about oxygen use during the COVID-19 pandemic sadly remains topical, as the disease continues to overwhelm critical care capacity in many hospitals.

Hall and Chakladar[1] suggest that higher FGF rates during the provision of non-inhalation general anaesthesia should be avoided until after the pandemic has abated, citing reports of hospitals that have experienced oxygen shortages.[1] In the UK, which continues to be severely affected by COVID-19, there have been several cases of oxygen supply issues during surges of hospital admissions. However, this appears not to be as a result of a lack of oxygen *per se*, but the per minute oxygen demand exceeding the flow capacity of supply systems.[3] Internationally, absolute shortages of oxygen have been experienced, for example in sub-Saharan Africa.[4] This appears to be largely as a result of economic and infrastructure factors, but is not representative of a global oxygen shortage.

Whilst low-flow anaesthesia should form part of a strategy to cope with exceptional surges in oxygen demand, its potential contribution to this endeavour is unfortunately minimal. The total consumption of oxygen per patient using higher FGFs via a circle system is not as great as it may at first appear when considered in context. Using Hall and Chakladar's[1] example, a 6-h case using FGF of 6 L min−1 and a fraction of inspired oxygen (FiO2) of 0.3 requires 252 L of oxygen. Whilst this may appear to be a large volume, it represents only 0.7 L min−1 of oxygen gas usage; lower than the minimal requirements of even low-flow nasal cannulae.

Furthermore, it has been shown that FiO2 may be significantly lower than the fraction of oxygen *delivered* at low FGFs because of both oxygen consumption and increased rebreathing of exhaled gases by the patient.[5] While Hall and Chakladar[1] suggest that providing an FiO2 of 0.3 at 1 L min−1 requires an oxygen flow of 0.1 L min−1, Hendrickx and colleagues[6] found in their *in vivo* study that oxygen flows of 0.2 L min−1 were required to maintain a steady-state FiO2 of 0.31 within the circle system at a total FGF of 1 L min−1. It is likely that even higher oxygen flow rates would be required for patients with increased oxygen consumption at low FGFs. Consequently, oxygen savings are not proportional to the reduction in FGF for a given FiO2.

During times of extraordinary oxygen demand we agree that clinicians may be required to take all available steps to safely minimise consumption. But even during the COVID-19 pandemic, increasing the FGF from 1 to 6 L min−1 during non-inhalation anaesthesia remains a reasonable option at most times, representing only a 0.5 L min−1 increase in oxygen gas usage when delivering an FiO2 of 0.3.

In addition to its tragic impacts on the health and wellbeing of millions of people, the COVID-19 pandemic is responsible for profound adverse economic and environmental effects.[7,8] Measures that clinicians can take to mitigate these problems, including optimising FGF rates, are perhaps now more relevant than ever.[9]

Declarations of interest

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Protection of healthcare workers during aerosol-generating procedures with local exhaust ventilation

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Editor—During the Severe Acute Respiratory Syndrome Coronavirus-1 (SARS-CoV-1) epidemic, performing tracheal intubation and noninvasive ventilation is associated with viral transmission to healthcare workers.[1] Medical interventions, in particular related to airway management, are classified as aerosol-generating procedures.[2] To protect healthcare workers during aerosol-generating procedures, the emphasis has been on wearing personal protective equipment.[3] Although this is an important measure, eliminating pathogens from room air is a superior intervention according to the US Centers for Disease Control and Prevention.[4] Air exchange rate is important to decrease aerosol concentrations, but adequate room ventilation is absent in many hospital environments.[5]

We explored local exhaust ventilation (LEV), an engineering technique used to reduce airborne particle concentration by capturing contaminants or fumes directly at their source.[6] Because positioning of the extraction hood is an operator-dependent process, we included different hood positions to determine if this influences aerosol extraction. We hypothesised that LEV reduces aerosol concentration and distribution during a simulated aerosol-generating procedure. The experiments were performed in a resuscitation room in the emergency department of our academic teaching hospital (Supplementary Appendix 1). Room ventilation consisted of a neutral pressure hierarchy towards the hallway and an air exchange rate of 24 changes h^{-1}.

We simulated a tracheal intubation setting by positioning a manikin (ALS Simulator, Laerdal, Stavanger, Norway) supine on the patient bed. Two particle counters (model 2016; Lighthouse, Boven-Leeuwen, the Netherlands) were positioned 35 cm above the face: one at the head end of the bed to simulate an intubator and one at 1.5 m away to simulate an assistant. Both counters sampled air at 2.8 L min^{-1} (0.1 ft^3 min^{-1}) and counted particles sized 0.2–10.0 μm, storing data every minute. The LEV unit (UT200.2; ULT AG Umwelt-Lufttechnik, Löbau, Germany) was placed on the left side of the bed and generated a 635 m^3 h^{-1} airflow with a 3200 Pa subatmospheric pressure at the tip of an extraction arm (Alsident 75–6555; Hammel, Denmark). The capturing hood (Square hood 1–754232; Alsident) was positioned near the face of the manikin (Fig. 1 online video). Air drawn in from the manikin facial area passed through a high-efficiency particulate air (HEPA-13) filter, certified to remove >99.95% of airborne particles, and was recirculated into the room. After recording baseline aerosol concentration, we nebulised normal saline into the manikin trachea for 10 min using a jet nebulizer (MaxiNeb® Duo; Medisize-Flexicare, Hoofddorp, the Netherlands). This was repeated three times for each of the following capturing hood positions: (i) vertically on the chest, (ii) at a 45° angle above the chest, and (iii) horizontally above the face (Supplementary Appendix 1). A sequence of three measurements was obtained during aerosol dispersal with the local exhaust ventilator switched off. Aerosol concentration was allowed to return to baseline after each measurement. All equipment was remotely operated and data were stored digitally for offline analysis. Room doors remained closed and no people were present in the room during the experiments.

MATLAB (R2018b; MathWorks, Inc., Natick, MA, USA) and SigmaPlot (version 12.5; Systat Software, Inc., San Jose, CA, USA) were used for data analysis. Data for particles ≥0.2 μm were used because SARS-CoV-2 aerosols are of similar aerodynamic diameter (0.25–0.5 and >2.5 μm).[7] Data from three

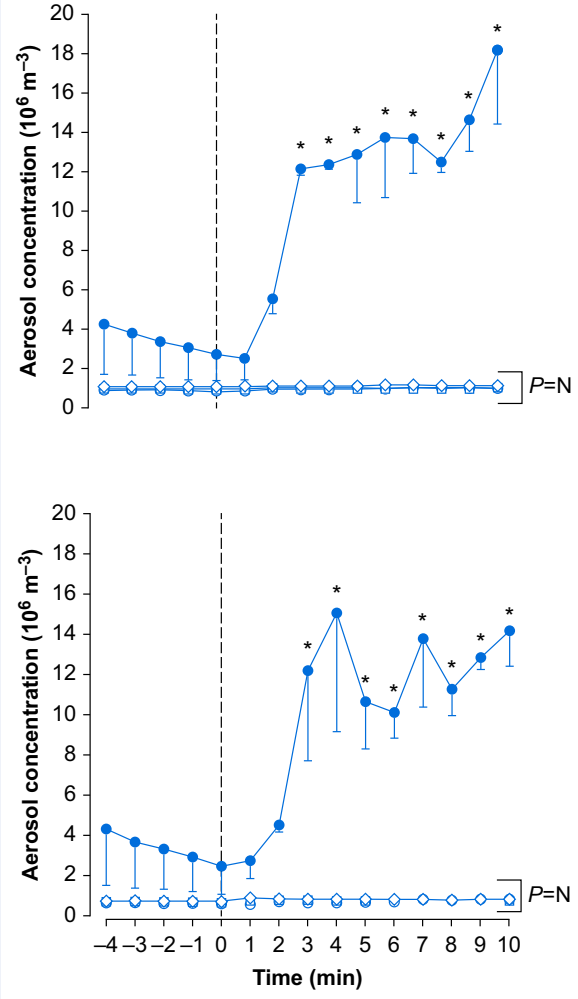

Fig 1. Mean (standard deviation) particle counts per minute before and during aerosol emission (t=0; dashed line) at the head end of the bed (upper panel) and at 1.5 m distance (lower panel). Exhaust ventilation was switched off (opaque circles) and switched on with hood position vertically on the chest (open circles), 45° above the chest (open squares), and horizontally above the face (open diamonds). *P<0.001 during aerosol emission compared with baseline. NS, non-significant.

measurements were synchronised and averaged per minute. Descriptive statistics were mean (standard deviation [SD]). To detect a change in aerosol concentration during dispersal, we performed analysis of variance for repeated measurements with *post hoc* Tukey testing, if applicable. *P*-values <0.05 indicated statistical significance. If an increase in aerosol concentrations was observed, we rejected the hypothesis that LEV can effectively eliminate aerosols from the room with the hood in that position. Based on data from an earlier project,[5] assuming a baseline aerosol concentration of 0.5 (SD 0.2)×10^6 m^{-3} and accepting a power of 90%, a sample size of three measurements was sufficient to detect a two-fold increase in aerosol concentration with a significance level of 0.05 (Z-test).

Aerosol concentration with the LEV enabled ranged between 0.6 (0.1) and 1.1 (0.1)×10^6 m^{-3} before and during aerosol emission, independent of various hood positions (Fig. 1). When the LEV was switched off, baseline aerosol concentrations were 3.3 (1.9) at bedside *vs* 3.4 (1.7)×10^6 m^{-3} at 1.5 m away. These increased to 11 (4.6) *vs* 12 (4.6)×10^6 m^{-3} during aerosol emission, respectively (*P*<0.001; Fig. 1). The results indicate that LEV effectively reduced exposure during a simulated aerosol-generating procedure independent of the position of the extraction hood. We conclude that LEV is a useful tool to protect healthcare workers from airborne pathogens.

Other modalities to prevent aerosol dispersion during aerosol-generating procedures have been reported, in particular the 'aerosol box', a barrier that can be placed over the head of the patient during aerosol-generating procedure. However, contrary to what the name might suggest, this does not protect healthcare workers against infectious aerosols.[8] Furthermore, adding an extra layer of complexity during aerosol-generating procedures might be time consuming and induce hypoxaemia in patients with COVID-19 and respiratory distress.[9,10] Upgrading room ventilation to decrease the removal time of airborne pathogens[5] is probably more effective, but may require extensive engineering measures. Local exhaust ventilation offers several advantages. Firstly, it captures airborne particles directly at the source, and therefore reduces aerosol removal time in the room to zero. Secondly, because capturing hood positioning is not critical, it does not add much complexity to a potentially stressful procedure. Thirdly, a wide array of quality-controlled LEV equipment is already available, which limits the risk of designing a custom-made solution. Lastly, a mobile LEV device gives the opportunity to perform aerosol-generating procedures in rooms with low air exchange rates. This could prevent transporting patients to suitable environments, and hence delay of acutely necessary care.

Two limitations need to be addressed. Firstly, people were absent in the room during the experiments, and their movements could create turbulence and thereby influence aerosol behaviour. This was necessary because people shed aerosols and particle counters are unable to distinguish these from nebulised saline. To approximate a real-world situation with turbulent airflow, we performed the experiment in a room equipped with a mixing-type ventilation system. Secondly, aerosols from normal saline were used, not from SARS-CoV-2. We do not expect that this influenced the results, because the aerodynamic diameter of SARS-CoV-2 aerosols is similar to the aerosols we used.[7]

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Declarations of interest

The authors declare that they have no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bja.2021.02.032.

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