# Inadvertent severe hypercarbia associated with anesthesia machine malfunction in one cat and two dogs

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- Severe hypercarbia can develop for various reasons and may go undetected during anesthesia.
- Machine malfunction causing hypercarbia can lead to delayed recovery as well as cardiac arrest.
- Routine monitoring of end-tidal carbon dioxide tension during anesthesia will aid in detection of adverse changes in carbon dioxide tension, thereby averting related complications.

A 7-month-old 2.9-kg (6.4-lb) female Maine coon kitten was hospitalized for a ventral bulla osteotomy. Physical examination findings were normal except for the presence of chronic otitis. The Hct was 34%, and serum total protein concentration was 7.2 g/dl. The kitten was premedicated with glycopyrrolate (0.01 mg/kg [0.005 mg/lb] of body weight, IM), oxymorphone hydrochloride (0.1 mg/kg [0.046 mg/lb], IM), and acepromazine (0.05 mg/kg [0.021 mg/lb], IM). One hour later, anesthesia was induced with ketamine hydrochloride (5 mg/kg [2.1 mg/lb], IV) and diazepam (0.25 mg/kg [0.11 mg/lb], IV). Anesthesia was maintained with isoflurane (1.25 to 2.5%) in oxygen. The kitten was positioned in dorsal recumbency and intubated, and the endotracheal tube was connected to a pediatric semiclosed circle anesthetic machine. A warm air blower[a] was placed over the kitten to maintain the body temperature between 98 and 100 F. Monitoring equipment consisted of a Doppler probe[b] and sphygmomanometer to measure blood pressure, an ECG to monitor heart rate and rhythm, a pulse oximeter to measure oxygen saturation of hemoglobin, and an esophageal temperature probe.

Intraoperatively, the oxygen saturation of hemoglobin remained at 100% throughout the anesthetic period. The spontaneous respiratory rate varied between 10 and 20 breaths/min and was supplemented with manual positive pressure ventilation 2 to 3 times per minute. Systolic blood pressure, as read by the sphygmomanometer, was between 50 and 75 mm Hg. Lactated Ringer's solution was administered at 10 ml/kg/h (4.5 ml/lb/h). Dopamine (7.5 μg/kg/min [3.4 μg/lb/min], IV) was infused before surgery and was increased to 10 μg/kg/min (4.6 μg/lb/min) throughout surgery to support blood pressure. Heart rate increased from 170 to 200 beats/min after the start of surgical drilling and remained high for the duration of anesthesia. The concentration of isoflurane was increased, and oxymorphone (0.05 mg/kg [0.021 mg/lb], IV) was administered at this time and again 1 hour later. Systolic blood pressure remained around 75 mm Hg. Surgery lasted for 2 hours, and anesthesia lasted for 2 hours and 40 minutes.

On completion of surgery, isoflurane and dopamine were discontinued, and the kitten was transported to the recovery area while still connected to the anesthesia machine. At this point, the kitten would not breath spontaneously, the sound from the Doppler probe was weak, blood pressure could not be determined with the sphygmomanometer, and the pupils were fixed and dilated. A blood sample was drawn from the medial saphenous vein and analyzed[c] for pH (6.6), partial pressure of oxygen (99 mm Hg), and partial pressure of carbon dioxide ($P_{CO_2}$; 264 mm Hg); these measurements were repeated immediately to confirm that the kitten had severe hypercarbia stemming from respiratory acidosis (pH 6.72; $P_{O_2}$, 172 mm Hg; $P_{CO_2}$, 195 mm Hg; bicarbonate, 24 mmol/L; base excess, –17 mmol/L). Intermittent positive pressure ventilation with 100% oxygen was instituted at a rapid rate, an infusion of dopamine (5 μg/kg/min [2.1 μg/lb/min], IV) was restarted, dextran (2 ml/kg [1 ml/lb], IV) was administered as a bolus, prednisone (10 ml/kg [4.6 ml/kg], IV) was administered, and mannitol (2 g, IV) was administered over 20 minutes. The warm air blower was replaced over the kitten to maintain normothermia. Subsequent blood samples were drawn from the medial saphenous vein or the femoral artery, and substantial increases in pH and decreases in $P_{CO_2}$ were detected over time (35 minutes into recovery: venous pH 6.78; $P_{vCO_2}$, 182 mm Hg; $P_{vO_2}$, 122 mm Hg; bicarbonate, 25 mmol/L; base excess, –14.1 mmol/L; 45 minutes: arterial pH 7.15; $P_{aCO_2}$, 64 mm Hg; $P_{aO_2}$, 553 mm Hg; bicarbonate, 21 mmol/L; base excess, –7.4 mmol/L).

Thirty minutes after discontinuing the anesthetic, the kitten recovered awareness. Diazepam (0.2 mg/kg [0.1 mg/lb], IV) was administered to maintain sedation and maintain placement of the endotracheal tube until another saphenous venous blood sample could be drawn to ensure return of $P_{CO_2}$ toward normal pressure. Ninety minutes after discontinuing the anesthetic, the venous $P_{CO_2}$ decreased to 53 mm Hg (pH 7.2; $P_{O_2}$, 47 mm Hg; bicarbonate, 20 mmol/L; base excess –7.3 mmol/L), and the kitten was breathing spontaneously and was extubated. Breathing continued without complication. It was evident that the kitten had vision, though anisocoria with miosis of the right pupil was noticed. The kitten had some signs of disorientation, which resolved over the next 3 days.

A 6-year-old 37.5-kg (82.5-lb) male castrated

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Greyhound was admitted for a thoracotomy and repair of chylothorax. On physical examination, the dog had mild signs of depression and was tachypneic, with a respiratory rate of 32 breaths/min. Results of a CBC were within reference ranges, although the dog was mildly hypoproteinemic with a total protein of 5.1 g/dl. Thoracocentesis was performed prior to induction of anesthesia, and approximately 2 L of effusive fluid was withdrawn. The dog was medicated with glycopyrrolate (0.01 mg/kg [0.0046 mg/lb], IM), midazolam hydrochloride (0.1 mg/kg [0.046 mg/lb], IM), and hydromorphone (0.1 mg/kg [0.46 mg/lb], IM). Fifty minutes later, anesthesia was induced with ketamine hydrochloride (10.7 mg/kg [5 mg/lb], IV) and diazepam (0.27 mg/kg [0.1 mg/lb], IV). Anesthesia was maintained with isoflurane (1 to 1.75%) in oxygen delivered through a semiclosed circle anesthetic machine. The dog was monitored with a Doppler probe over a metacarpal artery, an ECG, a pulse oximeter, and a temperature probe; an arterial catheter was inserted to measure blood pressure and to draw blood samples for analyses.

Intraoperatively, dopamine was administered intravenously to maintain the mean blood pressure above 60 mm Hg. Three intercostal nerves cranial and 2 nerves caudal to the incision were blocked with bupivacaine hydrochloride, and oxymorphone hydrochloride (0.04 mg/kg [0.02 mg/lb], IV) was administered. Intermittent positive pressure ventilation was instituted prior to the surgical incision, with the ventilator set to deliver a tidal volume of 10 ml/kg at a rate of 10 breaths/min. Ten minutes after the chest cavity was open (1 hour and 20 minutes after anesthetic induction), an arterial blood sample was analyzed.[d] The mechanical respiratory rate was then increased to 12 breaths/min based on a $Paco_2$ of 56 mm Hg (pH 7.27; $Pao_2$, 314 mm Hg; bicarbonate, 26 mmol/L; base excess, –1 mmol/L). One hour and 40 minutes later, the dog continued to require controlled mechanical ventilation, and another blood gas analysis revealed the $Paco_2$ was 36 mm Hg (pH 7.39; $Pao_2$, 373 mm Hg; bicarbonate, 22 mmol/L; base excess, –3 mmol/L). At the end of surgery, the respiratory rate on the ventilator was decreased to 10 breaths/min for 25 minutes and then to 5 breaths/min for 15 minutes until the dog began breathing spontaneously. Surgery had taken 3 hours, and the dog was anesthetized for 4 hours. The chest cavity was aspirated of air until negative pressure was attained in the syringe. Bupivacaine and sterile saline (0.9% NaCl) solution were administered intrapleurally for postoperative analgesia. Isoflurane was discontinued, and morphine (0.1 mg/kg [0.046 mg/lb]) was diluted to 6 ml with saline solution and injected into the epidural space.

A blood sample was analyzed approximately 10 minutes after the dog began breathing spontaneously and severe respiratory acidosis was present; $Paco_2$ was > 130 mm Hg (pH 6.94; $Pao_2$, 425 mm Hg). The anesthetic machine was immediately changed, and the dog was manually ventilated vigorously. After 35 minutes, another blood sample analysis revealed the $Paco_2$ (56 mm Hg) was much closer to reference limits (pH 7.29; $Pao_2$, 629 mm Hg; bicarbonate, 27 mmol/L; base excess, 1 mmol/L). The dog did not recover awareness or spontaneous ventilation for 2.5 hours. During this time, respiratory acidosis was mild, and the $Paco_2$ was maintained below 65 mm Hg (pH 7.19; $Pao_2$, 529 mm Hg; bicarbonate, 23 mmol/L; base excess, –5.8 mmol/L). The dog was given prednisone (1,125 mg, IV) and mannitol (37.5 g, IV). After recovery, the dog was placed in the intensive care unit for monitoring and maintained normal $CO_2$ concentrations with spontaneous ventilation.

A 3-month-old 10.8-kg (23.8-lb) male mixed-breed dog was admitted for routine castration. Physical examination findings were normal. The Hct was 34%, BUN was < 15 mg/dl, and total protein concentration was 5.8 g/dl. The dog was medicated with glycopyrrolate (0.01 mg/kg [0.0046 mg/lb], IM), morphine (0.2 mg/kg [0.1 mg/lb], IM), and acepromazine (0.05 mg/kg [0.021 mg/lb], IM). One and a half hours later, anesthesia was induced with sodium thiopental (15 mg/kg [7 mg/lb], IV) and maintained with isoflurane (1.3 to 2%) in oxygen. The dog was monitored with an ECG, a Doppler probe, a pulse oximeter, and an indirect oscillating blood pressure cuff.[e] Dopamine (2-10 μg/kg/min [1-5 μg/lb/min]) was administered intravenously to maintain the mean arterial blood pressure above 60 mm Hg. The oxygen saturation of hemoglobin remained between 93 and 100% throughout surgery, and respiration was spontaneous. The surgery lasted 40 minutes, and the duration of anesthesia was 67 minutes.

Isoflurane was discontinued at the end of surgery. Approximately 10 minutes later, the dog was still breathing spontaneously, monitoring equipment was disconnected, and the anesthesia machine was removed. A few minutes later, the student anesthetist noticed that the dog had stopped breathing, and a pulse could not be detected. Cardiac compressions were immediately started, and the dog was reconnected to the anesthetic machine for intermittent positive pressure ventilation. Atropine (0.02 mg/kg [0.01 mg/lb]) and epinephrine (0.2 mg/kg [0.1 mg/lb]) were administered intravenously. The pulse returned, and the ECG revealed sinus tachycardia. A jugular venous blood sample was drawn, and analysis revealed severe respiratory acidosis, with a $Pco_2$ of > 130 mm Hg (pH 6.7; $Pvo_2$, 77 mm Hg). The anesthetic machine was changed, and the dog was mechanically ventilated for 20 minutes, after which the $Pco_2$ was 49 mm Hg (pH 7.24; $Pao_2$, 245 mm Hg; bicarbonate, 20 mmol/L; base excess, 6.7 mmol/L). The dog began breathing spontaneously and was extubated 15 minutes later. Recovery was subsequently without complications.

Hypercarbia can occur during anesthesia from hypoventilation,[1] hypermetabolic states,[2] an exhausted soda lime canister,[3] or equipment failure, which allows rebreathing of expired $CO_2$.[3] Hypercarbia may initially cause increased arterial blood pressure and heart rate because of sympathetic stimulation.[4] Cardiac arrhythmias[5] and respiratory acidosis can also occur and eventually lead to vasodilation and decreased cardiac output,[4] CSF acidosis, and carbon dioxide narcosis,[6] which leads to a deeper plane of anesthesia, delayed recovery, and death. Hypercarbia may be detected during anes-

thesia by clinically assessing minute ventilation, but actual determination requires monitoring of end-tidal $CO_2$ tension or analyzing blood for $CO_2$ tension.

The cause of hypercarbia was investigated in each of the animals of this report. Hypoventilation was not clinically evident. In the Greyhound, ventilation was deemed adequate throughout surgery based on the known minute ventilation and blood gas values. In the kitten and the mixed-breed dog, visual estimations were made that spontaneous ventilation was adequate or close to adequate, requiring minimal augmentation. The kitten was placed on a rebreathing system with a low fresh gas flow to help maintain body temperature. The potential increased work of breathing was minimized with periodic supplemental manual ventilation throughout the anesthetic period.

As a cause of hypercarbia, equipment failure was addressed. Immediately after surgery, the anesthesia machine used in each of these animals was inspected by an anesthesiologist and an anesthetist. In no instance was any fault detected. The soda lime in the $CO_2$ canister was fresh. One-way valves were in the appropriate place and on inspection appeared to be functioning in the circle system. No obstruction could be found in the system, and the flow meters were functioning properly and were delivering fresh gas into the breathing circuit.

On further investigation, although anesthesia was administered by different anesthesiologists and anesthetists, only 1 anesthetic machine was used in all 3 animals. The machine[f] was older than the others in use. The machine was inspected once again by the authors, and on closer scrutiny it was found that the mounted disk, which serves as the exhalation valve in the circle system, appeared to be slightly warped, and 1 side was not seated across the entire opening, leaving a gap of approximately 2 mm. To test the system, the inspiratory limb was manually obstructed intermittently while a person breathed through the exhalation limb. The exhalation valve did not seat during shallow respiratory movements, and shallow nonforceful inspiration drew gas through the exhalation limb of the circuit directly from the rebreathing bag, thus bypassing the $CO_2$ absorber completely. The valve would seat completely and prevent bidirectional flow when large forceful breaths were attempted, providing for ventilation through the circle as intended.

In the kitten and mixed-breed dog, spontaneous ventilation would have contributed to rebreathing $CO_2$ through the expiratory limb of the machine. In the Greyhound, the arterial $CO_2$ tension was found to be close to normal limits while the patient was being ventilated mechanically by the ventilator, but significant hypercarbia with a $PaCO_2$ of greater than 130 mm Hg was attained during spontaneous ventilation. Because the peak inspiratory flow would be much higher during mechanical ventilation, the pressure generated would close the exhalation port and permit this patient to be ventilated with functioning 1-way valves. When the patient inspired spontaneously, the exhalation valve did not seal completely, and inspiration came through the expiratory limb.

The valves in this particular machine are attached vertically, appear to be made of a rubber type of material, and are not easily detached from the machine (Fig 1). A slight warping of the valve could easily result in the valve remaining in the open position unless a critical pressure gradient was generated across the valve. The other machines that are available in our institution have horizontally placed valves that are made of metal. Although these valves can stick, particularly when moist, they are less likely to develop the complication

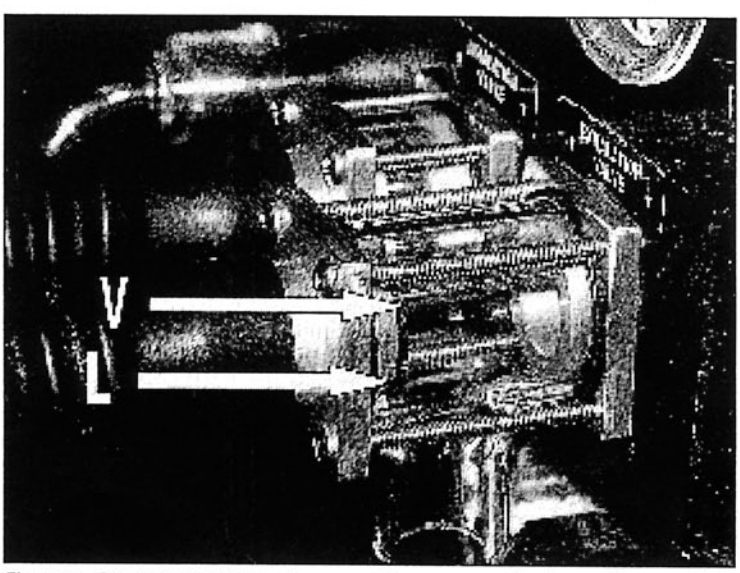


Figure 1—Photograph of the valve assembly on an anesthesia machine used in 1 cat and 2 dogs that developed severe hypercarbia. The exhalation assembly has a port for the rebreathing bag on the lower aspect and is attached to the rubber circle system hose on the left. The black rubber exhalation flutter valve (V) is closely situated against the airway port on the left. The warping on the exhalation valve is slight and very difficult to notice. The valve is not flat against the port, and the space causing valve incompetence is marked with L (leak).

that occurred in these cases. Anesthetic machines should be maintained in functional condition with the system checked for leaks and the valves cleaned, dried, and replaced in working order.

Hypercarbia can be detected most directly by blood gas analysis. Mixed-venous $CO_2$ concentrations are usually 3 to 6 mm Hg higher than arterial concentrations and can reflect a respiratory acidosis when arterial samples are not available. Because venous $CO_2$ is representative of tissue concentrations, hypotension or decreased perfusion to a tissue bed will increase the venous $CO_2$, compared with the arterial; this must be kept in mind when a peripheral vein is sampled.[7] The kitten of this report had samples drawn from the medial saphenous vein, and the $P_{CO_2}$ would be expected to be slightly higher than if obtained from an artery. However, the severity of $P_{CO_2}$ increase seen in this patient, compared with normal, was indicative of a severe respiratory acidosis. The disadvantage of routine blood gas analysis to detect severe hypercarbia is that sampling is usually sporadic and may not catch sudden changes at the time they occur. Also, many practices do not have blood gas machines, although the small portable units are becoming increasingly common. Methods of assessing minute ventilation, such as the use of respirometers, will determine whether low minute volumes are occurring but will not detect an increase in $P_{CO_2}$ that is not related solely to inadequate ventilatory mechanics. Monitoring of end-tidal $CO_2$ will, in most instances, detect hypercarbia. End-tidal $CO_2$ can be used to estimate $Paco_2$ continuously, although it is always slightly lower.[7] The degree to which it is lower is usually clinically insignificant in healthy small animals. Selection of the appropriate type of $CO_2$ analyzer is important to avoid a substantial increase in dead space in small patients. An increase in end-tidal $CO_2$ can signal hypercarbia that may be attributable to various problems such as hypoventilation, a considerably increased metabolic state, or equipment malfunctions. Alternatively, a sudden decrease in end-tidal $CO_2$ may be seen in a patient with a substantial decrease in cardiac output, pulmonary embolus, or inadvertent dislodging of the endotracheal tube.[8]

Although monitoring end-tidal $CO_2$ is routinely performed in our institution, it is not always used in patients < 10 kg (22 lb), because the mainstream capnogram we use could add considerable equipment deadspace to the airway and therefore allow for increased rebreathing of $CO_2$. When a nonrebreathing system is used, the high oxygen flows required would decrease the end-tidal $CO_2$ values registering on the monitor, which would therefore read inaccurately low. The end-tidal $CO_2$ will variably decrease from $Paco_2$ during a thoracotomy and, thus, not predictably reflect modest changes in arterial $CO_2$ tension.[9] In each of these situations, however, the capnogram could be used intermittently, and though the $Paco_2$ could not be estimated accurately, an excessively high end-tidal $CO_2$ would indicate an excessively high $Paco_2$. The application of capnography in our patients reported here would have immediately detected hypercarbia and allowed for rapid correction without physiologically stressing the patients.

Carbon dioxide is a respiratory stimulant and can induce tachypnea. At high concentrations (> 90 mm Hg), it is a cerebral depressant and will depress the ventilatory drive.[6] When the patient is anesthetized, however, cardiovascular and respiratory signs of hypercarbia can be masked, making it more difficult to make a clinical diagnosis. Use of vasopressors will also mask the cardiovascular response to hypercarbia. Under these circumstances, extreme hypercarbia may occur without the clinician being aware of the problem until recovery is attempted or cardiovascular collapse occurs.

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[a]Bair Hugger, Augustine Medical Inc, Eden Prairie, Minn.
[b]Parks Medical Electronics Inc, Aloha, Ore.
[c]Radiometer, Winter Park, Fla.
[d]ISTAT Corp, East Windsor, NJ.
[e]Datascope, Paramus, NJ.
[f]Anesco, Waukesha, Wis.

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