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Review Article

Airborne transmission of severe acute respiratory syndrome coronavirus-2 to healthcare workers: a narrative review

N. M. Wilson,[1] **A. Norton,**[2] **F. P. Young**[3] and **D. W. Collins**[4]

1 Fellow, 3 Resident, 4 Clinical Director, Department of Intensive Care Medicine, Prince of Wales Hospital, Sydney, NSW, Australia
2 Registrar, Emergency Department, Oamaru Hospital, New Zealand

Summary

Healthcare workers are at risk of infection during the severe acute respiratory syndrome coronavirus-2 pandemic. International guidance suggests direct droplet transmission is likely and airborne transmission occurs only with aerosol-generating procedures. Recommendations determining infection control measures to ensure healthcare worker safety follow these presumptions. Three mechanisms have been described for the production of smaller sized respiratory particles ('aerosols') that, if inhaled, can deposit in the distal airways. These include: laryngeal activity such as talking and coughing; high velocity gas flow; and cyclical opening and closure of terminal airways. Sneezing and coughing are effective aerosol generators, but all forms of expiration produce particles across a range of sizes. The 5-μm diameter threshold used to differentiate droplet from airborne is an over-simplification of multiple complex, poorly understood biological and physical variables. The evidence defining aerosol-generating procedures comes largely from low-quality case and cohort studies where the exact mode of transmission is unknown as aerosol production was never quantified. We propose that transmission is associated with time in proximity to severe acute respiratory syndrome coronavirus-1 patients with respiratory symptoms, rather than the procedures per se. There is no proven relation between any aerosol-generating procedure with airborne viral content with the exception of bronchoscopy and suctioning. The mechanism for severe acute respiratory syndrome coronavirus-2 transmission is unknown but the evidence suggestive of airborne spread is growing. We speculate that infected patients who cough, have high work of breathing, increased closing capacity and altered respiratory tract lining fluid will be significant producers of pathogenic aerosols. We suggest several aerosol-generating procedures may in fact result in less pathogen aerosolisation than a dyspnoeic and coughing patient. Healthcare workers should appraise the current evidence regarding transmission and apply this to the local infection prevalence. Measures to mitigate airborne transmission should be employed at times of risk. However, the mechanisms and risk factors for transmission are largely unconfirmed. Whilst awaiting robust evidence, a precautionary approach should be considered to assure healthcare worker safety.

Correspondence to: N. M. Wilson
Email: nickwilson247@gmail.com
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Twitter: @CoVcast

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Introduction

Severe acute respiratory syndrome (SARS) coronavirus-2 (SARS-CoV-2) continues to cause an international health crisis through coronavirus disease 2019 (COVID-19). The safety of healthcare workers is a global priority to prevent collapse of healthcare systems and transmission from hospital to the community. Due to frequent close contact with infected patients, healthcare workers are at high risk. Healthcare workers made up over 20% of all cases during the previous SARS-CoV-1 epidemic [1–6]. At the start of April 2020, over one million people had been confirmed infected with SARS-CoV-2. A healthcare worker infection rate as high as in the SARS-CoV-1 epidemic would involve enormous numbers of healthcare workers.

Current personal protective equipment (PPE) and infection control guidelines from the World Health Organization (WHO) are based on the assumption that the primary mechanism of transmission is direct and indirect droplet spread [7]. Direct droplet spread is said to occur when respiratory particles greater than 5 μm in diameter make contact with the mucosal surface of a recipient. Indirect spread occurs when a fomite or an intermediate surface is touched, usually by a hand, which then contacts mucosal surfaces. The faeco-oral route is also possible, with viral content noted in stools [8].

Airborne spread is thought to occur when respiratory particles less than 5 μm in diameter are inhaled and deposited in the lungs. These particles have been described interchangeably as aerosols, droplet nuclei, airborne and small particles. The WHO advises that airborne transmission can occur, but only when aerosol generating procedures (AGP) are performed [7]. The WHO-defined AGPs partly include: positive pressure ventilation; tracheal intubation; airway suctioning; nebuliser treatment; and bronchoscopy [7, 9]. Consequently, the advice of the WHO is for droplet precautions to be observed for all suspected patients, with the addition of airborne precautions around AGPs [7].

Mechanisms of airborne viral particle formation

There are three mechanisms that describe the formation of respiratory airborne particles. All necessitate surface tension disruption of the respiratory tract lining fluid [10, 11].

1 Open-close cycling of glottic structures (> 1 μm diameter)
2 Shearing forces due to high velocity gas flow (2–5 μm diameter)
3 Open-close cycling of terminal bronchiole airways (< 1 μm diameter)

Based on composition analysis, exhaled particles have been demonstrated to come from lower rather than upper respiratory tract origins [12]. Viral growth in-medium has been demonstrated from particles < 5 μm produced from infected humans [13–17]. Infected human subjects produce a greater number of particles when coughing compared with healthy controls; furthermore, particles from infected patients contain viable virions [13, 14, 16, 18, 19]. If inhaled, particles in the range < 5–20 μm have the ability to reach the respiratory portion of the airways [19–23].

Gas flow velocities vary with type of exhalation. Tidal volume breathing may generate airflow velocities up to 1 $m.s^{-1}$; talking 5 $m.s^{-1}$; coughing 2–50 $m.s^{-1}$; and sneezing > 100 $m.s^{-1}$ [10,24]. The explosive shear forces generated from coughing and sneezing lead to expulsion of large numbers of varyingly sized particles and the highest number of particles, but significant numbers and a range of sizes are produced during talking and even tidal volume breathing [10, 21, 25–28]. Exhaling to closing capacity has been strongly correlated with significant aerosol production [11, 29].

Particle exhalation and deposition

Exhalation creates a jet with a cone-shaped geometry. Sneezes and coughs can form a turbulent multiphase gas cloud protecting the droplets from evaporation. This may extend the lifespan of a droplet allowing it to travel further [30]. This cloud can travel up to 8 m, carrying a polydispersed range of droplets. Eventually the cloud loses momentum and the remaining droplets evaporate forming droplet nuclei that remain suspended for hours with the ability to cause longer-range infectious transmission [30, 31].

Mechanisms of particle deposition within the atmosphere and airways partly depend on particle diameter. Diameter is a constantly changing variable due to the effect of humidity. As a particle leaves the respiratory tract, the relative humidity decreases and a rapid decrease in particle diameter of 25–50% occurs. This process is reversed on inhalation of a particle [20, 32].

The distance particles may travel is dependent on numerous variables, making it impossible to precisely define a safe distance to avoid transmission [26, 30]. The number of particles reduces with an increasing distance from the source. Larger particles generally take a ballistic trajectory, travelling shorter distances, and smaller particles remain suspended indefinitely. Larger particles are subject

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to inertial impaction and gravitational settlement, governed by Stokes' Law, and smaller particles to diffusion described by Fick's law [10]. Depending on the droplet's density, aerodynamic diameter and momentum, droplets may move faster, slower or at the same speed as the airstream with which they are exhaled [10, 26]. When encountering a barrier, the stream will typically be deflected or bifurcate [24].

The site of particle deposition in the airway may depend on: (1) particle aerodynamic diameter, shape, velocity, charge, composition, density, temperature and humidity; and (2) subject-specific variables, disease and airway geometry [10, 20, 32]. Increased temperature and humidity have both been shown to increase the rate of respiratory viral decay. This is likely a factor in seasonal and regional differences in respiratory infections [33]. Even heat from the patient and healthcare worker will alter airflows due to thermal air-currents or plumes [34]. Determinants of airborne viral concentration are displayed in Fig. 1.

During inhalation negative pressure creates airflow in a spherical breathing zone around the mouth and nose. A 500-ml breath will generally draw gas from a radius approximately 10 cm from the healthcare worker's mouth. The nasopharynx filters some particles including aerosols, but mouth breathing involves less filtration. Approximate hourly healthy adult alveolar ventilation is over 200 l of air, which will be in contact with an alveolar surface area of 750 m^2 [23]. This is a large volume of gas which may carry a viral inoculum.

The airborne particle size continuum

The WHO 5-μm size threshold used to differentiate droplet from airborne transmission is an over-simplification of the multifactorial mechanisms governing aerosol dispersal and deposition [7]. It is not clear if 5 μm refers to the diameter obtained experimentally (which varies with measurement method and environmental conditions), or at which stage in the dynamic airborne journey. There is heterogeneity between individual subjects and between experimental methodologies with regard to particle size and number measured during expiration. Due to irregular particle geometric shape, 'aerodynamic diameter' is the preferred term which assigns a diameter as if the particle were a perfect sphere. The median aerodynamic diameter and the geometric standard deviation are more predictive of particle deposition than 'simple' diameter [20].

It is demonstrable that larger particles tend not to reach the respiratory airways but the exact particle size that determines this cannot be defined [20, 22, 25, 30, 32, 35]. There may be outliers from the median distribution that will deposit more deeply in the airway than the average. These particles may carry a disproportionately large viral inoculum due to their volume. Measuring the aerodynamic diameter of particles and determining exactly where in the lung they deposit is challenging. Rather than defining an exact 5-μm diameter cut-off to define droplet or aerosol spread, lung particle deposition should be considered a continuum under which variables define the risk of lung deposition.

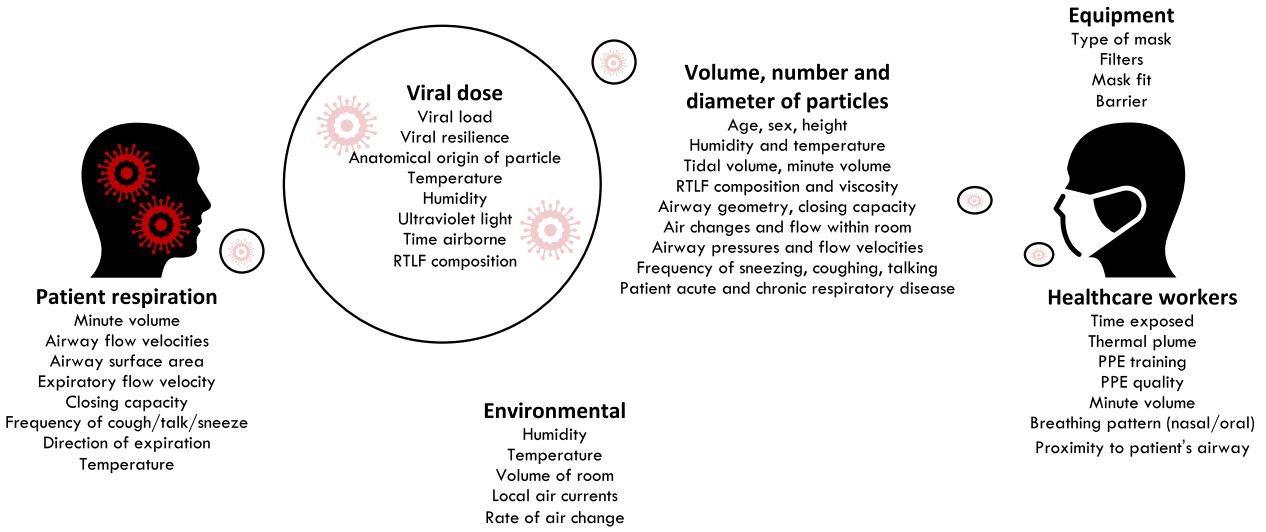

Figure 1 Key determinants of healthcare worker aerosol transmission in spontaneously breathing patient. RTLF, respiratory tract lining fluid; HCW, healthcare worker; PPE, personal protective equipment.

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SARS-CoV-2 airborne transmission

In human influenza models, aerosol inoculation is associated with increased disease severity and lower (rather than upper) respiratory tract infection, and may transmit infection even in a one-hundred-fold lower inoculum size [14, 18, 22, 25, 36–39]. Air sampling studies in commercial aircraft and health centres during influenza season demonstrated significant numbers of viral genome copies within airborne particles. The airborne viral content was calculated to be in excess of the minimal infectious dose [40]. Medical students contracted SARS-CoV-1 despite being considerably over a meter away from the hospitalised index patient. Post-hoc modelling postulated airflows that could have carried aerosols causing viral transmission [41]. An epidemiological study of SARS-CoV-1 using airflow modelling suggested that residents of a tower block were infected by airborne spread via a rising plume of contaminated air in a ventilator shaft [31]. During the same epidemic viral ribonucleic acid (RNA) was sampled from air within a patient's room [42].

Caution is required when directly inferring specifics of transmission from one respiratory virus to another as each has its own infective inoculum and aerosol characteristics. The SARS-CoV-2 virus uses the S-spike protein to bind to the angiotensin-converting enzyme-2 (ACE-2) receptor. Angiotensin-converting enzyme-2 has significantly greater expression on the surface of alveolar type-2 epithelial cells compared with bronchial epithelial cells [43]. The alveolar epithelium has less protection due to a thinner respiratory tract lining fluid, providing more direct access to the ACE-2 receptor possibly facilitating infection [43, 44]. Severe acute respiratory syndrome coronavirus-2 remains stable in artificially generated aerosols (< 5 μm) for up to 3 hours whilst maintaining an infectious titre [45].

Viral SARS-CoV-2 RNA have also been isolated on a ceiling extractor fan in a patient's negative pressure room where no AGPs had been reported [46]. Pre-submission articles, yet to be peer-reviewed, are suggestive of airborne RNA from normal breathing, the significance of which is undetermined (Liu et al., Chia et al. unpublished observations). Viral RNA in aerosol-sized particles in public, staff and clinical areas have been reported (Liu et al. unpublished observations). Levels were notably elevated in the protective apparel removal (doffing) room and patient toilets. Levels were lower in the intensive care unit, perhaps due to increased ventilation, and the peak size of particles was in the sub-micron range (0.25–1 μm) (Liu et al. unpublished observations).

It may prove difficult to unequivocally establish whether SARS-CoV-2 is infectious when airborne due to technical difficulties associated with air sampling of viable viral particles, and human-to-human transmission study being unethical. Current arguments against this and supportive of airborne transmission are displayed in Fig. 2.

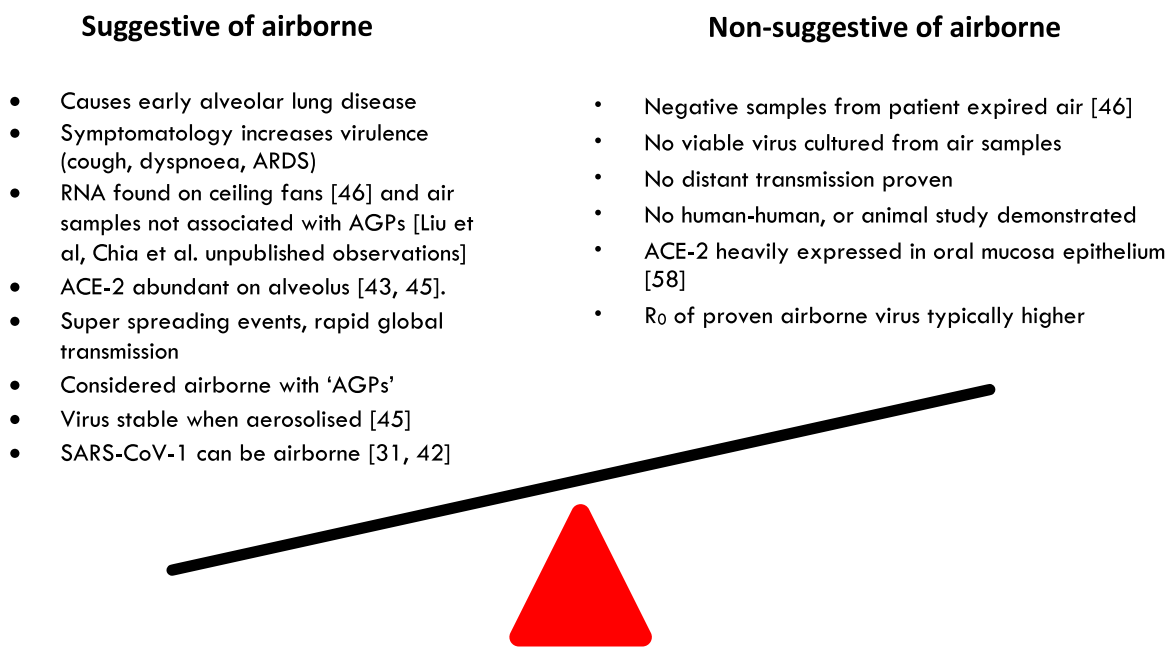

Figure 2 Evidence for and against airborne transmission of Severe acute respiratory syndrome coronavirus-2. ARDS, acute respiratory distress syndrome; ACE, angiotensin-converting enzyme; AGP, aerosol-generating procedure.

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Aerosol-generating procedures

A number of studies have shown an association between AGPs and healthcare worker infection during the SARS-CoV-1 epidemic. These are retrospective cohort studies and case series with multiple confounding factors, including: recall bias from retrospective questionnaires; variation in PPE; hand washing and training; incomplete follow-up; and small study sizes [1, 2, 4–6]. Crucially, aerosol levels were never measured in any of the studies. The authors of a systematic review of AGPs identified 10 studies suitable for inclusion, all of which were deemed 'very low-quality evidence' as per grading of recommendations, assessment, development and evaluations (GRADE) criteria [9]. Grading of recommendations, assessment, development and evaluations suggest caution when interpreting these results as 'any estimate of effect is very uncertain'.

An association is observed between healthcare worker infections and proximity to critically unwell patients who required emergency care [1, 2, 4–6]. Tracheal intubation was associated with a relative risk (95%CI) of healthcare worker infection of 4.2 (1.5–11.4), manipulation of an oxygen mask carried a relative risk of 9 (11.2–64) and urinary catheter insertion with a relative risk of 5 (2.4–10.2) [4, 9]. This may imply that physical proximity and time in the presence of a critical patient is high risk rather than the procedure per se.

Few studies have measured expired pathogen load in relation to AGPs [35, 47]. Particles containing viral RNA were found in the air around patients with influenza H1N1 in the intensive care unit, even during tidal volume breathing. The WHO-defined AGPs were not associated with a significant rise in airborne viral content, with the exception of bronchoscopy and in-line airway suctioning [9, 48]. Airborne viral content decreased with increasing duration of illness and with increasing relative humidity [47].

Shear stress and respiratory physiology

Surface tension occurs when two immiscible fluids share an interface. Across this surface of separation there is a discontinuity in density and the surface behaves like a stretched membrane under tension. Aerosol particle formation is dependent on shear forces across the airway walls overcoming respiratory tract lining fluid surface tension. The ratio of inertial to viscous forces described by the Reynolds number determines the likelihood of transition from laminar turbulent flow. Fluid velocity is increased by pressure difference and radius, and decreased by viscosity. As the velocity of gas flow rises, laminar shear forces will increase before a transition to turbulent flow with significant further increase in shear forces. Therefore, a higher differential between atmospheric and alveolar pressure causes a rise in respiratory tract lining fluid shear stress and increases aerosol particle formation [49].

Acute respiratory distress syndrome (ARDS) leads to alveolar inflammatory damage, compromise of lung mechanics and reduced respiratory function. Respiratory tract lining fluid composition is altered due to leucocyte infiltration and pulmonary oedema. Increased atelectasis, closing capacity and decreased compliance lead to a rise in pressure gradients to enable alveolar ventilation. During exercise, airway pressures may persistently swing from -30 cmH_2O to $+30cmH_2O$, with peaks in excess of 100 cmH_2O recorded [50]. It is likely that similarly high-pressure changes occur in spontaneously ventilating ARDS patients contributing to patient self-inflicted lung injury [50, 51]. Furthermore, distal airway collapse will lead to increased open-close terminal airway cycling, which also causes greater aerosol formation [11, 29].

Medical therapies and airborne transmission

Based on our interpretation of the current aerobiological and limited clinical evidence, we risk stratify key WHO AGPs with the addition of the 'natural' aerosol generators of coughing and dyspnoeic breathing in Table 1.

Formula A provides a simplified equation for the determinants of healthcare worker airborne risk. These can be mitigated by applying the methods listed in Table 2.

$$\text{Healthcare worker risk} \propto \frac{b \times v \times t}{e}$$

where:

b = breathing zone particle viable virion aerosol concentration

v = minute volume of healthcare worker

t = time exposed

e = mask efficiency.

Positive pressure ventilation

International airway management societies have developed guidelines to minimise the risk of healthcare worker COVID-19 transmission during tracheal intubation and extubation [52]. We defer to these, but offer some additional precautions based on the aerobiological literature summarised in Table 1.

Coronavirus disease 2019 patients with respiratory distress could produce high levels of aerosols secondary to cough; high airway pressures; minute volumes; altered

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Table 1 Procedures graded by risk of aerosol generation.

Aerosol generator	Applied physiology	Clinical evidence	Estimated risk of aerosol generation
Bronchoscopy	High airway pressures and distal airway collapse	Increased viral aerosols in H1N1 [35, 47]	Extreme
Percutaneous tracheostomy with bronchoscopy	High airway pressures and distal airway collapse with tracheostomy patent for unfiltered aerosols	Limited	Extreme
Bag-valve mask ventilation	Aerosol generation with high pressures and airway collapse	Associated with HCW transmission of SARS-CoV-1 [2, 4]	Technique-dependent
CPR	Airway collapse, shear forces from CPR, high airway pressures for ventilation	Strongly associated [6]	Extreme
Suctioning	Shear forces from significant negative pressure and flows. Causes coughing	Increased viral aerosols in H1N1 [47]	High
Frequent cough	Natural aerosol generator	Associated with HCW transmission of SARS-CoV-1 [1, 2, 4]	High
Dyspnoeic spontaneous respiration	Likely natural aerosol generator	Association with HCW transmission of SARS-CoV-1 [1, 2, 4]	High
Extubation	High risk due to coughing and distal airway collapse	Not studied	High
Laryngoscopy	Unlikely to cause aerosols per se	None showing rise in viral aerosols. Associated with HCW transmission of SARS-CoV-1 [2, 4]	Dependent on peri-intubation period
Oxygen facemask	De-humidified cold gas could promote viral viability.	Adjustment of mask strongly associated with risk of transmission of SARS-CoV-1 [2–4] Increased dispersal [24].	High – moderate
High-flow nasal cannula	Possibly reduce viral aerosols through decreased airway collapse and airway pressures. Unsealed circuit	Associated in limited quality studies. Used as part of Chinese COVID-19 protocol. Increased dispersal [53, 55, 56]	High – moderate
Non-invasive ventilation	Possibly reduce viral aerosols through decreased airway collapse and pressures. Sealed mask and circuit beneficial. High positive pressure may lead to leak	Association in limited quality studies. Used safely in small study [3]. Increased dispersal [24].	High – Moderate
Nebulisers	Alter the composition of RTLF and viscosity. Subject-dependent effect (24). Could reduce shear forces.	Associated in low quality studies. Increased dispersal [24].	High – Moderate

HCW, Healthcare worker; SARS, severe acute respiratory syndrome; CPR, cardiopulmonary resuscitation; RTLF, respiratory tract lining fluid.

secretions; and basal collapse. The same meticulous droplet and airborne precautions must be applied in these periods of close healthcare worker-patient contact as during the AGPs.

In a patient receiving non-invasive ventilation (NIV), airborne particle formation will be dependent on airway pressure differentials, gas flow velocities and open-close cycling of distal airways. The quantity of fugitive particles escaping into the atmosphere will depend on circuit, mask or hood leak, viral filters and minute volumes [34, 53]. During the 2003 epidemic, 20 SARS-CoV-1 infected patients were treated with NIV by over 100 health care workers. Using appropriate PPE, training and patient selection, zero transmission to healthcare workers was reported [3].

Spontaneously breathing patients exhale in a conical jet plume that is assumed to be at least 2 m in length, while

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Table 2 Precautions to prevent airborne transmission.

Environmental	Healthcare worker	Patient	Procedure
Increase room ventilation rates	Wear suitable PPE at times of transmission risk	Wear a surgical mask	Minimise shear stress on airways
If no formal ventilation system open windows and doors	Use a visor	Avoid coughing, sneezing, talking	Avoid airway open-close cycling
Increase temperature, humidity and UV light	Use the most efficient airborne mask protection available	Avoid high minute volumes, expiratory flows and volumes	Avoid bronchoscopy and CPR
Avoid small crowded rooms	Keep out of direct exhalation plume	Avoid atelectasis	Use fitted sealed masks or hoods with viral filters
	Minimise time in close contact with patient		Minimise suctioning
	Breathe nasally and reduce minute volume		Prevent coughing

PPE, personal protective equipment; UV, ultraviolet; CPR, cardiopulmonary resuscitation.

healthcare workers inhalation will be drawn from 10 cm around the face. Whenever possible, healthcare workers should stand over 2 m away and out of the direct exhalation plume. During a rapid sequence intubation, neuromuscular blockade should be protective as coughing will be prevented and high airway gas flow and expiratory output will terminate. When expiratory flow is ended, as shown by absent respiratory effort and flat end-tidal carbon dioxide trace, aerosol particles should start settling in the airways. The forces generated in gentle laryngoscopy are unlikely to cause aerosol formation. Suction typically generates a negative pressure of 100–200 cm H_2O and is associated with a measured rise in H1N1 aerosol particles [47].

The scalpel incision, insertion of a gum-elastic bougie and tracheal tube as part of an emergency surgical front-of-neck airway is unlikely to specifically generate aerosols per se. However, the newly formed cricothyroidotomy will immediately allow the escape of un-viral-filtered gases which will likely be high in aerosols due to recent high airway pressures and atelectasis. Extreme caution must be taken to minimise unfiltered gas leak through the new cricothyroidotomy and tracheal tube. In a 'cannot ventilate cannot oxygenate' scenario, the airway operator must avoid high pressures or volumes [52, 54].

Oxygen facemasks, nebulisers and high-flow nasal oxygen

Facemasks act as barriers to high velocity particle plumes, leading to redirection and dispersal of aerosols. The distance the exhaled plume will travel is reduced to as low as 0.1–0.4 m with the application of a facemask [24]. If the mask has an exhalation port gas will move directly out of this. Increased gas flow in the proximity of a patient should not increase the number of aerosols produced. It will disperse the expired tidal volume and plausibly increase the range of particles. Humidity is known to increase viral decay, so dry compressed gas potentially could increase viral viability.

Nebulisers increase the distance that an exhaled smoke jet plume will travel to 0.8 m [24]. Moistening the upper airways could increase the larger droplets produced. It is plausible medical-aerosol particles could collide with patient respiratory-airborne particles in the mask, becoming larger droplets and therefore travelling a shorter distance. If a bronchospastic patient generates marked intrathoracic pressures, this will theoretically increase the production of aerosols. Human laboratory studies have shown significant unexplained heterogeneity in the respiratory particle output of individuals. When given saline 3% nebulisers, high particle output producers considerably reduced aerosol output, whereas those who produced small numbers of particles at baseline exhibited a rise. The overall effect was a marked drop in aerosols as the high particle producers contribute more to the total output [19]. The benefit of using a nebuliser vs. the limited evidence against should be considered.

High-flow nasal oxygen will disperse a concentrated jet of aerosols, potentially spreading them over a further distance, in a more dilute concentration. It provides humidification which can reduce viable virus load and if inspiratory pressures and minute volume are reduced, this is aerosol-protective. However, unlike a continuous positive

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airway pressure (CPAP) mask or hood, there is no sealed exhalation path through a viral filter. At higher flows, for example 60 $l.min^{-1}$, it is plausible this could generate local turbulence driven droplets within the oropharynx which will be flow rate dependent. It is important to note that this generates flows significantly less than a cough [53, 55]. High-flow nasal oxygen was used by physicians in China as a standard part of escalating respiratory support in the current pandemic to good effect [56, 57].

Cardiopulmonary resuscitation

Distal airway collapse, chest compressions, suctioning, unsecured bag-mask ventilation and multiple people in close proximity to the airway will all create a high risk of healthcare worker transmission of SARS-CoV-2. This was demonstrated from the previous SARS-CoV-1 experience where multiple healthcare worker transmissions were recorded from one cardiopulmonary resuscitation (CPR) event [6]. Efforts must be made to recognise deterioration and either escalate care or withhold CPR, if appropriate. In the event of a cardiac arrest secondary to respiratory failure in a COVID-19 patient, it must be considered whether the risk to staff is acceptable when balanced with the likelihood of the patient surviving to a good functional outcome.

Conclusions

Due to the numerous complex dynamic variables, 'droplet-airborne' spread should not be viewed as a dichotomy based on exact particle size and specific safe distances, but as a continuum over which probability of lung inoculation alters. Coughing, talking and tidal volume breathing produce respiratory tract lining fluid-derived particles which could be inhaled into a respiratory portion of the lung [10, 11]. The mechanisms of SARS-CoV-2 transmission are currently undetermined leaving a potential role for airborne infection [7]. We speculate the respiratory pathophysiology of COVID-19 could increase exhaled infectious particles. These particles could gain direct access to alveolar surface ACE-2 receptors and transmit lung infection under suitable biological, physical and environmental conditions [58].

There is limited evidence to suggest AGPs cause an increase in airborne healthcare worker transmission, as this has not been studied. The few studies to sample pathogenic airborne particles in relation to procedures show no increase with the majority of AGPs [35, 47]. Several of the AGPs have been shown to be periods of high risk to healthcare workers but the exact timing and cause of transmission is unknown [9]. We observe an association between time in close proximity to SARS-CoV-1 patients requiring emergent respiratory therapy and increased staff transmission [1, 2, 4–6]. Therefore, we would not limit meticulous airborne precaution to the procedural periods alone but increase this protection to all times of risk. Unfortunately, the specifics of what defines a high-risk patient or activity remain undetermined. We have identified potential key determinants of airborne transmission (Fig. 1), which we combine with the limited known clinical evidence to risk stratify natural and medical aerosol generators (Table 1).

We speculate that in patients with a high viral load, respiratory symptoms and procedures that increase airway shear forces, open-close airway cycling and un-viral-filtered expired minute volume would increase risk. Conversely, certain AGPs employing enhanced techniques and equipment could minimise aerosol production compared with a coughing patient with a high work of breathing. However, the existence of poorly understood asymptomatic 'super-spreaders' highlights our knowledge-gaps and a need for sustained vigilance during a pandemic.

The environmental, healthcare worker, patient and procedural measures for mitigating airborne risk (Table 2) will deter 'direct-droplet' transmission reflecting the continuum across which these modes sit. Some of these measures can be applied without the addition of further PPE or cost. Given a global shortage in airborne protective equipment, regional centres must rationalise its use by appraising the current evidence and applying this to the risk of local transmission.

In the aftermath of the current pandemic, the exact mode of transmission may still remain controversial as was the case with SARS-CoV-1 and influenza. Urgent further research is required to investigate SARS-CoV-2 transmission, risk factors and strategies to assure the safety of healthcare workers. In the interim, healthcare workers may choose to take a precautionary approach until robust evidence is available.

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