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Excessive Airway Pressure Due to Ventilator Control Valve Malfunction during Anesthesia for Open Heart Surgery

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Ventilator malfunction can be classified as failure of the breathing circuit, of the bellows assembly, or of the control assembly.[1] The most commonly cited causes for breathing circuit malfunction include leaks,[2,3] disconnections,[4,5] or misconnections[5,6] in the breathing circuits. Morbidity or mortality can result from a no-flow (no-ventilation) state resulting in hypoxia, or from excessively increased airway pressure resulting in barotrauma.[5–9]

In this case report we describe North American Drager AV-E ventilator malfunction due to a defective valve in the ventilator control assembly that regulates gas inflow to the ventilator bellows chamber. The continuous flow of gas to the bellows induced a no-ventilation state and at the same time caused the patient's airway pressure to increase to hazardous levels.

CASE REPORT

A 44-yr-old, 73-kg man was scheduled for elective coronary artery bypass surgery. Preoperatively, systemic arterial and flow-directed

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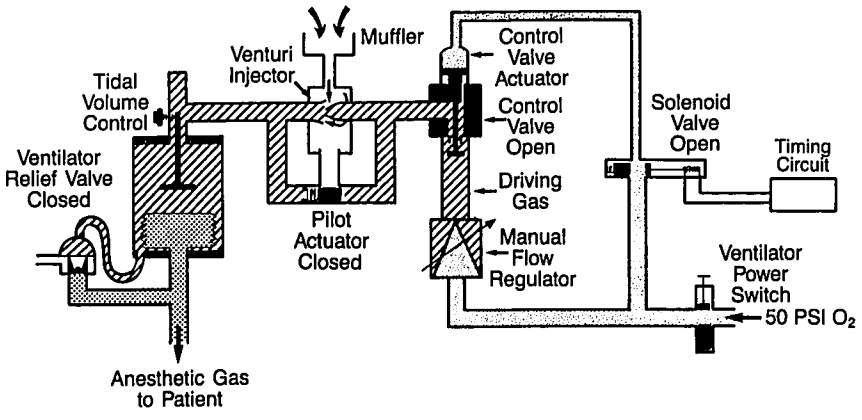

FIG. 1. AV-E ventilator operation: inspiratory phase. The ventilator timing circuit initiates mechanical inspiration by opening the solenoid valve to allow the flow of 50 psi oxygen to the control valve actuator. This flow opens the control valve. The control valve allows the driving gas to flow to the bellows chamber along with a variable amount of air entrained from the Venturi injector. The driving gas pressure is controlled by the manual flow regulator. The ventilator relief valve is closed by the influx of driving gas. This prevents anesthetic gas from escaping to the scavenger system, and causes the bellows to move downward with delivery of anesthetic gas mixture to the patient. (Figure adapted.[1,**,††])

pulmonary artery catheters were placed. The following hemodynamic values were obtained prior to induction of anesthesia: cardiac output (CO) 3.5 l/min, pulmonary artery pressure (PAP) 34/18 mmHg, pulmonary artery occlusion pressure (PAOP) 22 mmHg, and central venous pressure (CVP) 16 mmHg. Arterial blood pressure (BP) was 130/80 mmHg. The anesthesia machine was inspected preoperatively in accordance with the hospital's daily anesthesia machine inspection policy, which is similar to but also an extension of the Food and Drug Administration (FDA)-recommended guidelines. Fentanyl and midazolam were used for induction and maintenance of general anesthesia, and muscle relaxation was achieved with a combination of Metubine® iodide (Lilly) and pancuronium. Mechanical ventilation was with a North American Drager AV-E ventilator attached to a Narkomed (Telford, PA) 2A anesthesia machine. The tidal volume (TV) was set at 550 ml with a rate of 10 breaths per min. The baseline breathing circuit pressure noted on the airway pressure gauge was 20–24/2–3 cmH_2O. Peak inspiratory pressure (PIP) was 28 cmH_2O.

For 2 h postinduction, the patient's hemodynamic and ventilatory parameters were unchanged and stable. Twenty minutes prior to institution of cardiopulmonary bypass, the anesthesiologist noticed a gradual increase in PAP to 30/20 mmHg and in PIP to 33 cmH_2O. Shortly thereafter, inadequate excursion of the ventilator bellows was observed. Attempts to inflate the bellows with an oxygen flush were not successful. Since the airway pressure continued to increase (PIP was 35 cmH_2O), mechanical ventilation was discontinued and manual ventilation begun. The scavenging system and ventilator exhaust were checked for flow obstruction, but both appeared to be functioning properly. During cardiopulmonary bypass, the ventilator bellows was replaced and the anesthesia machine rechecked according to recommended FDA protocol.¶ There were no obvious malfunctions. Postbypass ventilator function was monitored closely, and for 20 min appeared normal. Ventilator settings (TV 640 ml, rate 10 breaths per min) resulted in a new baseline breathing circuit pressure of 22–25/2–3 cmH_2O and PIP of 25 cmH_2O. CO was 6.9 l/min, PAP 30/18 mmHg, CVP 11 mmHg, and arterial BP 125/65 mmHg. Shortly after this 20-min time period, PAP increased to 34/21 mmHg and CVP to 25 mmHg. The ventilator bellows were again noted to have reduced excursions and would not expand with an O_2 flush. The latter resulted in PIP of 55 cmH_2O and reduction in BP to 95/55 mmHg. The patient was disconnected from the ventilator and manual ventilation was used for the duration of the surgical procedure. Normal circulatory and respiratory function were restored, and the surgical procedure was completed successfully.

DISCUSSION

The forgoing case illustrates hemodynamic deterioration consequent to sustained high airway pressure due to apparent ventilator malfunction. Although the cause for ventilator malfunction was not immediately apparent, prompt action by the anesthesiologist to disconnect the patient from the ventilator and provide manual ventilation prevented potential lung injury.

The patient's lungs were ventilated with a 5-yr-old North American Drager AV-E ventilator. The anesthesiologist performed a routine preoperative check of the anesthesia machine. The ventilator was operated briefly with a reservoir bag attached to the Y-piece. Cycling of the ventilator was verified. All ventilator controls were rotated through their entire range and corresponding variations in ventilator function observed. The ventilator bellows assembly also was tested for leaks. No functional problems were identified.

The AV-E ventilator is classified as a pneumatically and electronically powered, double-circuit, pneumatically-driven, ascending-bellows, time-cycled, electronically controlled, tidal-volume-preset controller.[1] Its major components are the control assembly and the bellows assembly.[1] The operating principle of the AV-E ventilator is illustrated in figures 1 (inspiratory phase) and 2 (expiratory phase), which are schematics adapted from several sources.[1,**,††]

Later disassembly of the ventilator revealed the cause for failure to be malfunction of the control valve (manufactured for North American Drager by Clippard, Cin-

¶ Anesthesia apparatus checkout recommendations. Rockville, Maryland, Food and Drug Administration, August 1986. In: Andrews JJ. Understanding anesthesia machines, Review Course Lectures. San Diego, International Anesthesia Research Society, March 1988, pp 78–85

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†† Narkomed 2A Anesthesia System: Instruction Manual. Telford, PA: North American Drager, 1986

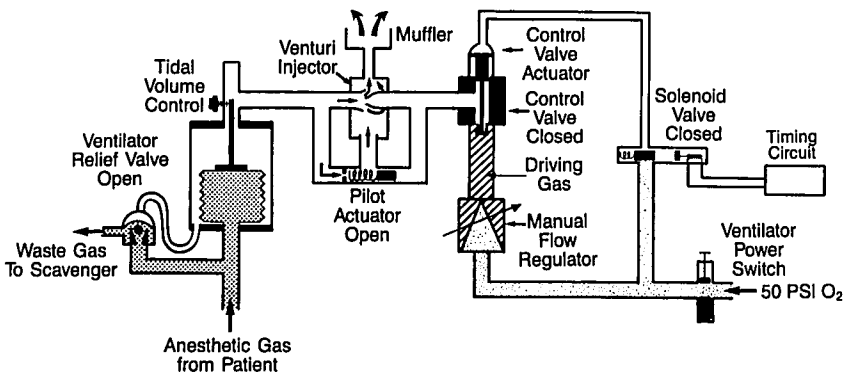

FIG. 2. AV-E Ventilator operation: expiratory phase. The expiratory phase begins with cessation of electrical current flow from the timing circuit to the solenoid valve. Closure of the solenoid valve terminates 50-psi oxygen flow to the control valve actuator. This causes the control valve to close, thereby interrupting the driving gas flow to the bellows chamber. During expiration, the driving gas exhausts in a retrograde fashion through the entrainment port of the Venturi injector device and also through the pilot actuator to the muffler. After the pressure within the bellows chamber decreases to zero, the ventilator relief valve opens permitting excess anesthetic gas to be vented into the scavenging system. (Figure adapted.[1,**,††])

cinatti, OH). This valve allows intermittent gas flow into the driving circuit, of which the flow is regulated by an electronically timed solenoid valve. During inspection prior to disassembly of the ventilator, it initially functioned normally at an inspiratory:expiratory ratio of 1:2. After approximately 30–45 min of operation, inspiratory time was noted to increase while expiratory time decreased. This continued until the ventilator failed while in a constant-inspiratory phase mode. The Clippard control valve was found stuck in the open or inspiratory position, as illustrated in figure 3. Inspection of the control valve revealed that the stem of the valve was moving sluggishly or not at all; at same time, however, the solenoid valve was heard to be operating at the correct time intervals. The control valve malfunction permitted a constant flow of driving gas to the bellows chamber during both the inspiratory and expiratory phases of the breathing cycle. The presence of driving gas prevented the opening of the ventilator relief valve, and consequently, prevented expansion of the bellows. When the ventilator relief valve is occluded‡‡ and the pop-off valve closed, excess volume from the breathing circuit system cannot be vented.[1] Additionally, all incoming fresh gas remains in the breathing circuit, resulting in excessively high airway pressure.[1] In our case, incoming gas flow consisted of continuous fresh gas flow (2.0 l/min) and oxygen flush flow (50 l/min) during attempts to expand the bellows with the oxygen. The acute increase in airway pressure can be attributed to this high fresh gas flow. Since this sequence of events occurred within a time interval shorter than that specified for low tidal volume alarm activation (15 s), the alarm remained silent.

The control valve was removed from the machine and examined. The O-ring, which forms a seal around valve stem, was found to be cracked in three locations extending half-way through the ring from the inner to the outer diameter. It is speculated that these cracks allowed for distortion of the O-ring during operation, and that this distortion impeded progress of the valve stem.

The North American Drager anesthesia machine is equipped to warn of excessive airway pressure through continuous pressure and high pressure alarms. Neither

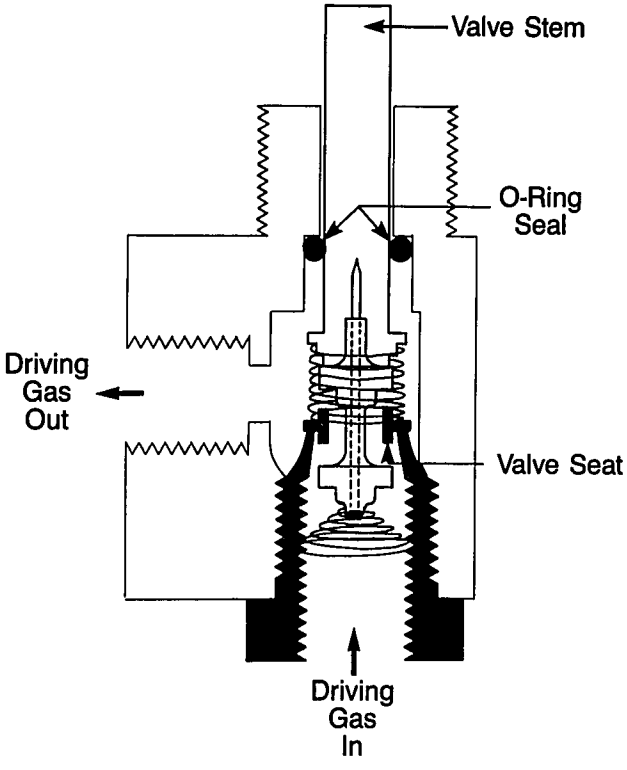

FIG. 3. Clippard control valve. A cross-section view is shown of the control valve in the open position. Note that an O-ring seal surrounds the valve stem. The valve closes normally due to recoil of the spring and to the presence of driving gas in the lower portion of the control valve assembly.

‡‡ This normally occurs during inspiration only; due to control valve malfunction in our case, however, occlusion of the ventilator relief valve occurred throughout the respiratory cycle.

alarm was activated during our case. However, both alarms were found to be operating within their specified tolerances. The high-pressure alarm did not activate, since mechanical ventilation was discontinued when PIP reached 55 cmH_2O, which is below the high-pressure alarm threshold (60 cmH_2O). The continuous pressure alarm is activated only if airway pressure does not drop below 20 cmH_2O during a 10-s period. In our case, ventilator malfunction was detected prior to the total failure of the control valve. It is likely that during mechanical ventilation airway pressure fell below 20 cmH_2O at some point during the expiratory cycle.

Diagnosis of this type of ventilator malfunction may have been expedited by the use of a breathing circuit pressure monitor with a real-time pressure waveform display (standard equipment for the North American Drager Narkomed 2B and Narkomed 3). A change in the waveform would be considerably easier to recognize than would a simple increase in PIP on the airway pressure gauge. The anesthesia machine we used (Narkomed 2A) was not equipped with the real-time breathing pressure waveform monitor.

The malfunction of the control valve, as presented in our case, resulted in a changed ratio of inspiration to expiration. Capnography would have detected this intraoperative problem. When the bellows cease to make respiratory excursions, the capnograph normally sounds a "no breath detected" warning; in our case, however, this alarm was silenced, and the anesthesiologist, rather than observing the capnography screen, was preoccupied with attempts to discover the cause for ventilator failure.

New devices have been developed to reduce the likelihood of barotrauma with ventilator malfunction. One such device, available from North American Drager, is referred to as a pressure limit controller, and can be retrofitted on the bellows assembly of the Narkomed 2A (fat bellows only), the Narkomed 2B, and the Narkomed 3.§§ The pressure-limit controller has a variable pressure-relief valve, which can be adjusted from approximately 10–100 cmH_2O. When the pressure inside the breathing circuit exceeds the pressure threshold selected by the operator, the driving gas is vented to the atmosphere. In this way, dangerously high breathing pressure may be prevented.

§§ North American Drager and Andrews JJ, personal communications.

In addition to the malfunction described in our case, other mechanisms may prevent the ventilator bellows from expanding with a consequent dangerous rise in PIP and barotrauma. These include mechanical obstruction of the driving gas exhaust system,[10] failure of the ventilator gas evacuation outlet valve,[9] misplacement of unidirectional valves,[8,11] and closure of the ventilator port.[7] Theoretically, bellows malfunction due to solenoid valve failure (if stuck in the open position) could present in a manner similar to our case, except that solenoid valve failure could be diagnosed by the absence of the audible click of a working-valve.

Our case illustrates that despite regular preventive maintenance of anesthesia equipment, as well inspection and testing of equipment prior to each anesthetic, some potential causes of malfunction can go undetected. In this case, a defective O-ring caused failure of the ventilator control valve. Although there currently is no recommended procedure for replacing these O-rings, our case suggests that control valves should be checked at regular intervals for proper function and possible replacement of O-rings.

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