PROGRESSIVE HYPOXEMIA, HYPERCARBIA AND HYPERTHERMIA ASSOCIATED WITH PROLONGED ANESTHESIA

- A Case Report -

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Abstract

The authors report a case of 66-year-old female patient, 55 kg, ASA I who, under general anesthesia in supine position, developed gradual hypoxemia (from a baseline PaO_2 of 250 to 91 mmHg), carbon dioxide build up (from a baseline $PaCO_2$ 31 to 41 mmHg) associated with gradual hyperthermia up to 38.3°C over seven hours, intra-operatively. These observations were noted while using a semi-closed carbon dioxide absorption circuit in conjunction with the Hygroster filter at a fresh gas flow of 4 l/min of 50% nitrous oxide in oxygen. While the ventilation pattern was unchanged throughout the procedure, there was a change in exhaled tidal and minute ventilation volume with a net decrease of 28 ml and 0.4 l/min respectively. Findings are probably the result of pulmonary atelecatasis under general anesthesia due to the use of a relatively high-inspired oxygen concentration (50%). In addition, the use of a high humidity and temperature heat moisture exchanger (HME) filter (Hygroster) in conjunction with the circle absorber system may have resulted in over humidification and aggravated the pulmonary atelecatasis over the long operative time.

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Key words: Pulmonary atelectasis; hypoxemia; hypercarbia; hyperthermia; long-term anesthesia; heat moisture exchanger filter.

Introduction

Atelectasis formation is an unavoidable adverse effect of general anesthesia. The patient's position and the composition of inspired gases influence the extent of atelectasis[1,2]. Also, the degree of atelectasis correlates closely with the magnitude of shunt[2,3].

We present a case report of progressive hypoxemia and carbon dioxide retention associated with hyperthermia over seven hours of general anesthesia, while using a high FiO_2 and a heat moisture exchanger.

Case Report

A 66-year-old female 55 kg was scheduled for anterior cervical decompression and fusion with iliac crest bone graft, C5-C6 discectomy + C5 corpectomy and bilateral foraminotomy, under general anesthesia with somatosensory evoked potential monitoring (SSEP). The patient was non-smoker, non-diabetic, non-hypertensive, with a negative cardiac history and good exercise tolerance. Pre-operative medications included Decalcit® (Vit. D_3, calcium) and Lindilane® (paracetamol, codeine). The patient's EKG and chest X-ray were normal. Laboratory tests including hematocrit, hemoglobin, blood urea nitrogen, creatinine, electrolytes, coagulating profile and urine analysis were also normal.

She was premedicated with Diazepam 5 mg PO and Glycopyrrolate 0.2 mg IM one hour preoperatively. On admission to the operating room, two intravenous lines and a radial artery were accessed. The pre-induction vital signs showed BP 118/69 mmHg, pulse 75/min, normal EKG tracing and oxygen saturation (SpO_2) of 99% on room air.

During pre-induction, the patient received 30-40% oxygen nasal cannula, local anesthetic pharyngeal spray and sedative doses of

midazolam. Aided by fiberoptic bronchoscope, the trachea was intubated with 7.5 mm-cuffed tube. Following tracheal intubation, anesthesia was induced with intravenous xylocaine 1.5 mg/kg, propofol 2 mg/kg, vecuronium bromide 0.1 mg/kg and fentanyl 5 μg/kg. Anesthesia was maintained with 4 l/min O_2/N_2O (50%/50%), and isoflurane 0.75%, supplemented with fentanyl 1 μg/kg/hr and vecuronium bromide 0.1-0.15 mg/kg/hr which were administered continuously via two syringe drivers.

Directly after induction, Kefzol® (cefazolin) 2 grams was given intravenously. Intermittent positive pressure ventilation (IPPV) was controlled by Ohmeda® 7000 ventilator. A semi-closed rebreathing system was used with soda lime canister Ohmeda® series 5A. A Hygroster filter DAR® (Mallinckrodt – Medical S.P.A, 354/5964) was placed between the Y piece of the breathing system and the endotracheal tube. Oxygen monitor (Ohmeda® 5120) and volume monitor (Ohmeda® 5400) were connected respectively to the inspiratory and expiratory limbs of the breathing circuit. Also a nerve stimulator was used. A temperature probe was inserted into the lower third esophagus. A Foley catheter was inserted, and a lower body-warming blanket was used. The patient received ambient temperature Lactated Ringer's solution and her position was supine.

The following parameters were monitored: invasive and non-invasive BP, pulse, EKG, patient's temperature, fraction of inspired oxygen (FiO_2), functional pulsatile oxygen saturation(SpO_2), end tidal carbon dioxide tension ($ETCO_2$), inspiratory airway pressure, expiratory tidal volume (V_T), and minute volume (V_{min}). A baseline recording was taken 3 min after establishing IPPV. Arterial blood gases were analyzed every hour and more frequently after the sixth hour, while the other monitoring parameters were taken every 15 min. Table 1 presents selected physiological parameters. The ventilation pattern was unchanged all through the procedure and total anesthesia time was 8 hours and 10 min.

Table 1

Physiologic parameters recorded over 7:45 hrs. of observation time intra-operatively

Time hrs:min	V_T ml	V_{min} l min	FiO_2	SpO_2 %	$ETCO_2$ mmHg	$PaCO_2$ mmHg	PaO_2 mmHg	pH	BP mmHg	AP cmH_2O	T °C
0:00	476	5.1	0.5	99	26	31	250	7.44	112/69	15	36.2
0:15	464	5.3		99	25				102/64	15	35.6
0:30	470	4.8		98	26				116/70	15	35.5
0:45	451	4.7		97	27				123/74	15	35.5
1:00	461	4.9		97	28	35	157	7.41	119/69	15	35.7
1:15	457	4.8		97	28				144/81	15	35.9
1:30	438	4.7		96	28				128/71	17	36.2
1:45	435	4.7		97	29				129/72	17	36.4
2:00	448	4.7		97	30	35	130	7.39	125/69	17	36.7
2:15	432	4.6		97	30				123/68	17	36.8
2:30	426	4.6		97	31				119/65	17	37.2
2:45	429	4.5		97	33				118/64	17	37.4
3:00	448	4.6		97	34	37	120	7.38	114/62	17	37.6
3:15	446	4.6		97	34				116/63	17	37.6
3:30	438	4.6		97	35				104/57	17	37.7
3:45	438	4.7		97	35				115/61	17	37.9
4:00	448	4.6		97	36	40	118	7.37	129/67	17	38.0
4:15	438	4.6		97	36				122/64	17	38.0
4:30	438	4.7		96	37				120/63	17	38.1
4:45	435	4.7		96	36				117/62	17	38.2
5:00	445	4.7		96	37	39.9	109.9	7.36	113/61	17	38
5:15	445	4.7		95	36				107/59	18	38.4
5:30	432	4.6		95	37				125/66	18	38.4
5:45	441	4.6		95	38				108/60	18	38.5
6:00	441	4.6		95	38	40.5	92	7.35	108/60	18	38.5
6:15	534	4.8		95	36				109/60	18	38.4
6:30	448	4.7		95	36	41	89	7.36	111/62	18	38.4
6:45	480	4.6		95	36				112/61	15	38.4
7:00	448	4.7		95	36	41	91	7.36	104/58	15	38.3
Filter removal and decrease of FiO_2 to 0.33											
7:15	464	4.8	0.33	95	35				123/65	15	38.3
7:30	464	5.0		96	31				125/67	14	38.3
7:45	465	5.0		97	31				125/68	14	38.2

V_T, exhaled tidal volume; V_{min}, exhaled minute volume; FiO_2, fraction of inspired oxygen; SpO_2, functional pulsatile oxygen saturation; $ETCO_2$, end tidal carbon dioxide tension; $PaCO_2$, arterial carbon dioxide partial pressure; PaO_2, arterial oxygen partial pressure; pH; BP, blood pressure; AP, inspiratory airway pressure; T, esophageal temperature.

By the seventh hour, there was a decrease in expired V_T and V_{min} with a net change of 28 ml and 0.4 l/min respectively. Also, there was a gradual decrease in PaO_2 reaching a value of 91 mmHg from a base line of 250 mmHg, while the net increase in $PaCO_2$ was 10 mmHg from a base line of 31 mmHg. The inspiratory airway pressure showed a consistent increase to 18 cmH_2O from a base line of 15 cmH_2O over six and a half hours, thereafter dropped to baseline. Except for the initial mild drop, the patient's temperature gradually increased to reach a maximum of 38.5°C at 5:45 hrs, with a gradual minor drop thereafter. At the seventh hour the PaO_2 and $PaCO_2$ recordings were 91 and 41 mmHg respectively. The filter was removed from the breathing system and FiO_2 was decreased to 0.33. Upon filter removal, and over the next 45 min, there was an increase in the expiratory V_T and V_{min} back to around their baseline, an increase in SpO_2 to 97% and a decrease in $ETCO_2$ to 31 mmHg. The inspiratory airway pressure dropped to 14 cmH_2O below baseline and blood pressure increased by about 15%. The EKG was normal all through the operation.

Fentanyl and isoflurane were stopped 1:30 and 0:30 hr before end of surgery respectively. Extubation was uneventful. Postextubation, bilateral lung auscultation revealed normal breath sounds. The blood pressure and pulse were within normal limits. The patient received 3900 ml lactated Ringer's solution, the total urine output was 400 ml, and the estimated blood loss was around 200 ml.

On admission to the recovery room, the patient's SpO_2 was 95% on room air. Administering 40% oxygen by facemask resulted in SpO_2 of 96-97%. Prior to patient's transfer to the floor and after two-hour stay in the recovery room, the SpO_2 was 95% on room air. The patient was transferred to the floor on 40% oxygen facemask and her oral temperature was 37.5°C.

Discussion

Our patient had general anesthesia in the supine position, using a carbon dioxide absorption circuit, and a fresh gas flow of 4 l/min of 50%

nitrous oxide in oxygen, in conjunction with a Hygroster filter. The patient demonstrated progressive hypoxemia and carbon dioxide retention associated with gradual hyperthermia over seven hours.

Numerous factors might have contributed to hypoxemia and hypercarbia in our patient. The inspired FiO_2 was set at a relatively high cncentration (0.5), and this was continuously verified by the oxygen monitor. Nitrous oxide 50% was administered for a better recording of the somatosensory evoked potential[4]. Also hyper, rather than normoventilation. was established at baseline. The patient did not have sepsis, liver failure, right to left cardiac shunt or arteriovenous malformation, which can account for a significant shunt. Also, pulmonary emboli can lead to an acute $ETCO_2$ decrease rather than an increase. The patient had a stable blood pressure, as a function of cardiac output and peripheral vascular resistance, and minimal blood loss. The reduction in oxygen carrying capacity, if estimated, could be the result of a decrease in oxygen saturation and PaO_2 rather than a drop in hemoglobin concentration and cardiac output. Ventilation-perfusion mismatch accounts considerably to hypoxemia[5]. Our patient had no bronchial intubation, endotracheal tube plugging, bronchospasm or pneumonia. The patient did not show basal lung crepitations after extubation, and consequently, frank pulmonary edema was ruled out. Based on medical history and hemodynamic stability of the patient intra-operatively, conditions that significantly increase carbon dioxide production such as malignant hyperthermia or thyrotoxicosis were excluded. In addition, sodium bicarbonate was not administered and the soda lime absorber was not exhausted, as indicated by its warmth and colour index. Also, the inspiratory and expiratory dome valves of the circle absorber system were functioning properly. Atelectasis is the most likely explanation for the hypoxemia and hypercarbia that our patient encountered. Pulmonary atelectasis under general anesthesia may be attributed to the use of high FiO_2 as well as to the over or under humidification. Excessive artificial humidification of the inspired gases may produce a situation as umphysiological as that observed by ventilation with under humidified gases[6].

Pulmonary atelectasis under general anesthesia in the supine

position was shown to reduce the cross sectional area of both lungs and cause atelectasis formation in the dependent lung regions in 4 out of 5 patients[1]. This is, presumably, due to the loss of forces that normally keep the lungs expanded[7]. In a randomized clinical trial, the incidence of computed tomography determined atelectasis was higher in the group assigned to 80% perioperative oxygen (94%) than the 30% oxygen group (64%), although the difference was not statistically significant[8]. In another randomized clinical trial, high concentrations of inspired oxygen (100%), whether at induction or during maintenance, were shown to increase significantly atelectasis formation under general anesthesia, in comparison to lower concentrations (30%[2]). The rate of atelectasis formation was around 3 times more with the 100% than with the 30% oxygen group. In our patient, the atelectasis might have been induced by the use of 50% oxygen in nitrous oxide during maintenance. Intra-operatively, PaO_2 and $PaCO_2$ reached respectively their maximal change after 6:30 hours of general anesthesia.

Our patient did not benefit from the positive end expiratory pressure (PEEP) effect created by the Hygroster filter[9]. Previous reports have shown that PEEP per se does not improve oxygenation in unselected groups of patients[3]. The PEEP should be preceded with a vital capacity manoeuvre (VCM, 40 cmH_2O) to maintain the reduction of pulmonary atelectasis induced by the VCM[10,11]. The relatively elevated magnitude of V_{min} at base line ($PaCO_2$ 31 mmHg) and the high inspired oxygen administration (50%) were not sufficient to effectively prevent carbon dioxide build up or to compensae for the mild hypoxemia over the long operative duration. Our patient had SpO_2 of 95% on room air in the recovery room and before discharge to the floor, which may suggest the possibility of intraoperative pulmonary atelectasis.

The patient experienced an initial drop in temperature, which is likely to be attributed to heat loss from wide skin exposure and scrubbing prior to surgery. However, the initial decrease in temperature was followed by a gradual increase over time. Several conditions, including sepsis, fever, stress of major surgery, bacteremia, hypothalamic lesions, hyperthyroidism, malignant hyperthermia or pheochromocytoma

(cathecholamine surges), may lead to an acute rather than a gradual increase in patient's temperature. The presence of fever on the first post-operative night is common and is usually ascribed to atelectasis[12]. In addition, both under and over humidification under general anesthesia may be associated with bronchial, bronchiolar and alveolar changes that can result in pulmonary atelectasis, hypoxemia, hybercarbia and changes in body temperature[6]. Under humidification leads to hypothermia due to heat loss from distal respiratory tract, dryness, mucociliary destruction and impaired surfactant activity[6]. In contrast, over humidification leads to hyperthermia due to heat gain by the distal respiratory tract, water condensation, inefficient diffusion oxygen exchange, mucociliary adhesions and surfactant dilution[6]. Consequently, while under-humidification is unlikely an explanation for the hyperthermia observed in our case, over humidification cannot be ruled out. Nevertheless, bronchial mucus transport inefficiency and atelectasis under conditions of mild over-humidification need not manifest in the full-blown clinical picture of severe alveolar over hydration.

Humidification under general anaesthesia arises from the heat moisture exchanger filter (HMEF) and the semi-closed rebreathing system. The Hygroster filter used in our patient has been shown to produce the highest moisture and temperature output in comparison to other filters such as the Humid-vent and PALL Ultipor BB 100 filter[13]. According to the manufacture's specifications, it has a moisture output of 34 mgH_2O/l, a temperature output of 33.2°C, relative humidity of 92 to 98% and resistance to flow 0.8-3.4 cmH_2O. The efficiency of this filter is derived from its simple water condensation and, additionally, to its hygroscopic element that chemically conserves moisture in the expired gas. In our case, the inspiratory airway pressure drop from 18 cmH_2O to the baseline of 15 cmH_2O at 6:45 hours intra-operatively may be attributed to the depletion of the hygroscopic element of the filter.

On the other hand, three main studies have been conducted to evaluate the humidity and temperature output of the circle absorber system at a fresh gas flow of 5 l/min, close to our case (4 l/min), with varying results[14,15,16]. In all studies, the humidity and temperature output

were evaluated over short duration not exceeding one-hour. While Bengtson and colleagues, in a laboratory set up, demonstrated an acceptable humidity and temperature output of inspired gases (RH 97%, AH 22.6 mgH_2O/l, T 24.6°C)[14], in a re-assessment, the humidity output was considered low (AH 11.3 mgH_2O/l), with the temperature being kept relatively constant (23.6°C)[15]. More recently, in a randomized clinical trial, the humidity output of the semi-closed rebreathing system alone was considered to be insufficient and remained unchanged over the one-hour duration (RH 74.14%, AH 17.63 mgH_2O/l, T 25.47°C)[16]. The addition of the Humid Vent 2 Gibeck filter, however, increased significantly the humidity and temperature output to acceptable levels (RH 83.44%, AH 29.29 mgH_2O/l, T 32.74°C). This increase was gradual over the one hour of investigation, and it is unclear whether this would have persisted continuously over a longer observation time. Previous studies have shown variations in the humidity and temperature output when using the circle absorber system with fresh gas flow of 5 l/min. Our case report indicates that mild over humidification may have contributed to the hyperthermia and hypoxemia observed. Further studies are needed to evaluate the effect of the Hygroster filter on inspired gases from the anesthesia circle absorber system with different fresh gas flows over long duration. Driven by the hypothesis that mild over humidification might be a contributory factor, the Hygroster filter was disconnected from the breathing circuit, which was followed by an increase in the oxygen saturation up to 97%, despite decreasing the FiO_2 from 0.5 to 0.33.

In conclusion, the hypoxemia and carbon dioxide build up over seven hours intra-operatively, may be attributed to the development of pulmonary atelectasis under general anesthesia secondary to the use of 50% inspired oxygen. The pulmonary atelectasis is likely to have been aggravated by over humidification resulting from the use of a highly efficient heat moisture exchanger (HME) Hygroster filter. Findings of the present case report concurs with earlier work recommending the administration of low inspired oxygen (30%) whenever possible[2], and indicates that adequate attention should be given when high inspired oxygen concentration is used over a long operative time. We also advise the use of low humidity and temperature output HME filter in conjunction

with the anesthesia circle absorber system.

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