and should decrease the chances of overinflation of the bronchial cuff.

Other recommendations to protect against tracheobronchial rupture during the use of double-lumen tubes include removing the stylet after the tip of the tube is passed through the cords, deflating the tracheal and bronchial cuffs when repositioning the patient or the tube and inflating the bronchial cuff only during one-lung ventilation. If the bronchial cuff fails to seal with 2–3 ml of air, the size and position of the tube should be reassessed. One should also check the integrity of the intubated bronchus with the bronchial cuff deflated at the time of testing the resected bronchus for air leaks. If N_2O is used during bronchial cuff inflation, frequent checking of the balloon pressure has been recommended.

Bronchial rupture associated with the use of the PVC double-lumen tubes is a serious potential complication. Extreme care with positioning and bronchial cuff inflation is, therefore, needed. Selecting the appropriate tube size for a given patient may require more accurate estimation by calculating the diameter of the bronchus to be intubated from PA and lateral chest x-rays. The appropriate tube size for a given bronchial diameter remains to be determined.

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Inadvertent Development of Subatmospheric Airway Pressure during Cardiopulmonary Bypass

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Gas sampling devices are widely used to monitor both anesthetic and respiratory gases during the perioperative period. Although misinformation derived from monitoring artifacts[1,2] may lead to errors in patient management, devices to qualitatively analyze gases are generally viewed as safe, posing little risk of physical injury to the patient. We describe here a circumstance where a noninvasive monitor, *via* its mechanical sampling function, created a potential hazard for a patient during cardiopulmonary bypass.

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Case Report

A 57-yr-old man, scheduled for coronary revascularization and mitral valvuloplasty, was monitored prior to induction *via* radial and pulmonary artery catheters, electrocardiography, and pulse oximetry. After induction of general anesthesia and tracheal intubation, additional respiratory monitors were applied, including a time-shared mass spec-

TABLE 1. Flow Rates of Commonly Used Gas Sampling Devices

Gas Sampling Devices	Gas Sampling Rate (ml/min)
Albion®	less than 200
Biochem® (all models)	120
Criticare®	50, 150 (user selectable)
Datascope® (Accucap)	50, 150 (user selectable)
Datascope® (Multinex)	50, 100, 150, 200 (user selectable)
Diatek® (Model 223)	50, 150 (user selectable)
Diatek® (all other models)	150
Drager® (Capnomed)	150
Drager® (Multispec)	200
Engstrom® (all models)	100
Marquette® (Model 7060)	150
Marquette® (Advantage 2000)	250
Nellcor®	50
Novametrix® (Model 1250)	50, 150 (user selectable)
Ohmeda® (Model 5200)	150, 300 (user selectable)
Ohmeda® (RGM)	200
PPG Saracap®	120, 240 (user selectable)
PPG Saracap® Plus	330 (when room is selected) 230 (with another room selected)
Sensormedics® (Model LB-2)	continuously variable between 100 and 800
Sensormedics® (Model LB-3)	continuously variable between 30 and 500
Spacelabs®	200
Teledyne®	continuously variable between 0 and 200
Traverse® (220)	continuously variable between 75 and 400
Traverse® (3000)	continuously variable between 100 and 300

Sampling rates listed are nominal values provided by manufacturers. Actual rates may vary substantially, depending upon the status of the gas sampling device (see text).

trometer (Marquette, St. Louis, MO) in series with an infrared capnograph (Lifewatch), and a volumeter and airway pressure monitor, both on the expiratory limb of the anesthesia machine (Ohmeda Modulus II®, Madison, WI).

After cardiac exposure and cannulation of the aorta and vena cavae, cardiopulmonary bypass (CPB) was instituted. Mechanical ventilation was discontinued, the bag-ventilator selector switch was placed in the bag/APL (adjustable pressure limiting) position, and fresh gas flow (FGF) into the breathing circuit was reduced from 2000 ml/min to 50 ml/min (minimal oxygen flow setting of the anesthesia machine). The pulse oximeter and expiratory volumeter were turned off, and the patient display unit of the mass spectrometer was placed in the off-line mode.

Approximately 30 min later, the anesthesiologist noted that the reservoir bag in the breathing circuit was tightly collapsed and that pressure on the airway manometer exceeded the maximal subatmospheric pressure measurable by the gauge (−20 cm H_2O). To release this subatmospheric pressure, the breathing circuit was disconnected from the patient; this produced a noise (interpreted as turbulent air entrainment) and an immediate return of airway pressure to 0 cm H_2O. At this time, we checked the scavenging system. Gas flow from the breathing circuit to scavenging system was unobstructed, and disc valves that relieve positive or negative pressure in the scavenging system had normal freedom of movement (checked *via* the interface relief valve button). Next, we increased fresh gas flow (FGF) of oxygen to 300 ml/min, and the remainder of CPB was uneventful.

Weaning from CPB proceeded uneventfully, positive pressure ventilation was resumed, and respiratory monitoring restored. Postbypass arterial blood gases, respiratory mechanics, and chest radiographs were consistent with the usual postoperative state; there was no evidence of pulmonary edema. The patient was weaned from mechanical ventilation and circulatory support on the first postoperative day and discharged home within the week.

Of note, during a postoperative discussion, the surgeons mentioned that shortly after initiation of CPB, the heart and great vessels had retracted into the left hemithorax. (The right pleural space had been entered earlier.) They recalled that entry into the left pleural space produced a loud, popping sound and rapid return of mediastinal structures to the midline.

DISCUSSION

Subatmospheric airway pressure in the anesthesia breathing circuit was noted during CPB. This resulted from gases being removed from the circuit at a greater rate than they were being added.[3] The possibility that malfunction of the negative-pressure-relief valve on the gas-scavenging system contributed to gas removal was immediately investigated and dismissed as a cause. The problem occurred because gas removal by the mass spectrometer continued at a rate of 250 ml/min (even though the patient display unit was placed in the off-line mode) while FGF of O_2 added to the circuit was only 50 ml/min. Thus, there was a net gas loss of 200 ml/min, and subatmospheric pressure developed after the residual volume of the reservoir bag was evacuated.

This problem prompted us to investigate various respiratory gas sampling devices. Some devices sample at fixed rates; others allow the user to select the rate (table 1). Sampling rates of commonly used devices vary widely, from less than 50 ml/min to as high as 800 ml/min. With any particular device, however, sampling rate may vary depending upon status of alarm system, calibration, operating room being sampled *via* time-shared unit, or mode (standby or off-line). It may be necessary to contact the manufacturer to obtain information about sampling rates in the standby or off-line mode. Most stand-alone units having a standby option (*e.g.*, PPG, Ohmeda, Albion, and Puritan Bennett) discontinue sampling in this mode. Conversely, both commercially available time-shared mass spectrometers (Marquette and PPG) continue to aspirate gas in the standby or off-line mode.

What role does an APL or ventilator-relief valve play in the development or dissipation of subatmospheric pressure in the patient breathing circuit (refer to fig. 1)? High subatmospheric pressure in the scavenging system (*e.g.*, a faulty negative-pressure-relief valve with evacuation hose connected to house vacuum) would open the APL valve, introducing subatmospheric pressure into the breathing circuit. On the other hand, excess gas removal from the breathing circuit (*e.g.*, *via* a gas sampling device) would create subatmospheric pressure in the breathing circuit, thereby closing the APL valve. This in theory

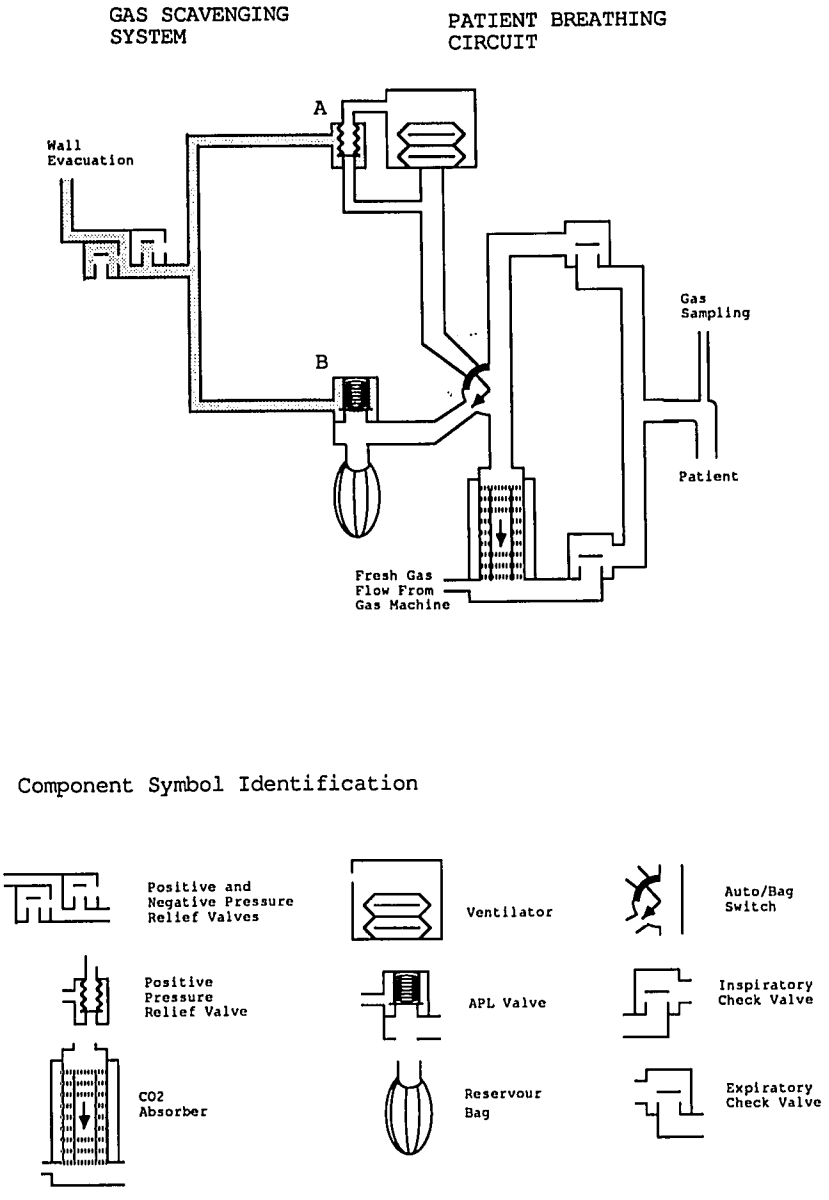

FIG. 1. Breathing circuit (clear) and scavenging system (stippled) are separated by the ventilator-relief valve (*A*) or APL valve (*B*). These valves, when functioning properly, facilitate development of subatmospheric pressure in the breathing circuit; they allow subatmospheric pressure generated in the scavenging system to enter the breathing circuit and prevent the scavenging system from dissipating subatmospheric pressure generated in the breathing circuit. Abnormalities of these valves, however, may relieve subatmospheric pressure (see text).

should prevent the scavenging system from relieving subatmospheric pressure in the breathing circuit. Large pressure gradients, however, may distort or unseat APL or ventilator-relief valves, allowing gas flow from scavenging system to breathing circuit, thus reducing the pressure gradient.

Large subatmospheric pressures in the breathing circuit are unlikely to develop during low-flow or closed-circuit anesthesia because manual ventilation mandates an awareness of the volume of the reservoir bag, and mechanical ventilation, in compliance with ANSI Z79 standards, incorporates protective alarms (minimal pressure, subatmospheric pressure, and low-minute volume [apnea] alarms). Current standards, however, do not require an alarm for subatmospheric pressure in the breathing circuit of a patient not ventilated mechanically (cardiopulmonary bypass). We feel that such an alarm should be present and have subsequently added one to our Ohmeda Modulus II machine. This alarm is triggered only when subatmospheric pressures exceed those normally generated by spontaneous ventilation. To be useful during cardiopulmonary bypass, the alarm must be uncoupled from the minimum pressure detection alarm to avoid a continuous alarm in the absence of positive-pressure ventilation. Such alarms are available from various manufactures, including Ohmeda and North American Drager.

How much subatmospheric pressure can be generated in a breathing circuit? Theoretically, the Marquette mass spectrometer can produce subatmospheric pressures in excess of −500 mmHg in a breathing circuit without gas

leaks. Maximum subatmospheric pressures, however, were much less when we measured them in breathing circuits with patient-connection site obstructed, mass spectrometer connected, and no fresh gas added to the system. On four anesthesia machines tested (two Ohmeda Modulus II, two North American Drager Narkomed 2), maximum subatmospheric pressures ranged from −11 to −148 mmHg; this variability was primarily a function of gas leakage past the APL or ventilator-relief valves. A similar test on the machine used in our case revealed a pressure of −130 mmHg when the selector switch was set to the reservoir bag.

Subatmospheric pressure in airways could lead to injury. For example, high subatmospheric pressures exerted on the tracheo-bronchial mucosa could produce mucosal edema, except at the site of contact with an endotracheal tube, where mucosal ischemia might occur. The physiologic impact of subatmospheric airway pressure in patients on CPB has not been characterized, but small airways might be expected to collapse, decreasing transmission of subatmospheric pressures to alveoli or pleural spaces. Nevertheless, subatmospheric intrapleural pressure did occur as evidenced by a sudden rightward shift of the mediastinum when the left pleural space was entered. Such a rapid, unexpected movement of mediastinal structures during cardiac dissection could result in vascular injury.

It is interesting to compare subatmospheric pressure observed in this case with that created by forced inspiration against a closed glottis (*i.e.*, Mueller maneuver). This maneuver can rapidly generate subatmospheric pressure and produce pulmonary edema[4,5] *via* an increase in cardiac afterload[6,7] or transpulmonary vascular pressure.[8] In our case, subatmospheric pressure probably developed slowly, and could not be transmitted to the heart or great vessels because of an open mediastinum. Also, low pulmonary blood flow during CPB minimizes the fluid transudation caused by high transmural pulmonary vascular pressures, which are theoretically possible with high subatmospheric airway pressures. Thus, the absence of postbypass pulmonary edema was not surprising.

In summary, this report highlights a potential hazard associated with noninvasive gas sampling monitors. We observed subatmospheric airway pressure during CPB, which occurred because the rate of gas removal by the sampling system exceeded FGF into the circuit. The large number of devices used to sample respiratory gases and their differing effects on the breathing circuit adds complexity to anesthesia monitoring. Ultimately, risk-free patient monitoring mandates an awareness that seemingly harmless monitors, when not thoroughly understood, may be potentially hazardous.

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