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Inadvertent severe hypercapnia during general anesthesia: drop-in oxygen saturation or electrocardiography changes; which one warns us earlier?

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Abstract

Background: During general anesthesia especially when the nurse or anesthesiologist forgets to change manual to controlled mode after successful endotracheal intubation, capnography shows End-tidal Co₂ above 20 mmHg after checking the place of the tracheal tube and will remain on the screen permanently. In this scenario, the patient receives a high concentration of oxygen, and Spo₂ (oxygen saturation) does not drop for a long time which is too late to intervene. It has been all-time questionable which one of the cardiac dysrhythmias or Spo₂ dropping occurs earlier.

Results: Medical records of seven deceased patients reviewed. All of them had electrocardiogram changes including premature ventricular contraction or bradycardia as a first warning sign. Oxygen saturation remains above 95% even with cardiac dysrhythmia.

Conclusions: Bradycardia and premature ventricular contraction were the first warning findings for severe hypercapnia during general anesthesia and occurred earlier than dropping oxygen saturation. Furthermore, the normal capnography waveform is more reliable than the End-tidal Co₂ number for monitoring.

Background

Monitoring during general anesthesia is the most important issue to notify anesthetic complications. This monitoring usually includes heart rate, blood pressure, oxygen saturation, and end-tidal CO₂ (Etco₂) for simple general anesthesia. The pulse oximeter is widely used as a standard of care to monitor patients during anesthesia and postoperatively (Ayas et al., 1998). Once patients are receiving a high concentration of supplemental oxygen without ventilation, oxygenation can be maintained with inadequate alveolar ventilation because of the diffusion of oxygen molecules from the airways into the alveoli. In this scenario, severe hypercapnia and respiratory acidosis may result. This event usually happens when an

anesthesiologist or nurse anesthetist forgets to shift the mode of the anesthetic machine from manual to controlled after checking the right endotracheal tube placement in manual mode. In this setting, oxygen saturation almost always does not drop and hemodynamic changes will not occur until Pco₂ increases dramatically. If we use capnography for monitoring and pay attention to the capnography waveform, we will notify that no waveform pattern exists or disappears suddenly and this can prevent catastrophic events, but the problem may occur when the patient is intubated and the place of the tracheal tube confirmed by auscultation or by the appearance of the proper number of Etco₂ and capnography waveform on the screen while the anesthetic machine is on the manual mode and manual bag pressure provided Etco₂ > 20 mmHg during checking the place of the tube and then it is forgotten to change to controlled mode. In this situation, if we look at

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Etco₂, the number will be fixed on the screen, above 20 mmHg (this Etco₂ is for the moment of endotracheal tube confirmation on manual mode) and will not disappear on most screens used for ETCO₂ monitoring and the only and remaining parameter warns us is the cessation of capnography waveform.

To our knowledge, there is no literature to clarify which parameter of monitoring other than Etco₂ will change and direct our attention to check this malfunction. This event prompted us to know that which one of “drop-in oxygen saturation or cardiac arrhythmia” will appear earlier.

Methods

We reviewed 7 out of 9 charts and medical records of deceased patients in forensic medicine which the cause of death, determined as cardiac arrest due to anesthetic malfunction or anesthesiologist or nurse anesthesia fault led to severe hypercapnia. Two out of 9 medical records were not complete and we could not find electrocardiography record before the cardiovascular arrest. These deceased patients were reported from different hospitals to the forensic medicine organization for further consideration. Although it was hard to find these medical reports among all medical reports from 2008 to 2018 in forensic medicine organization, 5 staves helped us for looking for the patients' medical records and it took about 1 year to be completed. Moreover, several anesthesiologists who were involved and were exposed to or heard about these catastrophic events helped us to better find the medical records of these deceased patients among all medical records archived over the last 10 years.

Prior to starting this study, we met the requirement to obtain the permission and received signed consent form patients offspring or their legal executors, to be eligible to have access their records. Patient anonymity was

preserved and the research project was approved by Ethic committee of Anesthesiology Research Center, Shahid Beheshti University of Medical Sciences.

In this study, medical records of seven deceased patients reviewed. Of them 3 patients underwent general surgery, 3 urologic surgeries, and 1 of them orthopedic surgery. All of them were between 32 and 61 years old. In forensic medicine organization, lack of proper ventilation was determined as a cause for cardiovascular arrest during general anesthesia for all of these deceased patients.

We review the charts and medical reports of these 7 patients and assess the changes recorded in their monitoring parameters including Spo₂, Mean Arterial Pressure (MAP), hear rate, and electrocardiogram prior to starting general anesthesia induction and 3 min prior to starting cardiopulmonary resuscitation (CPR) until CPR. We selected 3 min precedent to CPR, since in all 7 cases the first signs and changes did not appear earlier than this particular point of time.

Results

Medical records of seven deceased patients were reviewed. Records discovered changes before starting CPR or any intervention including administration of atropine. There were EKG (Lead II or III) in medical records of all patients from 5 min before intervention. As it is shown in Table 1, severe hypercapnia during general anesthesia while the patient is receiving oxygen through the endo tracheal tube was not associated with any drop in O₂ saturation and any remarkable changes in blood pressure within the last minutes before the time needed to intervene for cardiopulmonary resuscitation (CPR). On the contrary, bradycardia and cardiac dysrhythmia were common (*p* value = 0.17 versus 0.018 for blood pressure and heart rate; respectively) (Table 1).

Table 1 Parameters monitoring and changes before induction of anesthesia and during the last 3 min prior to starting standard CPR based on patients' records

	SPO ₂		MAP		<i>P</i> value	HR		EKG		
	Prior to induction of GA in room air	3 min prior to starting CPR	Prior to induction of GA	3 min prior to starting CPR		Prior to induction of GA	3 min prior to starting CPR	<i>P</i> value	Prior to induction of GA	3 min prior to starting CPR
Case#1	≥ 95%	≥ 95%	85	88	0.17	88	55	0.018	NL	Sinus bradycardia
Case#2	≥ 95%	≥ 95%	81	79		93	49		NL	Sinus bradycardia and PVC
Case#3	≥ 95%	≥ 95%	84	88		97	48		NL	Sinus bradycardia and PVC
Case#4	≥ 95%	≥ 95%	80	82		89	53		NL	Sinus bradycardia and PVC
Case#5	≥ 95%	≥ 95%	90	88		67	49		NL	Sinus bradycardia and PVC
Case#6	≥ 95%	≥ 95%	84	85		101	44		NL	Sinus bradycardia and PVC
Case#7	≥ 95%	≥ 95%	78	85		96	56		NL	Sinus bradycardia

CPR cardio-pulmonary resuscitating, EKG electrocardiogram, GA general anesthesia, HR heart rate, MAP mean arterial pressure, NL normal rhythm of EKG, PVC premature ventricular contraction, SPO₂ oxygen saturation measured by pulse oximetry

Wilcoxon test for p value was used to compare within each group. $P < 0.05$ was considered statistically significant.

Discussion

In our study, we understood that in case of severe hypercapnia during general anesthesia while receiving pure oxygen (3–6 L/min) through endotracheal intubation, EKG and heart rate monitoring is the most reliable monitoring used to notify nurse anesthetist or anesthesiologist to have hypercapnia following lack of ventilation.

Several cases of severe hypercapnia have been reported in the literature, many of which are a result of a defective apparatus used during anesthesia or an oversight (Potkin & Swenson, 1992; Dinnick, 1968). For example, in one case, the directional valves were inadvertently removed from a circle anesthesia unit, and the endotracheal tube was connected to the breathing tubes; thus, the patient was allowed to rebreathe exhaled carbon dioxide for 3 h, resulting in a Pco₂ of 234 mmHg. Obstruction to flow through the inner tube of a coaxial circuit, due to compression, twisting, or kinking is reported as gas flow failure to the patient end before induction of anesthesia (Grag, 2009; Gooch & Peutrell, 2004; Inglis, 1980). Berner reported a disconnection in the metallic part of the anesthesia machine (Berner, 1987) a malfunction of the inner tube resulting from laceration of its wall and consequent dead space breathing evident in the first half-hour of anesthesia was identified as a cause for hypercapnia. In our patient, no equipment malfunction was discovered during the anesthesia, but nurse or physician errors incurred to create a threatening situation for patients. In all of the cases reported previously, hypercapnia and hypoventilation are caused by an anesthetic machine malfunction, and by using a new anesthetic machine, these malfunctions can be notified by making alarm sounds.

Hypercapnia during general anesthesia has three main causes or fall into 3 categories and our cases fall into the third category as follows:

Firstly, the patient may have inadequate ventilation, which will result in a slow rise of Pco₂. This category is also seen among patients after receiving premedication for general anesthesia (Mosafa et al., 2016).

The second cause of gross hypercapnia is the re-inhalation of exhaled carbon dioxide. Most of these cases reported as a result of missing valves in a circle absorber system which prevented the exhaled carbon dioxide from being absorbed by the soda-lime or malfunction of the inner tube of a co-axial (BAIN'S) circuit.

The third cause of gross hypercapnia is the inhalation or injection of exogenous carbon dioxide. The height to which the pco₂ can raise under these circumstances is

limited only by atmospheric pressure, and a level of 600 mmHg has been attained in dogs (Graham et al., 1960).

If alveolar ventilation stopped, the Pco₂ increases at a rate of 2.7 to 4.9 mmHg/min. In the absence of supplemental oxygen, the maximal carbon dioxide pressure compatible with life is approximately 100 mmHg. Beyond this value, there is the excessive displacement of alveolar oxygen leading to lethal hypoxia and death. Oxygenation may be maintained during insufficient and or absent ventilation if a high concentration of oxygen is administered. Because of the extraction of oxygen by erythrocyte hemoglobin in the pulmonary capillaries, an oxygen concentration gradient develops between the upper airway and the alveoli. This leads to the mass effusion of oxygen molecules down the airways and into the alveoli, even in the absence of ventilation.

As we know, in patients receiving concentrated oxygen, profound alveolar hypoventilation and hypercapnia can be present despite adequate oxygen saturation. All health care workers who use pulse oximeters should be aware of this limitation.

Hypercapnia induces catecholamine release. The patient may present with tachycardia, arrhythmias, excessive sweating, hypertension, and peripheral vasodilation which may result in excessive intraoperative blood loss (Youssef & Iyasere, 2020). Berner reported hemodynamic changes, pupillary dilation, hyperkalemia, and severe respiratory acidosis (Berner, 1987).

Arterial blood pressure usually rises as the Pco₂ increases in both conscious and anesthetized men. A fall in Pco₂ is associated with a fall in blood pressure and cardiac output (Pyrns-Roberts & Klman, 1996).

The upper limits of abnormally high CO₂ that jeopardize the safety of patients receiving high concentrations of oxygen through tracheal intubation are still open to debate. Mild hypercapnia in anesthetized patients has been shown to increase blood pressure, cardiac output, and heart rate. However, hypercapnia that leads to a pH below 7.2 has been associated with cardiovascular depression. Severe hypercapnia can also result in cardiac dysrhythmias. Without the monitoring equipment, the decision when to intervene becomes more difficult. Hypercapnia is mostly associated with decreased CO₂ elimination during anesthesia, not increased CO₂ production. Faulty unidirectional valves, exhausted soda lime, and inadequate O₂ flow in non-rebreathing systems are machine-related problems that lead to hypercapnia. The anesthetic machine should be checked regularly to detect faulty unidirectional valves and exhausted soda lime.

Conclusions Despite the existence of new modern anesthetic machines which notify us apparatus malfunction, negligence of changing manual to controlled mode

after induction of anesthesia at the beginning of operation will not come to our notice even with a new modern anesthetic machine. This can lead to irreversible conditions and delay intervention may do harm patients forever. Bradycardia and premature ventricular contraction are the first findings of severe hypercapnia that appeared before dropping O₂ saturation and need to start CPR.

Abbreviation

CPR: Cardiopulmonary resuscitating

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Authors' contributions

A S managed the survey and wrote the paper. A T gathered the data. SS wrote and edited the paper. MM analyzed and gathered the data. MAP contributed to the data analysis. FS managed and coordinated the writing. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This study received ethics approval from the Ethics committee of the Anesthesiology Research Center, Shahid Beheshti University of Medical Sciences. Written informed consent was obtained from all patients' legal guardians for this study based on our country regulations provided for having access to patients' data, analysis, and publication.

Consent for publication

All patients involved in this study have died in the past, consent form signed by all patients' legal guardians based on our country regulations provided for having access to patients' data, analysis, and publication. Consent form signed by legal guardian for publication have been received.

Competing interests

All authors declare that they have no competing interests.

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