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Comments

Our results data show that PGE_1 attenuated 5HT-induced bronchoconstriction. Similarly, we have previously observed that PGE_1 also antagonized histamine-induced bronchoconstriction.[2] As PGE_1 is a an EP_2 receptor agonist, adenylate cyclase is activated to increase intracellular cAMP concentrations which produces airway smooth muscle relaxation.[1] Therefore, increased intracellular cAMP may contribute to the observed spasmolytic effects.

In the present study, plasma epinephrine slightly but significantly increased after administration of PGE_1 10 μg kg^{-1}. This suggests that PGE_1-induced systemic vasodilation increases sympathetic activity although PGE_1 attenuates arterial baroreceptor reflexes.[8] As circulating catecholamine concentration is one of the most important factors controlling airway tone,[9] catecholamine release may also be involved in the observed bronchodilation. However, as PGE_1 0.1 and 1.0 μg kg^{-1} produced significant bronchodilation without increases in plasma catecholamines, PGE_1 may have direct bronchodilatory effects.

PGE_1 has been used clinically for the treatment of pulmonary hypertension.[3] Fullerton and colleagues[10] have also shown a direct relaxant effect of PGE_1 on isolated rat pulmonary artery rings. In the present study, PGE_1 10 μg kg^{-1} significantly attenuated pulmonary hypertension although at $\leqslant$1.0 μg kg^{-1} PGE_1 was ineffective. However, as the catabolism of PGE_1 in the lungs of dogs is about six times that in humans,[2] clinically relevant doses may not attenuate pulmonary hypertension. Consistent with this, several reports[5] suggest that PGE_1 does not attenuate pulmonary hypertension by pulmonary vasodilation.

In conclusion, the present study indicates that clinically relevant doses of PGE_1 may produce direct bronchodilation, but not pulmonary vasodilation.

Acknowledgement

This work was supported, in part, by a grant-in-aid from Ono Pharmaceutical Company, Japan.

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British Journal of Anaesthesia **85 (3)**: 462–5 (2000)

Influence of airway-occluding instruments on airway pressure during jet ventilation for rigid bronchoscopy

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We measured changes in airway pressure (P_{aw}) caused by microsurgical instruments introduced into a rigid bronchoscope during high frequency jet ventilation (HFJV). With approval of the

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institutional Ethics Committee, 10 adults undergoing elective tracheobronchial endoscopy and endosonography during general anaesthesia were investigated. Inflation of an endosonography probe balloon in the left main stem bronchus caused airway obstruction. Pressure measurements proximal and distal to the obstruction were compared after three degrees of obstruction (0%, 50% and 90%) and with two different driving pressure settings. Airway obstruction increased the mean (SD) peak inspiratory pressure (PIP) from 7.5 (2.6) to 9.5 (3.5) mm Hg for 2 atm (P=0.0008) and from 9.7 (3.7) to 13.0 (5.1) mm Hg for 3 atm (P=0.0001). Airway obstruction did not alter peripheral PIP (7.2 (4.1) to 7.1 (3.7) mm Hg for 2 atm and 8.8 (4.3) to 9.4 (5.2) mm for 3 atm), but resulted in an end-expiratory pressure (EEP) beyond the narrowing being significantly greater than in the unobstructed airway (2.5 (3.4) to 5.5 (3.7) mm Hg for 2 atm; P=0.0005) and 3.2 (3.6) to 8.0 (4.3) mm for 3 atm; P<0.0001). Severe airway narrowing increases inspiratory pressure proximal and expiratory pressure distal to the obstruction in relation to the applied driving pressure. Since the distal EEP never exceeded PIP, even near-total airway obstruction should not cause severe lung distension or barotrauma in subjects with normal lungs.

Br J Anaesth 2000; **85**: 463–5

Keywords: anaesthetic techniques, bronchoscopy; high frequency ventilation; airway, obstruction, airway, pressure

Accepted for publication: March 21, 2000

High frequency jet ventilation (HFJV) for rigid bronchoscopy is a convenient method that gives optimal visibility and easy access of diagnostic and surgical instruments to the airway, mainly because HFJV does not require a sealed airway. In constrast, the rigid bronchoscope must be kept open to the surrounding atmosphere to allow passive exhalation of the insufflated gas. Unless the egress of gas is impaired, inadvertent airway pressure (P_{aw}) elevation and lung distension are unlikely. However, there is a risk of airway obstruction when instruments are introduced into the bronchoscope. A newly developed probe for tracheobronchial endosonography, recently introduced for scanning of mediastinal structures, to assess lung tumours and affected lymph nodes, is such an instrument.[1 2] The tip of this probe has an expandable balloon which is filled with water as a transmission medium. To obtain the image, the balloon is expanded until sufficient contact is achieved between probe and airway wall. During inflation of the balloon, the airway is progressively obstructed; there is a risk that air will be trapped in the lung supplied by the obstructed airway.[3 4] The purpose of this study was to assess proximal and distal P_{aw} during progressive airway occlusion caused by intermittent inflation of the endosonography probe.

Methods and Results

Following approval by the institutional ethical committee and informed consent, 10 adults undergoing elective diagnostic or interventional tracheobronchial endoscopy and endosonography under general anaesthesia were enrolled. All patients received total intravenous anaesthesia with propofol, remifentanil and succinylcholine. HFJV was applied with an AMS 1000 jet ventilator (Acutronic Medical Instruments, Hirzel, Switzerland) via the jet port at the proximal end of a rigid bronchoscope (Karl Storz, Tuttlingen, Germany). Monitoring consisted of electrocardiography, non-invasive measurement of arterial pressure and pulse oximetry.

After insertion of the rigid bronchoscope, the endosonography probe was placed in the main left bronchus, where the artificial airway obstruction was to be created. Proximal and distal P_{aw} were measured with sensors derived from standard intravascular pressure measurement equipment, consisting of transducers and water-filled tubing. The proximal P_{aw} sensor was located in the tip of a steel tube with an internal diameter of 1.0 mm, which was introduced into the light guide channel of the bronchoscope. The distal P_{aw} was measured through a Cavafix Certo 358 catheter (B. Braun, Melsungen, Germany), internal diameter of 1.1 mm, which was placed with its tip into the left mainstem bronchus 10 cm distal to the tip of the bronchoscope. The validity of intratracheal P_{aw} measurement as an approximation for alveolar pressure was shown in previous investigations.[5] Both transducers were placed at the xiphoid level.

Airway obstruction was created by gradually filling the balloon of the endosonography probe, so that obstruction increased steadily from 0% to 100% over an 8 s period (Figure 1). P_{aw} was recorded throughout the balloon expansion. Three specific levels of obstruction, 0%, 50%

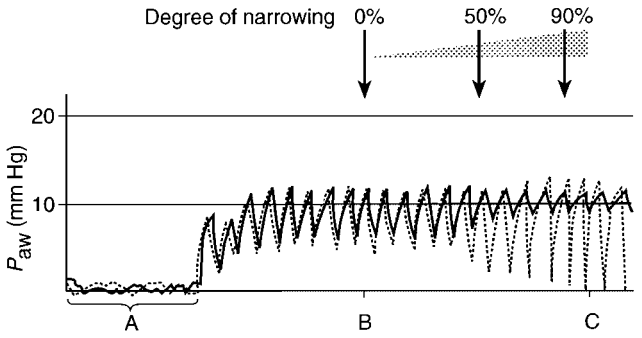

Fig 1 Proximal (dotted line) and distal (solid line) airway pressure (P_{aw}) in patient no. 6 during increasing obstruction of the airway: Period A: apnoea during zeroing of both pressure sensors; period B: beginning of expansion of endosonography probe balloon; period C: complete occlusion of the airway.

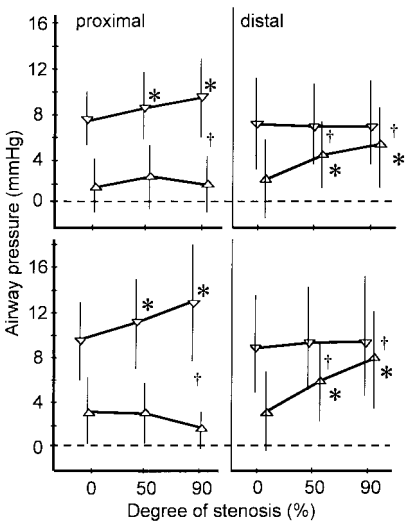

Fig 2 Proximal and distal P_{aw} during 0%, 50% and 90% obstruction of the airway and a driving pressure of 2.0 atm (upper segment) and 3.0 atm (lower segment). Values are expressed as mean±SD; *Significantly different from P_{aw} without obstruction (P<0.025), †Pressure amplitude significantly different from that without obstruction (P<0.025).

Table 1 Biometrical and clinical data (mean (SD)). VC=vital capacity; Fe_{V_1}=forced expiratory volume in 1 s

Number of patients	10
Gender (females/males)	1/9
Age, yr (range)	66 (47–75)
Height, cm	172 (5)
Weight, kg	77 (9)
VC, litres	3.6 (0.9)
Fe_{V_1}, litres	2.5 (0.8)
Fe_{V_1} (% of VC)	68 (10)

and 90%, were evaluated further. The measurements were performed under two different driving pressure (DP) settings (2.0 and 3.0 atm); all other variables, such as FI_{O_2} (1.0), inspiration duration (50%) and frequency (100 cpm) remained unchanged. From the continuous P_{aw} tracings we measured peak inspiratory pressure (PIP) and end-expiratory pressure (EEP).

Changes of P_{aw} during progressive airway obstruction were analysed using nonparametric analysis of variance for repeated measurements (Friedman test) followed by paired Wilcoxon's signed rank tests with Bonferroni correction. Proximal and distal P_{aw} values without obstruction were compared using Wilcoxon's signed rank test. The level of significance was set at P=0.05. Continuous data are presented as mean (SD).

All patients had a pulse oximeter saturation of ⩾90% throughout the procedure. No lung injury or surgical emphysema was noted up to 4 h after the intervention. Biometric and clinical data of the patients are summarized in Table 1.

During jet ventilation with a DP of 2.0 atm (Figure 2), gradual obstruction of the left mainstem bronchus resulted in a progressive increase of proximal PIP from 7.5 (2.6) to 9.5 (3.5) mm Hg (P=0.0008). In contrast, proximal EEP remained unaffected by airway obstruction (1.8 (2.6) and 1.9 (2.5) mm Hg). Without airway obstruction, there was no difference between distal and proximal PIP, or between distal and proximal EEP. Gradual airway narrowing did not affect distal PIP, but caused a highly significantly increase of distal EEP compared with the unobstructed airway lumen, from 2.5 (3.4) to 5.4 (3.6) mm Hg (P=0.0005). With a DP of 3.0 atm, these effects on P_{aw} were more pronounced: proximal PIP increased from 9.7 (3.7) to 13.0 (5.1) mm Hg (P=0.0001), proximal EEP decreased from 3.2 (2.9) to 1.8 (1.8) mm Hg (P=0.0001), distal PIP did not change and distal EEP increased significantly from 3.2 (3.6) to 8.0 (4.3) mm Hg (P<0.0001).

Comment

HFJV is a convenient method of ventilation for rigid bronchoscopy, but P_{aw} must be monitored continuously, especially if additional instruments are inserted into the bronchoscope. These devices may cause some airway obstruction: the recently introduced endosonography probe causes near complete airway obstruction.[4] We have shown that both PIP and EEP were affected by the degree of airway obstruction and by the applied DP: proximal PIP and distal EEP increased significantly, while proximal EEP and distal PIP remained unaffected. P_{aw} alterations were more pronounced with a DP of 3.0 atm than at 2.0 atm. Air trapping became significant when the obstruction occupied 50% of the original airway cross-sectional area. This corresponds to findings of Ayuso and colleagues, who found sufficient gas exchange with <50% of the cross-sectional area of the airway obstucted.[6]

The relevance of these findings for the routine application of HFJV during rigid bronchoscopy depends on factors such as jet flow, airway dimensions and degree of obstruction, and on the interaction between these factors. The increase in distal EEP during progressive airway obstruction may not necessarily be dangerous, since it was always less than the distal PIP observed without airway obstruction. The slight increase in proximal PIP is not likely to damage the lungs.

In summary, short-term near-total occlusion of the airway during jet ventilation beyond the tip of the rigid bronchoscope may slightly increase proximal peak P_{aw} and increase distal EEP. These effects depended on the degree of obstruction and the DP, but were never high enough to cause overinflation of the lung or even barotrauma. Nevertheless, careful and continuous monitoring of airway pressure (as used with modern jet ventilation equipment) and observation of the thoracic excursions during jet ventilation are indispensable during endoscopic instrumentation.

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British Journal of Anaesthesia **85 (3)**: 465–7 (2000)

Transcranial magnetic-evoked potentials under total intravenous anaesthesia and nitrous oxide

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Magnetic stimulation of the cortex and recording of the motor-evoked potentials (MEPs) by electromyography (EMG) is a well proven method to assess the descending pathways of the spinal cord and detect neurological impairment. We have assessed, in 33 adult patients undergoing spinal surgery, the influence of four total i.v. anaesthesia regimens (TIVA) on this recording technique. In 20 patients, the effect of 50% nitrous oxide was also studied. MEP amplitudes, latencies and success rates of stimulation were obtained in the steady-state after induction of anaesthesia. Combinations of midazolam and ketamine, and alfentanil and etomidate had the least effect on MEPs. Propofol (in combination with alfentanil or ketamine) showed marked depression of the MEP amplitude and the lowest success rates of stimulation. The latencies did not change at all. The addition of nitrous oxide significantly depressed the registered MEPs and lowered the success rates.

Br J Anaesth 2000; **85**: 465–7

Keywords: brain, electromyography; brain, evoked potentials; brain, cortex; anaesthesia, i.v.; anaesthetic gases, nitrous oxide

Accepted for publication: April 17, 2000

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