Exhaled gases and the potential for cross-infection via noninvasive ventilation machines

To the Editor:

Use of long-term ventilation (LTV) benefits patients with a diverse range of conditions, including Duchenne muscular dystrophy, motor neurone disease and scoliosis [1]. Patients with pulmonary disease as well as neuromuscular disease can benefit from LTV. COPD patients treated with LTV experience a reduction in hospital admissions and the use of LTV in cystic fibrosis (CF) patients is increasing [2, 3].

Despite improvements in outcomes, patients receiving LTV are at risk of pneumonia, with *Pseudomonas aeruginosa* commonly isolated [4]. Infections with *P. aeruginosa*, *Burkholderia cepacia* complex and *Mycobacterium abscessus* complex in CF are associated with worse clinical outcomes [5, 6]. Furthermore, oral options for treatment of *P. aeruginosa* infections are limited, increasing the likelihood of hospital admissions. These organisms are considered transmissible between patients and can survive for extended periods within the environment [6]. In the context of the coronavirus disease 2019 (COVID-19) pandemic, the requirement for respiratory support in an unprecedented number of patients resulted in intense resource pressures, including the re-distribution of LTV-allocated ventilators to the acute wards for treatment. Throughout the COVID-19 pandemic, all forms of assisted ventilation have been considered aerosol generating, with an associated high risk of transmission of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus through the use of noninvasive ventilation (NIV) [7]. However, emerging evidence challenges this, suggesting that assisted ventilation is not associated with increased aerosol production, and may even reduce aerosol [8].

It is recognised that NIV equipment can become infected with potentially pathogenic organisms [9]. However, the only study to investigate the contamination of NIV machines investigated seven machines from CF patients using bacterial filters and did not find evidence of contamination with potentially pathogenic organisms [10].

NIV machines are reused between patients and cannot be sterilised. In the UK, there is a national recommendation that bacterial filters be used between the ventilator outlet and the NIV circuit tubing, for all patients receiving acute NIV [11]. There is no such guidance in LTV. Survey data from the Specialists in Long-term Ventilation at Home (SiLVaH) network suggest that 50% of LTV centres do not use bacterial/viral filters [12].

Most ventilator circuits used for LTV are single-limb circuits. Such circuits require an intentional leak in the circuit through which exhaled gas can escape to allow exhalation and avoid re-breathing carbon dioxide (CO2). This can be achieved in one of three ways: 1) the use of a vented mask, 2) the use of an exhalation port, and 3) the use of an active exhalation valve circuit. It is assumed that the leak/exhalation device prevents exhaled gas reaching the ventilator outlet and therefore there is no risk of contaminating the ventilator and transmitting bacteria or viruses between patients. British Thoracic Society guidance for acute NIV supports this assumption, stating “In most ventilators used for NIV there is no airflow from the patient back into the ventilator” [11].

Irrespective of their underlying disease pathology, patients developing respiratory failure requiring LTV are living with severe illness, often with multimorbidity and frailty. Given the increasing prevalence of LTV
and the potential implications of bacterial or viral lower respiratory tract infection or pneumonia in this group of patients, it is critically important that the risks of contamination of NIV machines and the potential transmission of organisms between patients are investigated. The first step in the understanding of this is investigation of gas flow within the NIV circuit. We aimed to assess whether the belief that exhaled gas does not reach the ventilator during treatment with NIV is valid.

We investigated whether exhaled gas reached the ventilator outlet using three noninvasive ventilators and a continuous positive airway pressure (CPAP) machine. The machines used were: Vivo 50 (Breas, Gothenburg, Sweden), NIPPY 3+ (Breas, Stratford-upon-Avon, UK), A40 (Philips Respironics, Murrysville, PA, USA) and AirSense CPAP (ResMed Ltd, Bella Vista, Australia).

To establish whether exhaled gas reached the ventilator outlet, we used a side-stream CO₂ analyser (Intersurgical, Wokingham, UK) attached to a 23G needle placed into the ventilator tubing (Armstrong Medical, Coleraine, UK) immediately adjacent to the ventilator outlet (figure 1a). The deadspace of the tubing was 500 mL.

![FIGURE 1](https://doi.org/10.1183/23120541.00109-2022)  
**FIGURE 1 a)** Positioning of the side-stream carbon dioxide analyser adjacent to the ventilator outlet and **b)** typical carbon dioxide trace recorded during analysis.
One member of the team (B. Messer) underwent NIV/CPAP using three different circuit configurations (except where the device functionality did not allow): 1) vented mask (AirFit F10, ResMed), 2) non-vented mask with an exhalation port (Intersurgical), and 3) active exhalation valve (Breas).

The settings used, which would reflect standard clinical practice were as follows. Vented mask configuration settings: mode pressure control (PC), inspiratory positive airway pressure (IPAP) 20 cmH₂O, expiratory positive airway pressure (EPAP) 4 cmH₂O, 12 breaths·min⁻¹. Exhalation port configuration settings: mode PC, IPAP 20 cmH₂O, EPAP 4 cmH₂O, 12 breaths·min⁻¹. Active exhalation valve configuration settings: mode PC, IPAP 20 cmH₂O, EPAP 0 cmH₂O, 12 breaths·min⁻¹. CPAP settings: 8 cmH₂O.

In order to establish whether the exhaled CO₂ was related to tidal volume, we used a Wright’s respirometer in the circuit with the exhalation port configuration, to adjust the NIV pressures to give a measured exhaled tidal volume between 2000 mL and 500 mL. We voluntarily controlled the tidal volume in the CPAP circuit.

In each circuit configuration using each ventilator and CPAP machine, CO₂ was detected at the ventilator outlet, demonstrating that exhaled gas can reach the ventilator. Figure 1b shows a typical exhaled CO₂ trace. In our experiments, the exhaled CO₂ varied between 1.5 kPa and 3.7 kPa depending upon the circuit and machine used (table 1). With tidal volumes >800 mL, CO₂ was detected at the ventilator outlet. At <800 mL, no CO₂ was detected.

We have shown that, contrary to published guidelines, exhaled gas from a patient reaches the ventilator outlet in all trialled circuit configurations during NIV/CPAP treatment, with standard clinical settings, when tidal volumes were >800 mL. Although 800 mL represents a relatively high tidal volume, it is well within the clinical range for CPAP and NIV users, especially those with recruitment breaths, a recruitment profile or during coughing.

NIV and CPAP machines are not considered single-patient-use devices. These findings therefore demonstrate the potential for contamination of such devices through exhaled air. This has profound implications for the LTV and sleep medicine community, where the use of bacterial and viral filters is not universal, not least in the context of current risks of COVID-19 infection, to which LTV patients are particularly vulnerable [7].

If the theoretical risks of contamination are shown to result in machine colonisation and transmission of bacteria and viruses between patients, the implications for LTV patients are substantial. There are potential adverse outcomes from transmission of pathogenic bacteria in patients using LTV, particularly those with chronic lung diseases such as CF, non-CF bronchiectasis and COPD. There is therefore an urgent need within the infection control and LTV community to investigate whether ventilators are contaminated, including discussion about practices relating to reuse of ventilators, decontamination of ventilators and routine use of bacterial filters during treatment with LTV.

This experiment does not prove contamination of the machines from users, nor does it demonstrate cross-infection. However, it disproves the assumption that exhaled (thus potentially infected) gases cannot travel down the ventilator tubing and back to the device outlet. We believe this understanding of the function and workings of the breathing circuit is the first step in establishing the risk(s) of device contamination. Further studies investigating bacterial growth within ventilator devices and circuitry should be undertaken to help assess the risks of contamination.

| Circuit          | Breas Vivo 50 | Breas NIPPY 3+ | Philips Respironics A40 | ResMed AirSense CPAP |
|------------------|---------------|----------------|-------------------------|----------------------|
| Exhalation port  | 2.9           | 3.1            | 3.2                     | 3.7                  |
| Active valve     |               |                |                         |                      |
| 0 cmH₂O PEEP     | 2.1           | 1.6            |                         |                      |
| 4 cmH₂O PEEP     | 1.5           |                |                         |                      |
| Vented mask      | 2.7           | 2.8            | 2.7                     | 3.6                  |

Data are given in kPa. CPAP: continuous positive airway pressure; PEEP: positive end-expiratory pressure.
Benjamin Messer¹, Alison Dawn Armstrong¹, Nicholas David Lane¹,², Ali Robb³ and Robert Edward Bullock¹

¹The North East Assisted Ventilation Service, The Newcastle upon Tyne Hospitals NHS Foundation Trust, Royal Victoria Infirmary, Newcastle upon Tyne, UK. ²Translational and Clinical Research Institute, Faculty of Medical Sciences, Newcastle University, Newcastle upon Tyne, UK. ³Newcastle Microbiology and Virology Services, The Newcastle upon Tyne Hospitals NHS Foundation Trust, Royal Victoria Infirmary, Newcastle upon Tyne, UK.

Corresponding author: Benjamin Messer (ben.messer@nhs.net)

Provenance: Submitted article, peer reviewed.

Data availability: Data sharing requests will be considered immediately following publication. Requests should be made via the corresponding author.

Author contributions: B. Messer undertook the literature search, designed the study, collected and analysed the data, and prepared the manuscript. A.D. Armstrong assisted in the study design, collected and analysed the data, and reviewed the manuscript. N.D. Lane reviewed the manuscript. A. Robb undertook the literature search and reviewed the manuscript. R.E. Bullock assisted in the study design and reviewed the manuscript.

Conflict of interest: B. Messer reports personal fees from Fisher & Paykel outside the submitted work. A.D. Armstrong reports personal fees from Fisher & Paykel and grants from Breas, outside the submitted work. N.D. Lane reports grants from the ResMed Foundation and Bright Northumbria, and nonfinancial support from Chiesi, outside the submitted work. A. Robb has nothing to disclose. R.E. Bullock has nothing to disclose.

Support statement: The authors thank the Newcastle Hospitals charity for support with open access fees.

References

1. Messer B, Clark A. Chapter 11 – Neuromuscular disease. In: Bourke SJ, Peel T, eds. Integrated Palliative Care of Respiratory Disease. 2nd Edn. Cham, Springer, 2019; pp. 173–197.

2. Murphy PB, Rehal S, Arbane G, et al. Effect of home noninvasive ventilation with oxygen therapy vs oxygen therapy alone on hospital readmission or death after an acute COPD exacerbation: a randomized clinical trial. JAMA 2017; 317: 2177–2186.

3. Archangelidi O, Carr SB, Simmonds NJ, et al. Non-invasive ventilation and clinical outcomes in cystic fibrosis: findings from the UK CF registry. J Cyst Fibros 2019; 18: 665–670.

4. Chenoweth CE, Washer LL, Obeyesekera K, et al. Ventilator-associated pneumonia in the home care setting. Infect Control Hosp Epidemiol 2007; 28: 910–915.

5. Araújo D, Shteinberg M, Aliberti S, et al. The independent contribution of Pseudomonas aeruginosa infection to long-term clinical outcomes in bronchiectasis. Eur Respir J 2018; 51: 1701953.

6. Saiman L, Siegel JD, LiPuma JJ, et al. Infection prevention and control guideline for cystic fibrosis: 2013 update. Infect Control Hosp Epidemiol 2014; 35: Suppl. 1, S1–S67.

7. UK Health Security Agency. Infection prevention and control for seasonal respiratory infections in health and care settings (including SARS-CoV-2) for winter 2021 to 2022. Date last accessed: 22 February 2022. www.gov.uk/government/publications/wuhan-novel-coronavirus-infection-prevention-and-control/covid-19-guidance-for-maintaining-services-within-health-and-care-settings-infection-prevention-and-control-recommendations

8. Wilson NM, Marks GB, Eckhardt A, et al. The effect of respiratory activity, non-invasive respiratory support and facemasks on aerosol generation and its relevance to COVID-19. Anaesthesia 2021; 76: 1465–1474.

9. Rodríguez González-Moro JM, Andrade Vivero G, de Miguel Díez J, et al. Colonización bacteriana y ventilación mecánica domiciliaria. Prevalencia y factores de riesgo [Bacterial colonization and home mechanical ventilation: prevalence and risk factors]. Arch Bronconeumol 2004; 40: 392–396.

10. Mutagi A, Nash EF, Cameron S, et al. Microbial contamination of non-invasive ventilation devices used by adults with cystic fibrosis. J Hosp Infect 2012; 81: 104–108.

11. British Thoracic Society Standards of Care Committee. Non-invasive ventilation in acute respiratory failure. Thorax 2002; 57: 192–211.

12. Armstrong A, Messer B. Is there a risk to home mechanical ventilation patients who are issued with previously used room air ventilators? Poster 37. ERS Respiratory Failure and Mechanical Ventilation Conference, Berlin, 2020.