

EFFECTIVENESS OF PREOXYGENATION IN MORBIDLY OBESE PATIENTS

M. C. BERTHOUD, J. E. PEACOCK AND C. S. REILLY

SUMMARY

The time taken for the oxygen saturation (Sp_{O_2}) to decrease to 90% after preoxygenation was studied in six morbidly obese patients and six matched controls of normal weight. During apnoea the obese patients maintained $Sp_{O_2} > 90\%$ for 196 (SD 80) s (range 55–208 s), compared with 595 (SD 142) s (range 430–825 s) in the control group ($P < 0.001$). One patient in the obese group had desaturation before the onset of complete relaxation and tracheal intubation, without complications. Bedside lung function tests were not significantly different between groups and cannot be used as a predictor of the effectiveness of preoxygenation.

KEY WORDS

Anaesthetic techniques: preoxygenation. Complications: obesity.

Preoxygenation decreases the risk of hypoxia associated with induction of anaesthesia and intubation of the trachea. It is valuable also in patients whose respiratory or cardiac reserve is reduced, and when a delay in establishing a clear airway may be anticipated.

Patients with morbid obesity—that is, a body mass index (BMI) $> 35 \text{ kg m}^{-2}$ —have a greater risk of acid regurgitation and aspiration, a reduced respiratory and cardiac reserve and are associated with increased difficulty at tracheal intubation. It has been recommended that a rapid sequence induction of anaesthesia should be used for these patients [1]. We have examined the effectiveness of preoxygenation in morbidly obese patients.

METHODS AND RESULTS

After local Ethics Committee approval and written informed consent by the patients, we studied 12 patients undergoing elective abdominal sur-

gery. Six morbidly obese patients (group 1) (mean weight 123.5 (SD 23.2) kg (range 104–153 kg) and BMI 49 (7.3) kg m^{-2} (range 42–58.4 kg m^{-2})), were matched for sex, age and height with six control subjects (group 2) (weight 63 (8.3) kg (range 52–76 kg) and BMI 23.1 (3.0) kg m^{-2} (range 18–26.3 kg m^{-2})). BMI and weight were significantly greater in the obese group (Student's t test, $P < 0.001$). Full preoperative assessment was performed on the ward on the day before surgery. One patient in each group was a smoker. All patients were assessed for difficulty in tracheal intubation using established scoring systems [2, 3]. No patient was excluded from the study because of anticipated difficulty. Forced vital capacity (FVC), forced expiratory volume in 1 min (FEV_1) and peak expiratory flow rate (PEFR), were performed before operation with the patient in the sitting and in the supine position.

Results are presented as percentage of predicted for height and age. FVC in group 1 was 85.0 (20.4)% (range 54–115%) in the sitting position and 85.3 (14.3)% (range 68–110%) supine; in group 2 it was 97.3 (8.9)% (range 87–110%) sitting and 96.0 (10.0)% (range 85–108%) supine. FVC_1 in group 1 was 97.5 (20.6)% (range 60–122%) sitting and 92.7 (17.1)% (range 63–116%) supine; in group 2 it was 103.0 (14.5)% (range 87–118%) sitting and 96.0 (10.0)% (range 84–109%) supine. PEFR in group 1 was 84.2 (16.4)% (range 59–104%) sitting and 78.8 (17.7)% (range 52–95%) supine; in group 2 it was 89.5 (16.5)% (range 65–105%) sitting and 89.8 (17.4)% (range 63–110%) supine. No statistical difference was found either between groups

MIREILLE C. BERTHOUD, B.M., F.C.ANAES.; JOHN E. PEACOCK, M.B., CH.B., F.C.ANAES.; CHARLES S. REILLY, M.D., F.C.ANAES.; Department of Anaesthesia, University of Sheffield Medical School, Beech Hill Road, Sheffield S10 2RX. Accepted for Publication: April 26, 1991.

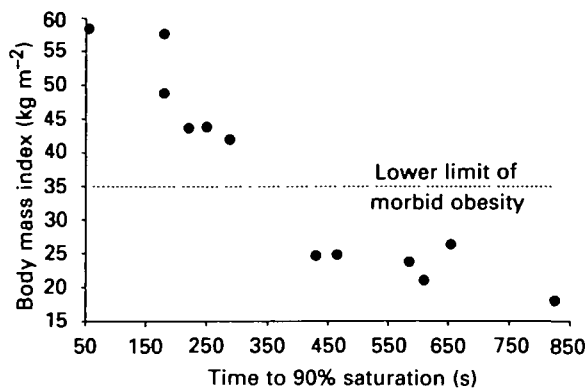


FIG. 1. Times to 90% saturation compared with BMI.

(Student's *t* test) or within groups between positions (paired *t* test).

Oral premedication consisted of ranitidine 150 mg the night before surgery and temazepam 20 mg with ranitidine 150 mg 1 h before induction of anaesthesia.

In the anaesthetic room, venous cannulation and, in the obese patients, left radial artery cannulation were performed under local anaesthesia. Monitoring consisted of arterial pressure measurement every 1 min throughout the period of study using a Dinamap or via the arterial cannula, continuous ECG, oxygen saturation (Sp_{O_2}) using the finger probe of a pulse oximeter (Kontron Instruments), and end-tidal carbon dioxide (E'_{CO_2}) (Kontron Capnograph).

Preoxygenation was performed using a fresh gas flow of 100% oxygen at 8 litre min^{-1} , from a flushed Mapleson A circuit, with an air-tight seal on the face mask. After 3 min of preoxygenation, anaesthesia was induced with propofol 10 mg ml^{-1} at a rate of 1200 ml h^{-1} , with cricoid pressure to minimize the risk of aspiration. On loss of verbal contact with the patient, alfentanil 0.01 mg kg^{-1} and suxamethonium 1 mg kg^{-1} (to a maximum of 100 mg) were given i.v. The patient's trachea was intubated and the tracheal cuff immediately inflated. The position of the tracheal tube was confirmed by seeing it pass between the cords, and by giving the patient a single breath of oxygen, in order to establish the presence of carbon dioxide in the expired gas. When the airway was secured, vecuronium 0.1 mg kg^{-1} (maximum 10 mg) was given to ensure apnoea. Anaesthesia was maintained by an infusion of propofol at 6 mg $\text{kg}^{-1} \text{h}^{-1}$. The tracheal tube was disconnected from the gas supply, and the patient

remained apnoeic until Sp_{O_2} decreased to 90%. The time from the injection of the suxamethonium, to this point was noted. The lungs were then ventilated with 100% oxygen. E'_{CO_2} at this point, and the time to recover to Sp_{O_2} 96% were noted. In the obese patients, arterial blood-gas tensions were measured before the onset of preoxygenation ($P_{a_{O_2}}$ 11 (1.9) kPa (range 8.6–13.4 kPa); $P_{a_{CO_2}}$ 5 (0.6) kPa (range 4.1–5.6 kPa)), at tracheal intubation ($P_{a_{O_2}}$ 42.9 (20.1) kPa (range 7.3–60.4 kPa); $P_{a_{CO_2}}$ 5.8 (1.0) kPa (range 4.3–7.3 kPa)), and at Sp_{O_2} = 90% ($P_{a_{O_2}}$ 7.9 (0.9) kPa (range 6.9–9.0 kPa); $P_{a_{CO_2}}$ 6.2 (1.1) kPa (range 4.6–7.3 kPa)). The patient then proceeded to surgery.

Results for the time taken to desaturate to 90% and to resaturate to 96% after the start of IPPV were analysed using Student's *t* test. The mean time to 90% saturation was 196 (80) s (range 55–208 s) in the obese group and 595 (142) s (range 430–825 s) in the control group ($P < 0.001$) (fig. 1). The time to reach a saturation of 96% after the start of IPPV was 36.7 (13.3) s (range 25–45 s) in the obese group, and 22.5 (9.3) s (range 10–35 s) in the controls ($P = 0.062$). In one patient, Sp_{O_2} decreased to 82%, but in no other was it less than 87%. In no patient was $E'_{CO_2} > 7.3\%$, and there were no arrhythmias or hypertension.

COMMENT

This study demonstrates that preoxygenation for 3 min in morbidly obese patients provided a significantly shorter period of adequate oxygenation than in patients of normal weight. This is in agreement with the findings of Jense and colleagues [4]. One of our patients (BMI 58.4 kg m^{-2}) clearly demonstrated the increased risk associated with induction and tracheal intubation in this group, desaturating to 90% only 55 s after administration of suxamethonium, and before complete onset of relaxation. This patient's trachea was intubated, at first attempt, 65 s after the injection of suxamethonium, by which time Sp_{O_2} was 82%.

Bedside lung function tests with the patient in either the sitting or the supine position were not significantly different between groups. Preoxygenation is designed to replace the FRC nitrogen with oxygen, and can normally be achieved to an end-tidal nitrogen concentration of 4% in 3 min in volunteers [5]. This increases the stores of oxygen within the body by the volume of the FRC. Obesity is associated with a decreased

FRC, which is reduced further by placing the patient in the supine position [6], allowing the tidal volume to approach and even fall within the closing capacity. Ideally, measurements of FRC, together with closing capacity, may produce better prediction of the effectiveness of preoxygenation in obese patients; however, these volumes are difficult to measure and are unlikely to be available routinely. All our patients achieved an Sp_{O_2} of 100% within 30 s of the start of preoxygenation, indicating that the likely limitation is one of size of stored volume of oxygen in the body, rather than ability to reach that store, or to saturate the blood. This is supported by the blood-gas results.

This study illustrates further that awake tracheal intubation should be considered in any morbidly obese patient presenting for emergency surgery, especially those with expected difficulty in tracheal intubation, and preoperative bedside

lung function tests cannot be used to predict the effectiveness of preoxygenation.

REFERENCES

1. Robertson IK, Eltringham RJ. Anaesthetic management of the morbidly obese. *British Journal of Hospital Medicine* 1985; **34**: 224–228.
2. Wilson ME, Spiegelhalter D, Robertson JA, Lesser P. Predicting difficult intubation. *British Journal of Anaesthesia* 1988; **61**: 211–216.
3. Samsoon GLT, Young JRB. Difficult tracheal intubation: a retrospective study. *Anaesthesia* 1987; **42**: 487–490.
4. Jense HG, Dubin SA, Silverstein PI, O'Leary-Escolas U. Effect of obesity on duration of apnea in anesthetized humans. *Anesthesia and Analgesia* 1991; **72**: 89–93.
5. Berthoud M, Read DH, Norman J. Preoxygenation—How long? *Anaesthesia* 1983; **38**: 96–102.
6. Damia RW, Mascheroni D, Croci M, Tarenzi L. Preoperative changes in functional residual capacity in morbidly obese patients. *British Journal of Anaesthesia* 1988; **66**: 574–578.