Potential Rebreathing After Continuous Positive Airway Pressure Failure During Sleep*

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Background: Continuous positive airway pressure (CPAP) is widely applied as a home treatment during sleep. Conventional CPAP devices are based on a blower to generate nasal pressure and to maintain air washout from the circuit. Because common CPAP systems do not incorporate alarms, failure in the device or in the electric supply could result in rebreathing.

Aim: To assess the potential rebreathing to which a patient could be subjected after CPAP failure.

Methods: Four conventional CPAP devices, PV100 (Breas Medica; Molnlycke, Sweden), CP90 (Taema; Antony, France), and SoloPlus and BiPAP (Respironics, Murrysville, PA), and three common exhalation ports (Whisper Swivel [Respironics], Plateau [Respironics], and 4-mm orifice) were tested in a bench study. Rebreathing after failure was assessed by measuring the resistance of the exhalation port (REP) and the resistance of the tubing plus CPAP device (RTUB), and by measuring O₂ and CO₂ concentrations in the nasal mask in a subject breathing through a CPAP system.

Results: REP was much higher (approximately 30 cm H₂O × s/L) than RTUB (approximately 1 cm H₂O × s/L). Most (approximately 90%) of the breathing tidal volume would flow from/to the tubing plus CPAP device, which represents a dead space (> 0.5 L) similar to the patient’s tidal volume. After CPAP failure, end-tidal O₂ in the mask changed from 16.8 to 9.2% and end-tidal CO₂ in the mask changed from 4.2 to 6.2%. By contrast, O₂ and CO₂ did not change when a nonrebreathing valve was placed in the mask.

Conclusions: Common CPAP systems run a risk of inducing rebreathing in case of failure. This risk could be easily avoided by including a passive valve in the apparatus.

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Key words: device failure; health technology; home ventilation; nasal continuous positive airway pressure; patient safety; sleep apnea

Abbreviations: CPAP = continuous positive airway pressure; REP = flow-dependent resistance of the exhalation port; RLOAD = load to breathe imposed by the CPAP system; RTUB = flow-dependent resistance of the tubing plus continuous airway pressure device; SAHS = sleep apnea/hypopnea syndrome

The sleep apnea/hypopnea syndrome (SAHS) is a highly prevalent dysfunction¹ that is commonly treated by means of long-term application of nasal continuous positive airway pressure (CPAP).² In

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sequently, any failure in the domestic electric supply or in the CPAP device that results in blower arrest would prevent air renewal and, hence, induce rebreathing.

In contrast to life-support ventilators, common home CPAP systems used for patients with SAHS do not incorporate any alarm or nonrebreathing valve in case of power loss or device failure. These uncommon, but in no way impossible, events are issues that are frequently raised by patients and constitute a source of anxiety that could decrease the patient’s reliance on the treatment. CPAP failure at home affects the safety of this health-care equipment. Indeed, after CPAP failure, the sleeping patient would be subjected to rebreathing unless he or she spontaneously breathes through the mouth or wakes up and removes the mask. The aim of this study was to assess potential rebreathing after a CPAP failure in conventional systems used in home treatment for patients with SAHS.

**Materials and Methods**

Four conventional CPAP devices with their tubing were studied: (1) PV100; Breas Medica; Molndyke, Sweden; (2) CP90; Taema; Antony, France; and (3) SoloPlus and (4) BiPAP; Respirronics; Murrysville, PA. We also studied three common exhalation ports: a Whisper Swivel (Respirronics), a Plateau (Respirronics) exhalation port, and an orifice-type port (4-mm diameter) in the nasal mask. To assess the amount of rebreathing resulting from a failure of the CPAP device, the flow-dependent resistance of the exhalation port ($R_{ep}$) and the flow-dependent resistance of the tubing plus CPAP device ($R_{tub}$) were independently measured.

$R_{ep}$ and $R_{tub}$ were measured with the CPAP device switched off. As described in Figure 1, the nasal mask of the CPAP system was tightly connected to a servocontrolled pump (PWG; MH Custom Design & Mfg; Midvale, UT) by means of a special plastic piece that ensured no air leak. The pump was driven by a computer and simulated the breathing flow generated by a patient. Flow was recorded by a Fleisch-type pneumotachograph (Metabo; Epalinges, Switzerland) connected at the outlet of the flow generator and a differential pressure transducer ($\pm 2$ cm $H_2O$; MP-45; Validyne Engineering; Northridge, CA). Pressure at the nasal mask was directly recorded by a similar transducer ($\pm 50$ cm $H_2O$). To measure $R_{ep}$, the tubing connecting the CPAP device and the mask was occluded and a flow through the exhalation port was imposed by means of the generator (Fig 1). $R_{ep}$ was computed as the quotient between nasal pressure and flow through $R_{ep}$, which in this case coincided with the flow measured by the pneumotachograph. $R_{tub}$ was measured for flows ranging from 0 to 0.5 L/s. $R_{tub}$ was measured following the same procedure, but in this case the exhalation port was occluded and flow was imposed through the tubing plus CPAP device.

The load to breathe imposed by the CPAP system ($R_{load}$) was computed as the parallel combination of $R_{ep}$ and $R_{tub}$ at the different flow levels. The magnitude of rebreathing corresponding to a sinusoidal breathing flow ($\pm 0.5$ L/s, 15 breath/min) simulating spontaneous ventilation was assessed. Direct measurement of the fraction of breathing flow through each pathway ($R_{ep}$ and $R_{tub}$) would require the placement of a pneumotachograph in each pathway. Taking into account that the resistance of conventional pneumotachographs is similar to the $R_{tub}$ (Fig 2), inserting a pneumotachograph would completely modify the distribution of breathing flow between tubing and exhalation port. Accordingly, the distribution of the breathing flow between the two parallel pathways, which is a direct assessment of the rebreathing induced, was numerically computed from the pressure-flow relationship of each pathway (ie, flow-dependent values of $R_{ep}$ and $R_{tub}$; Fig 2) on the basis that these two effective resistances are connected in parallel.

To illustrate the effect of a failure in the CPAP system, we recorded nasal pressure, and the $O_2$ and $CO_2$ concentrations (CPX; Medical Graphics; St. Paul, MN) in the air of the mask before and after switching-off a conventional CPAP device (SoloPlus and Plateau valve) applied to an awake normal subject.

![Diagram of the experimental system](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21972/ on 06/26/2017)
breathing spontaneously. To assess whether including a protecting valve in the setting was useful to avoid rebreathing after CPAP failure, we placed a commercially available nonrebreathing valve (Spectrum; Respironics) in the mask during a switching-off trial in the same subject. This valve was connected at the nasal mask and allowed communication between the air inside the nasal mask and the atmosphere. When CPAP was applied (ie, nasal pressure was higher than atmospheric pressure) the valve was closed. In the absence of CPAP (ie, equal pressure inside the mask and the atmosphere), the valve was open and avoided rebreathing. Given its operation, this kind of valve did not modify the patient’s load to breathing nor altered the normal operation of the CPAP device.

**Results**

The tubing plus CPAP device exhibited an effective $R_{tub}$ that was much lower than the $REP$s in all the investigated systems (Fig 2). The difference between $RTUB$ and $REP$ increased with flow and was considerably high for typical values (0.5 L/s) of peak inspiratory and expiratory breathing flows ($RTUB$, approximately 1 cm H$_2$O × s/L vs $REP$, approximately 30 cm H$_2$O × s/L). Accordingly, in the case of CPAP failure, the $R_{load}$ imposed by the apparatus under failure was negligible: $R_{load} < 1$ cm H$_2$O × s/L in all the CPAP settings.

Figure 3 shows the distribution of tidal breathing flow between the exhalation port (Plateau) and the tubing plus CPAP device (SoloPlus). As $REP$ was much greater than $RTUB$, most (approximately 90%) of the patient’s breathing flow was derived through the dead space pathway and only a small fraction (approximately 10%) was fresh air through the exhalation port. These results were similar for all the CPAP systems investigated.

The O$_2$ and CO$_2$ concentrations in the mask before and after switching off a conventional CPAP system is shown in Figure 4, left. After switching off the system, the load to breathe imposed on the subject was even lower than under normal CPAP application, as evidenced by the decrease observed in the nasal pressure oscillations induced by breathing. The reduction in O$_2$ and the increase in CO$_2$ concentrations observed immediately after mimicking a CPAP failure illustrate the magnitude of the rebreathing induced. Figure 4 (right) illustrates the effectiveness of a nonrebreathing valve placed in the mask during a switching-off trial in the same subject as in Figure 4, left. As expected, and in contrast to that found with the conventional system, the concentrations of O$_2$ and CO$_2$ did not change after switching off the CPAP device.

**Discussion**

In the case of CPAP failure, we demonstrated that the exhalation port exhibits a much higher resistance than the tubing plus CPAP device and that, consequently, most of the tidal breathing flows through the latter pathway. As the volume of conventional tubing and CPAP device (≥ 0.5 L) is similar or even greater than normal tidal volume, breathing through a conventional CPAP system after a failure results in considerable rebreathing.

In the CPAP system routinely used to treat SAHS...
at home, the patient breathes through the tubing connecting the CPAP device to the nasal mask and the exhalation port (Fig 1). Under normal operating conditions, the blower inside the CPAP device imposes a flow of fresh air through the tubing and the exhalation port. The level of nasal CPAP applied corresponds to the pressure drop across the exhalation port and is regulated by modifying the flow generated by the blower. The use of an exhalation port with a high $R_{ep}$ reduces the flow (and hence the operating noise) required to generate common CPAP values. $R_{tub}$ is minimized to facilitate the patient’s breathing. Any failure causing blower arrest eliminates both CPAP and air washout. Consequently, rebreathing ensues as the patient breathes through a dead space pathway with low resistance (tubing plus CPAP device) and through a high resistance pathway to the atmosphere (exhalation port). The magnitude of the induced rebreathing (Fig 4) depends on the distribution of breathing flow between these two pathways (Fig 3), i.e., on the relative magnitudes of their resistances ($R_{tub}$ and $R_{ep}$). Accordingly, changes in $O_2$ and $CO_2$ concentrations similar to those shown in Figure 4 would be found from the other equipment studied (Fig 2).

The evaluation of the potential rebreathing after CPAP failure was carried out in a laboratory study. This procedure allowed us to systematically assess the mechanical characteristics of the setting under controlled conditions, and to quantify the rebreathing induced by a failure in the generation of CPAP. Given that most CPAP and bilevel pressure settings for home treatment are based on generating nasal pressure by means of the same principle (blower and exhalation port), the results found in the investigated CPAP systems should be representative.

Immediate awakening of the sleeping patient after CPAP failure is not ensured because the load to breathe would not be increased and the reduction in operating noise would be negligible for currently available CPAP devices. Consequently, a common CPAP device under failure would induce rebreathing and thereby promote progressive deterioration of blood gases in the sleeping patient. This situation could be reversed only after blood gas deterioration was enough to induce mouth breathing or patient awakening. There is no guarantee that a conventional CPAP system prevents the rebreathing it produces in the event of failure during patient’s sleep at home. This constitutes a key issue concerning the safety of

![Figure 4](image-url)  
**Figure 4.** Nasal CPAP and $O_2$ and $CO_2$ concentrations in the nasal mask before and after switching off a conventional CPAP system (left) and the same conventional system including a nonrebreathing valve (right).
this equipment. Indeed, given the absence of an alarm system, the responsibility for avoiding rebreathing is actually transferred from the equipment under failure to the physiologic control mechanisms of the patient. We wonder whether a patient using a conventional CPAP device should be explicitly and formally informed about this potential risk. This study also highlights the need for clearly informing the patient about the different practical issues of CPAP therapy. In particular, the patient should be instructed to verify the performance of CPAP each time he/she awakes during the night and to immediately take the mask off in the case of a failure in the device.

In this laboratory study, we carried out an analysis of the potential consequences of a sudden arrest of the CPAP apparatus during sleep. To this end, we carefully characterized the different elements of a conventional CPAP system and illustrated the potential rebreathing actually induced in an awake healthy subject. Accordingly, we demonstrated that there is a potential risk of rebreathing. In this bench study, we did not investigate the frequency of occurrence or the clinical implications of CPAP failure at home. The probability of a serious accident after CPAP failure and its clinical relevance is probably low from a statistical viewpoint, since in most cases the patient would be able to spontaneously respond to the challenge. Nevertheless, patient response during sleep is not ensured in all the individual cases, particularly taking into account that CPAP is being prescribed for an increasing number of patients and different pathologies that even may coincide in a same patient (eg, sleep apnea, Cheyne-Stokes, obesity, nocturnal hypoventilation). Indeed, patients with SAHS who also have COPD, respiratory muscle weakness, low CO2 response, or who are receiving depressant drugs may present reduced response to a rise in CO2 concentration and decreased arousability and, therefore, they could be more exposed to the risk of rebreathing associated to CPAP failure. Taking into account that home CPAP is applied to an increasing number of patients during the whole night for a very long period, we wonder whether the inclusion of alarms or safety valves (Fig 4) to avoid this risk is advisable.

In conclusion, common home CPAP systems used in patients with SAHS run a risk of inducing rebreathing in the event of electrical or equipment failure. The inclusion of a passive valve, which could be inexpensive and easy to attach to any CPAP device already in use, is a possible procedure to improve both the safety of the equipment and the reliance of the patient.

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