LUNG FUNCTION AFTER VECURONIUM PRETREATMENT IN YOUNG, HEALTHY PATIENTS

R. P. MAHAJAN AND J. LAVERTY

SUMMARY
Lung function and clinical evidence of muscle weakness were assessed in 12 ASA I patients who received vecuronium 0.01 mg kg\(^{-1}\) pretreatment as a part of their anaesthetic management, before and 3 min after pretreatment. Most patients demonstrated ptosis and diplopia, while five of the 12 were unable to raise the head for > 4 s and had difficulty in swallowing. Significant reductions occurred in forced vital capacity, forced expiratory volume in 1 s, and maximum mid-expiratory flow rate. Among static lung volumes, functional residual capacity and expiratory reserve volume decreased significantly. However, these changes were not serious enough to cause clinically significant impairment of coughing or a decrease in oxygen saturation in any patient.

KEY WORDS

Pretreatment with a non-depolarizing neuromuscular blocking drug is widely recommended either to prevent side effects of the depolarizing neuromuscular blocker, suxamethonium, or to hasten the onset of action of a subsequently paralysing dose of a non-depolarizing blocking agent [1]. This practice involves injecting a small dose of blocker 3-5 min before induction of anaesthesia. Patients, being awake during this period, often experience diplopia, ptosis, weakness in the limbs, restlessness, a sensation of suffocation and difficulty in breathing [2]; inhalation of gastric contents has also been reported [3]. The present study was conducted to assess lung function changes after pretreatment with vecuronium.

METHOD AND RESULTS
The study was approved by the Ethics Committee of the hospital. We studied 12 ASA I patients (two male) undergoing routine dental surgery and receiving vecuronium 0.01 mg kg\(^{-1}\) pretreatment as a part of their anaesthetic management. Patients with cardiorespiratory, neurological or muscular diseases were excluded. A history of anaesthetic problems in the past, regurgitation or hiatus hernia, were also criteria for exclusion. Informed consent was obtained from all patients, who were aged 21-35 yr (mean 25.8 yr), heights 1.50-1.78 m (mean 1.68 m) and weights 51-82 kg (mean 63.4 kg). Premedication comprised temazepam 10 mg and metoclopramide 10 mg, 45-60 min before surgery.

The investigation was carried out in the anaesthetic room with the patient lying supine on a tilting trolley. The ECG and arterial oxygen saturation (by pulse oximetry) were displayed continuously. Arterial pressure was measured non-invasively at regular 3-min intervals throughout the investigation. An i.v. infusion was set up and, after a resting period of 5 min, lung function was assessed (see below). After a further 3-min rest period, vecuronium 0.01 mg kg\(^{-1}\) was administered i.v. Respiratory function tests were performed again 3 min after injection of vecuronium. In addition, other evidence of muscle weakness (ptosis, diplopia, inability to raise the head for > 4 s, difficulty in swallowing and difficulty in protruding the tongue) were noted. Anaesthesia was induced immediately after completion of the second set of measurements.

The following ventilatory variables were studied using a Morgan transfer test machine, calibrated before studying each patient: forced expiratory volume in 1 s (FEV\(_{1}\)), forced vital capacity (FVC) and maximum mid-expiratory flow rate (MMEF) were measured from the FVC manoeuvre. Functional residual capacity (FRC) was measured by the helium dilution technique. Other lung volumes and capacities (inspiratory capacity (IC), expiratory reserve volume (ERV), residual volume (RV), vital capacity (VC) and total lung capacity (TLC)) were derived from a recorded spirogram and FRC measurement. Three sets of FVC manoeuvre and spirometry were obtained and the best reading was selected for analysis. The results were analysed using the Wilcoxon signed-rank test. A value of \(P < 0.05\) was considered significant.

All the patients developed ptosis, 11 had diplopia and five of the 12 were unable to swallow or to lift the head for > 4 s, demonstrating a significant muscular weakness. Data from one patient could not be included in the analysis of ventilatory variables as she was unable to produce an effective seal around the mouthpiece of the lung function test machine after receiving vecuronium pretreatment. Dynamic
 spirometry revealed decreases of 7–10% in FEV1, FVC and MMEF (P < 0.05). Among static lung volumes, the most prominent change was in ERV, which decreased by 25% and contributed to the significant reductions in FRC and VC. Residual volume, TLC and IC remained unchanged (table I). There was no change in oxygen saturation and none of the patients had an SpO2 less than 95% at any time during the investigation. Changes in heart rate and arterial pressure were insignificant.

### COMMENT

In the past, only a few studies have attempted to measure lung function after pretreatment with non-depolarizing neuromuscular blocking drugs. Rao and Jacobs [4] demonstrated a significant decrease in forced inspiratory and expiratory flow rates while peak expiratory flow rate (PEFR) remained unchanged after pancuronium 0.014 mg kg⁻¹. Other workers [5] reported significant reductions in PEFR while VC, FEV1, inspiratory force and ventilatory frequency remained unchanged after 0.01 mg kg⁻¹ of either vecuronium or pancuronium, or 0.015 mg kg⁻¹ of pancuronium. These studies used ventilatory measurements which are mainly effort-dependent, which is probably the reason for the contradictory results. De Troyer and Bastenier-Geens [6] investigated the effect of submaximal neuromuscular block on the lung mechanics in sitting, awake volunteers and demonstrated significant reduction in IC, VC, ERV, FRC and TLC. The submaximal neuromuscular block was achieved by infusing pancuronium 1.6–1.8 mg, which is twice the dose used for pretreatment. The effect of myoneural blockers in pretreatment doses, on effort-independent ventilatory variables (FRC) and other lung volumes (IC, RV, TLC), has not been described before.

We chose pretreatment with vecuronium 0.01 mg kg⁻¹ (the same dose recommended by many workers) as being effective and acceptable for clinical practice. None of our patients became distressed after pretreatment, but all were informed of possible muscle weakness. We found significant reductions in FVC, FEV1, and MMEF. An important finding of this study was the reduction in ERV, which was responsible for significant changes in FRC. These findings are in agreement with the observations made by De Troyer and Bastenier-Geens [6]. However, unlike their study, we could not demonstrate changes in IC or TLC. This is probably because of the smaller dose of neuromuscular blocker used in our study. We have not investigated the mechanism for reduction in ERV and FRC in our study, but decreased outward recoil of the chest wall in the lower part of the vital capacity, as described earlier [6], may be an explanation. Slight weakness of the diaphragm, causing proximal shift of its end-expiratory position, may be another possibility. We are unable to comment on the effect of reduction in FRC on shunt as we did not measure PaO2 in our patients, but the effect was not sufficient to cause significant changes in SpO2. Five of the 12 patients in our study experienced difficulty in swallowing, indicating a significant impairment of muscles involved in maintaining the integrity of the upper airway. Therefore, pretreatment with a neuromuscular blocking drug in a patient at risk of regurgitation may not be safe.

### REFERENCES


