgesia after a remifentanil-based anaesthesia. However, there are no clear guidelines in the literature on the safe use of an intraoperative loading dose of morphine based on ideal body weight (IBW) in morbidly obese patients. When studying the influence of diurnal variation and morbid obesity on the morphine requirements using PCIA, Graves and colleagues³ found that morphine dosing rate (mg kg⁻¹ h⁻¹) normalized to IBW was a better predictor of analgesic requirements. More recent studies on the use of PCIA with morphine in morbidly obese patients have demonstrated its safety^{4,5} even in obese patients with obstructive sleep apnoea syndrome (OSAS).⁶ All authors used i.v. morphine in the PACU titrated individually in divided doses of 2.5–5 mg with 10 min intervals to achieve an acceptable pain score before instituting their PCA device. Ahmad and colleagues⁶ used a prudent intraoperative loading dose of 50 µg kg⁻¹ IBW morphine at the end of

the pneumoperitoneum. In our study, performed on morbidly obese patients without OSAS or serious cardiopulmonary disease, the PCIA device delivered a morphine loading dose (0.15 mg kg⁻¹ IBW) at the moment of first analgesic request. Our postoperative data on arterial blood gas analysis and spirometry show the safety of this strategy in this type of obese patients. Nevertheless, it took 2 h to register VAS pain scores below three in both study groups. Until we see prospective studies on the accurate timing and safety of an intraoperative loading dose of morphine for morbidly obese patients in the range of 0.15 mg kg⁻¹ IBW, the PCIA recommendations of Levin and colleagues⁷ still apply: no basal infusion of morphine, bolus doses of 0.5–1.0 mg with a 10 min interval, and titration to a desirable effect within the first few hours after surgery. Finally, we agree with Dr Al-Tamimi that PCIA morphine is not a routinely used postoperative analgesic regimen for laparoscopic gastric banding. For study purposes, morphine consumption was used as an objective and quantitative measure of postoperative pain. We are convinced that with simple oral analgesics (and infiltration with local anaesthetics), satisfactory postoperative analgesia can be achieved.

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