Acid-Base Disturbances and Related Factors in Patients under General Anesthesia

M Lak¹, H Araghizadeh¹

¹Department of Anesthesiology, Faculty of Medical Science, Baqiyatallah (a.s.) University of Medical Sciences, Tehran, Iran

Abstract

Background: In patients undergoing surgery, acid-base imbalance during general anesthesia causes disruption in the function of cardiovascular, respiratory, musculoskeletal and the central nervous system. Since the potential complications of acid-base disturbances are serious, we decided to explore these changes and related causes so that prevention and compensation would be feasible.

Methods: In this study, acid-base disruption and its causes during general anesthesia were studied in 60 patients. The patients were divided into three groups based on the location of surgery: abdominal (I), extremities (II), and head and neck (III). Body central temperature, type, and volume of injected fluids and bicarbonate (as needed) were recorded. In the second, third, and fourth hours of surgery, arterial blood gases and pH were measured.

Results: The incidence and degree of metabolic acidosis in all patients were significantly related to the duration of the surgery. The number of patients with acidosis and the number of patients requiring treatment were also greater in groups I and II than in group III. Although all patients became hypothermic during the surgery, there was no significant relationship with development of acidosis. No relationship was found between the type and volume of the solution administered and development of acidosis.

Conclusion: In our opinion, monitoring of acid-base status in prolonged surgeries especially in abdominal and extremity operations seems necessary.

Keywords: Acid-base disturbances; General Anesthesia; Surgical Operation; Monitoring; Blood Gases

Introduction

All living organisms need to keep the acid-base balance and oxygenation to survive. The acid-base balance regulation is dependent on the concentration of H⁺ and HCO₃⁻ ions in the body fluids. In a healthy and conscious person, the compensatory mechanisms act to keep the acid-base balance appropriate for normal enzymes activities, electrolytes diffusion, hemoglobin saturation and cardiac contraction power. These all lead to normal functioning of vital organs.¹

In patients undergoing surgery through general anesthesia, acid-base disturbances have been frequently seen. In one study, about 50% of patients under general anesthesia were affected with metabolic alkalosis; however, other acid-base changes are not prevalent.² Both the patient and anesthesiologist were aware that the healthy people might be at vital risk during anesthesia.² Fear about these events has caused utilization of different instruments to prevent it.³ Patient monitoring has been a key aspect of anesthesiology since its beginning as a medical specialty as anesthesiology has grown more sophisticated and complex, so have the monitors and data that they produce.⁴ Nowadays, the acid-base monitoring is only done in special occasions and not routinely for all patients with general anesthesia, and its necessity has not been assessed so far.

Since the potential complications of acid-base disturbances are serious and assessment in critical occasions is impossible, we decided to explore these
changes and related causes so that prevention and compensation would be feasible.

Materials and Methods

In this descriptive study, sixty 15-50 years old ASA Class I patients who came to the operating room of Baqiyatallah (a.s) hospital and were predicted to have more than 2 hours of surgery were enrolled. They were divided into three surgery groups: head and neck, extremities and abdomen with 20 patients in each. Patients with a change in blood pressure (BP) more than 30% of base, intestinal readiness before surgery, nasogastric tube, blood transfusion during surgery, with limbs tourniquet or a need to decrease Paco2 below 35 mmHg were excluded. The patients were examined and visited the night before surgery and their consent was taken.

The monitoring of ECG, BP, and pulse oxymetry (Rs232 instrument) were done after the patient was transferred to the surgical bed. For all patients, the Drager Sulla 909 anesthesia machine equipped with capnograph was used. All the patients received a narcotic premedication based on their need, and then a thermometer probe with a disposable cover was put in the nasopharynx. The patients’ basic central temperatures were recorded as the zero time temperature. Anesthesia was induced using sodium thiopental (5 mg/kg), and succinyl choline (1.5 mg/kg) to facilitate intubation. Maintenance of anesthesia was done with halothane (proportionate with the patients’ hemodynamic status), N2O (50%) and O2 (50%). Pancuronium was used to provide muscular paralysis during surgery based on the patient’s need. Respiration was controlled to keep the ventilation based on ETCO2 between 30-40 mmHg (PaCO2=35-45 mmHg). All the patients had normal ventilation and any hyper/hypoventilation was prevented. BP was recorded on arrival to the operation room, before and after induction of anesthesia, after intubation, and then every 5 minutes. Temperature was monitored continuously, before and after induction of anesthesia, and then every 15 minutes. They were recorded in a questionnaire by the researcher or a colleague.

The amount and type of the fluids that patients received at each hour were recorded based on kg/h. In the 2nd hour of anesthesia, every 1 hour, an arterial blood sample was taken to be analyzed for blood gases and assessed for acid-base status and the results were recorded in the questionnaire. Metabolic acidosis was considered if pH<7.35, Hco3<20 mmol/l and BE<-2 meq/l in arterial blood; and if pH<7.2 or BD>7 meq/l. The patient’s acidosis was corrected with sodium bicarbonate as follows:

0.2×deviation in plasma bicarbonate concentration×weight (kg)=sodium bicarbonate deficit (meq).

Half of the calculated dose was administered as intravenous sodium bicarbonate in 15 minutes and the administered bicarbonate dose was recorded in the questionnaire as meq/kg. Non-parametric statistical tests such as Kruskall-Wallis, McNemar and Chi-Square (exact as needed) were used to analyze the data. A P value less than 0.05 was considered significant.

Results

Among the 60 patients enrolled, 2 cases of head and neck group were excluded because of unpredicted need to hypotension more than 30%; thus, 58 patients were finally assessed.

The patients were 35 males (60.3%) and 23 females (39.7%). The average surgery time was 3 hours and 11 minutes (SD=37 min). There was no significant difference between the duration of different surgical operations (P>0.05). There was a 0.42°C decrease in body central temperature of basic, 2 hours after the initiation of operation, while it was 0.39 and 0.27 at the third and fourth hours after operation, respectively. Although temperature decreased according to the time, it did not follow any special pattern. At the second, third and fourth hours, 37, 42, and 11 patients received normal saline and 21, 10, and 3 persons received ringer solution. No significant difference was seen between patients who received normal saline, ringer or a combination of both (P>0.05).

Thirty six patients (62%) were affected with acidosis until the 2nd hour and 22 (38%) had no change in pH. Forty height patients (83%) were affected with acidosis until the 3rd hour and 48 (83%) until the 4th hour. There was a significant difference in the incidence rate of acidosis in the 2nd and 3rd hours after anesthesia (P<0.001).

Seventeen patients (85%) underwent abdominal surgery and developed acidosis until the 2nd hour while 4 and 15 (22% and 75%) patients undergoing head and neck and extremities surgeries were affected respectively.

There was a significant difference between the type of surgery and acidosis (P<0.001) (Table 1). This difference was between head and neck surgery group with two others but no difference between those two other groups was discovered.
Nineteen patients (95%) undergoing abdominal surgery developed acidosis until the 3rd hour after the initiation of the surgery while 12 and 17 (67% and 85%) patients undergoing head and neck and extremities surgeries developed acidosis until this time, respectively.

There was no significant difference between the location of surgery in relation to acidosis until the 3rd hour after initiation of the surgery ($P=0.077$) (Table 1).

Only one patient (5%) with abdominal surgery did not develop acidosis until the 4th hour while 6 and 3 (33% and 15%) from head and neck and extremities surgery groups were so, respectively. There was a significant difference between the location of surgery in relation to acidosis until the 4th hour ($P=0.007$) (Table 1).

There was a significant difference between the location of surgery and the need to bicarbonate in the 2nd hour ($P=0.017$) (Table 2). A significant difference was observed between head and neck surgery with the two others but no difference was noticed between those two groups. Sixteen patients (80%) with abdominal surgery and 15 patients (75%) with surgery on extremities required to receive bicarbonate until the 3rd hour, while only 9 patients of the third group (50%) were so.

There was no significant difference between the need to bicarbonate and the kind of surgery until the 3rd hour ($P=0.105$) (Table 2). Sixteen patients (80%) in each of the abdominal and extremities surgery groups needed bicarbonate until the 4th hour while only 10 patients (55%) in head and neck group did so. There was no significant difference between the need to bicarbonate and the type of surgery in the 4th hour ($P=0.56$) (Table 2).

From 48 patients (83%) affected with hypothermia, 27 patients (56%) required bicarbonate until the 2nd hour and 21 (44%) did not require it. From 10 patients (17%) not affected with hypothermia, only 2 (20%) required bicarbonate and 8 (80%) did not. Although a difference was visible but statistical tests showed no difference. There was no significant correlation between the body central temperature and acidosis at the 2nd and 3rd hours ($P=0.073$). Twenty nine patients (50%) at the 2nd hour needed bicarbonate and the same number did not. There was no significant correlation between the intake of fluids until the 2nd hour and the need to bicarbonate ($P=0.352$).

Forty patients (68%) needed bicarbonate until the 3rd hour and 18 (32%) did not. There was no significant correlation between the intake of fluids until the 3rd hour and the need to bicarbonate ($P=0.438$). Forty two patients (72%) needed bicarbonate until the 4th hour while 16 (28%) did not. There was no significant correlation between the intake of fluids until the 4th hour and the need to bicarbonate ($P=0.203$).

**Discussion**

This study showed that the patients undergoing inhalation anesthesia are affected with metabolic acidosis which has a relation with the surgery duration. As duration increases, pH decreases significantly. Also, the prevalence of these changes has a relationship with the location of surgery. These changes are significantly higher in abdominal and extremities surgeries than head and neck surgery.

We could not find any study about acid-base changes during general anesthesia in the related literature. However, the contributing factors on acid-base disturbances in conscious patients were assessed in some studies. Kulkarni and Webster found that metabolic alkalosis is the most common acid-base abnormality in very sick patients. They quoted from

<table>
<thead>
<tr>
<th>Location of surgery</th>
<th>Abdomen No. (%)</th>
<th>Extremities No. (%)</th>
<th>Head and neck No. (%)</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>2nd</td>
<td>13 (65.0)</td>
<td>15 (60.0)</td>
<td>4 (22.2)</td>
<td>0.02</td>
</tr>
<tr>
<td>3rd</td>
<td>16 (80.0)</td>
<td>15 (75.0)</td>
<td>9 (50.0)</td>
<td>0.11</td>
</tr>
<tr>
<td>4th</td>
<td>19 (95.0)</td>
<td>17 (85.0)</td>
<td>12 (66.7)</td>
<td>0.16</td>
</tr>
</tbody>
</table>

**Table 1:** Frequency of patients developing acidosis in the 2nd, 3rd, and 4th hours after anesthesia based on the location of surgery

<table>
<thead>
<tr>
<th>Location of surgery</th>
<th>Abdomen No. (%)</th>
<th>Extremities No. (%)</th>
<th>Head and neck No. (%)</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>2nd</td>
<td>17 (85.0)</td>
<td>15 (75.0)</td>
<td>4 (22.2)</td>
<td>0.001</td>
</tr>
<tr>
<td>3rd</td>
<td>19 (95.0)</td>
<td>17 (85.0)</td>
<td>12 (66.7)</td>
<td>0.08</td>
</tr>
<tr>
<td>4th</td>
<td>19 (95.0)</td>
<td>17 (85.0)</td>
<td>12 (66.7)</td>
<td>0.08</td>
</tr>
</tbody>
</table>
another article that about 50% of the patients undergoing general anesthesia are affected with metabolic alkalosis after surgery and other acid-base changes are uncommon.  

About the kind and amount of the fluids received by patients, as it is mentioned in Miller anesthesia textbook, the administration of high volumes of normal saline causes a metabolic acidosis in patients. In the study done by Hayhoe and Bellamo, metabolic acidosis due to administration of normal saline was indicated as preoperative dose dependent. Hirota and Kuze showed that ringer lactate during resuscitation with fluids had been effective to prevent metabolic acidosis.

In the current study, no significant difference was seen between patients who received normal saline, ringer or a combination of both, being opposite the research assumption. About changes in the body central temperature and its relation with acid-base disturbances under anesthesia, in the range of normal changes in central temperature under general anesthesia, no human study has been done. In an animal study by Palacios and Alfaro with two anesthesia methods on rabbits, obvious changes were not seen in acid-base amounts between hypotherm and normotherm groups.

In another study a few years later, these two researchers showed that in hypothermia, pH and HCO₃⁻ had been decreased more, maybe due to accumulation of endogen acids in the plasma accompanied by gathering of acidic products due to the metabolism of anesthesia drugs (such as Yorthan) causing by acute renal failure, as indicated by researchers. But in the current study in which the central temperature reduced 42% °C, 39% °C, and 27% °C after 2nd, 3rd, and 4th hours, respectively, the changes did not follow a definite pattern and the relation between hypothermia and developing acidosis was not significant. This might be due to a decrease in the rate of metabolism 8% for each 1°C decrease in temperature, being frequently indicated in textbooks.

We thought that higher prevalence rate of acidosis in abdominal surgeries is due to intestinal exposure followed by an increased evaporation and loss fluids to the third space and the higher need to parenteral fluids to keep hemodynamics followed by a more decreased temperature in this group; however, statistical tests did not prove it.

Since the relation between acidosis and changes in central temperature, kind and amount of received fluids was not significant, we suppose that other factors except the above mentioned factors contribute to more acidosis in abdominal surgery group. So, in inhalation anesthesia method (inhalation of Halothane and N₂O), metabolic acidosis is a common result and acidosis cases requiring the treatment with bicarbonate are abundant.

Thus, we suggest that acid-base monitoring is done in prolonged surgical operations. Obviously the above mentioned acid-base changes occur during inhalation anesthesia method, i.e. using N₂O, O₂, and inhalative Halothane. Therefore, acid-base changes in other anesthesia methods such as TIVA, spinal, epidural and/or anesthesia with other inhalation drugs require another investigation.

Acknowledgements

We wish to thank the Office of Vice Chancellor of the university for financial support of this study.

Conflict of interest: None declared.

References