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Real-life measurement of size-fractionated aerosol concentration in a plethysmography box during the COVID-19 pandemic and estimation of the associated viral load

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SUMMARY

Introduction: There are concerns about pulmonary function tests (PFTs) being associated with aerosol generation and enhanced virus transmission. As a consequence, the number of PFTs was reduced significantly during the coronavirus disease 2019 pandemic. However, there are no robust data supporting this fear.

Objectives: To perform real-life measurement of aerosol concentrations in a PFT laboratory to monitor the concentration of particles near the patient, and to model the associated potential viral load.

Methods: Two optical particle counters were used to sample the background concentration and the concentration of particles near the patient's mouth in a whole-body plethysmography box. Statistical evaluation of the measured particle concentration time series was completed. The particle exhalation rate was assessed based on the measured particle concentration data by applying the near-field/far-field theory. The number of exhaled viruses by an infected patient during the test was compared with the emission of viruses during quiet breathing and speaking.

Results: Twenty-five patients were included in the study. Eighteen patients showed a significant increase in aerosol concentration [mean 1910 (standard deviation 593) particles/L]. Submicron particles dominated the number size distribution of the generated particles, but large particles represented a higher volume fraction in the generated particles compared with background. An average gene exhalation rate of 0.2/min was estimated from this data. This is one order of magnitude higher than the release rate for the same infected person during quiet breathing, and of the same order of magnitude as the release rate during normal speaking.

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Conclusions: This study demonstrated that PFTs are aerosol-generating procedures. Based on these results, the moderate increase in viral load does not underpin stopping such examinations.

Introduction

Pulmonary function tests (PFTs) are important and necessary diagnostic tools in respiratory medicine; however, there are concerns that forced breathing manoeuvres may increase the amount of particles (droplet and aerosol) exhaled by the patient. In addition, atypical breathing can provoke coughing, which may also be associated with increased particle exhalation. As patients do not wear a facemask during PFTs, the chances of pathogen transmission may be increased. There is increasing evidence that an important route of severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) transmission is via inhalation of virus-containing droplets and aerosols [1,2], which deposit in different regions of the airways [3]. Therefore, shortly after the onset of the coronavirus disease 2019 (COVID-19) pandemic, the number of PFTs was reduced drastically or even stopped in some places. National and international medical and respiratory societies (e.g. European Respiratory Society, Association for Respiratory Technology and Physiotherapy) released statements and position papers with warnings on the possible increased risks linked to PFTs, and recommended different measures for risk mitigation. Some of these recommendations were updated as experience and knowledge accumulated [4]. However, the precaution is based, in part, on theoretical considerations rather than a solid experimental basis. In the months following the first peak of the pandemic in 2020, efforts were made to reveal whether PFTs are significant sources of aerosol generation, and whether or not the increased risk of transmission associated with PFTs is real [5—8]. The values of the increased particle concentration attributable to PFTs documented in the above studies range from a few hundred particles up to several thousand particles per litre, but there were notable differences between the studies regarding the circumstances. On the other hand, speaking without a facemask may also produce an additional load of a few hundred particles per litre, and it has been demonstrated [9] that intersubject variability in the concentration of aerosol generated is more than an order of magnitude. To date, there are only a few results regarding the number of particles attributable to PFTs. Some related studies concluded that aerosol generation during PFTs is significant, but the excess risk of infection due to these particles remains unknown. Moreover, in all but one of the previous studies, the subjects were not real patients, and the conditions were more or less representative of 'real-life' (e.g. waiting artificially long times to reach the background particle level; use of ultraclean, laminar flow theatre for measurements).

The aim of this study was to contribute to the evidence base related to PFT measurements during the COVID-19 pandemic and other pandemics by performing real-life aerosol concentration monitoring without any artificial intervention or change during working hours in a PFT laboratory. The measurements were taken during standard tests performed by medical professionals on real patients (with proven disease or during diagnosis) in order to capture the possible changes in the realistic aerosol environment due to the examinations. Another aim was to use the results of the measurements and data from the literature to estimate the potential risk associated with PFTs during the pandemic.

Methods

A single-centre observational study was conducted in the whole-body plethysmography box in the PFT laboratory at the Department of Pulmonology, Semmelweis University, Budapest, Hungary on 28 July 2020. The plethysmography box (PDT-111/pd, Piston Medical) had dimensions of $0.7\times0.9\times1.7$ m and it was located in a room with dimensions of $5.6\times3.5\times3.0$ m. Figure 1 shows the layout of the room, including the positions of the doors, windows, body box and other major objects.

The room air characteristics were monitored by a mobile temperature and humidity data logger (Testo 174H, Testo SE & Co, KGaA). The same quantities were monitored automatically by built-in sensors inside the body box for the correction of spirometric data. There was no air conditioner in operation during the measurements. The windows of the laboratory were half-opened. The doors of the laboratory were usually closed, but the patient entrance door was opened every time a patient entered or left the room. The air exchange rate of the laboratory was 6.5 times per hour. The door of the plethysmography box was opened between two tests, and closed or opened during the measurements depending on the type of measurement. Normal spirometry, lung volume measurement (plethysmography) and diffusion testing [diffusing capacity of the lung for carbon monoxide (DLCO)] were conducted by a PFT technologist standing outside the plethysmography box while the patient was sitting in the plethysmography box. Two observers sat in the laboratory (outside the transparent cabin at 2 m distance) and documented the timeline of the relevant events and conditions. All medical personnel and observers wore FFP2 facemasks throughout the measurement period. Each patient took off their facemask exclusively during their stay in the plethysmography box. Disposable bacterial and viral filters (PBF-100) were used.

The study participants were volunteers and provided written consent. The study was completed based on ethical approval (no. SE RKEB 212/2020).

As the study aimed to observe real-life circumstances, there was not always sufficient time to ensure that the particle concentration reached a relatively constant background level (as in some previous studies) between the tests. Therefore, it was necessary to use two similar sampling devices in parallel, with one device measuring changes in background particle concentration, and one device measuring the aerosols near the patient. For this purpose, two identical optical particle counters (OPCs; Grimm Aerosoltechnik, Portable Aerosol Spectrometer, model

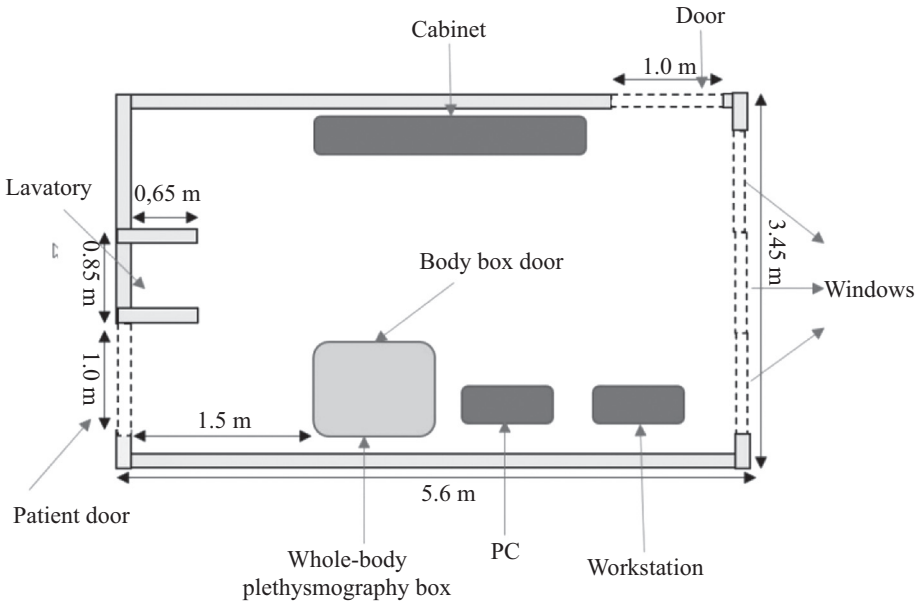

Figure 1. Layout of the pulmonary function testing laboratory.

1.109) were used with 6 s time resolution. The size distributions were recorded in 31 size bins between 0.25 and 32 μm. The upper concentration limit (<5% coincidence error) of the instrument is 2 million particles/L, which was not expected to occur in a PFT laboratory in the given size range. The small size of the OPC devices (24 × 13 × 7 cm^3) and their quiet operation made them suitable for the measurements. OPC-A had a fixed position inside the cabin in the furthest possible position from the source [i.e. mean 140 (SD 15) cm from the mouth of the patient, depending on the patient's height]. Based on previous studies [6], it was not expected that the particle concentration would increase 1.4 m from the patient, so OPC-A provided the background concentration in the cabin. OPC-B sampled the aerosols inside the body box while the patient was inside the cabin (30 cm from the patient's mouth) and inside the laboratory but outside the cabin when the cabin was empty (between two measurements).

Statistical evaluation of the measured particle concentration time series was performed using OriginPro 2021 Version 9.8.0.200. Background concentration and near-patient concentration time series were compared using two-sample *t*-tests. Concentration time series measured outside and inside the box were compared by correlation analysis (Pearson coefficient). The agreement between the two devices (OPC-A and OPC-B) sampling the same environment at the same time was verified using the Bland–Altman test [10].

As virus detection was not completed within the present work, only a theoretical estimation of the number of emitted gene copies was performed in this study. As the number of viable viruses is two or three orders of magnitude lower than the total number of detectable viruses [11], this study used 'gene copies' instead of 'virus copies'.

As the number of exhaled gene copies depends on many factors, a comparative estimation was performed by comparing the number of copies due to PFTs with those that would be emitted by the same person in the same environment, but breathing normally or speaking. For evaluation of the number of gene copies that an infected patient may emit during a PFT, it was necessary to evaluate the emitted particulate mass (at the patient's mouth) starting from the data obtained 30 cm from the subject's mouth. For this purpose, the equations of the 'near-field/far-field' well-mixed room model were used [12,13]. The time-dependent mass concentration of the emitted particles was expressed as the sum of near-field and far-field concentrations obtained by solving mass balance equations:

$$c = c_{N-F} + c_{F-F} \tag{1}$$

where:

$$c_{N-F} = \frac{g}{\beta}\left(1 - e^{-\frac{\beta}{V_{N-F}}t}\right) \tag{2}$$

and:

$$c_{F-F} = \frac{g}{q + \frac{V_{F-F}\ln(2)}{t_{\frac{1}{2}}}}\left(1 - e^{-\left(\frac{q}{V_{F-F}} + \frac{\ln(2)}{t_{1/2}}\right)t}\right) \tag{3}$$

In Equation (2), g denotes the rate of particle generation by the patient (mass/time), β is the interzonal flow rate (volume/time) between near-field and far-field zones, t is the time from the exhalation of particles, and $V_{N\text{-}F}$ is the volume of the spherical near-field zone (with 0.6 m radius). The interzonal flow rate (volume/time) was expressed as:

$$\beta = 0.5 \times S \times u_{avg} \tag{4}$$

where S is the surface area of the above-defined sphere and u_{avg} is the average wind speed in the room (0.1 m/s). In Equation (3), $V_{F\text{-}F}$ denotes the volume of the PFT laboratory (58 m^3), q is the room ventilation rate (5.6 m^3/min), and $t_{1/2}$ is the virus half-life on aerosols. In this study, the value of $t_{1/2} = 66$ min was adopted from the publication by van Doremalen *et al.* [14]. For the near-field component, the loss due to virus inactivation [see Equation (2)] was neglected because it is much less than the loss due to interzonal air exchange. In addition to the exhaled particle losses due to near-field and far-field air exchange, the detected mass of

particles may also be lower because of gravitational settling. A 32 μm-diameter particle (which was the upper size limit of the sampled particles) may fall outside of the near-field zone within 20 s, and a 10 μm-diameter particle may fall outside of the near-field zone within 200 seconds [15]. As the examinations took, on average, 2.5 min, it is plausible to consider the PM10 fraction of the particles alone, and to compare the number of estimated viruses in PM10 emitted during PFTs with the available data on virus load of PM10 due to other types of activities [16]. Another relevant phenomenon is droplet evaporation. As data on viral load are available for the emitted mass, it is important to convert the sampled mass into emitted mass by also considering evaporation. The time needed for a droplet containing water and non-volatile solutes to reach its equilibrium size can be estimated by the formula:

$$\tau_{ev} = \frac{D_0^2}{4\theta(1-RH)}\left[1-\left(\frac{\phi_0}{1-RH}\right)^{2/3}\right] \quad (5)$$

where D_0 is the initial diameter of the droplet, θ is 4.2×10^{-10} m^2/s [15], RH is the relative humidity, and ϕ_0 is the initial volume fraction of solutes in the droplet. The value of ϕ_0 may vary depending on the NaCl, surfactant and protein content of the droplet. In this work, the value of 0.025 was considered, which is the average of the volume fraction values characteristic of saliva droplets with low (3 mg/mL) and high (76 mg/mL) protein content [17]. The final (after evaporation) diameter of the droplets can be estimated by the expression:

$$D_{ev} = D_0\left(\frac{\phi_0}{1-RH}\right)^{1/3} \quad (6)$$

A conservative approach has been applied for the estimation of the particulate mass that an infected patient may emit during a PFT assuming that all the generated particles originate from exhalation by the patient. By the same token, τ_{ev} defined in Equation (5) was low in comparison with the duration of the PFT, implying that all the droplets have already reached their equilibrium size when detected. All these approximations led to the highest possible particle mass at the patient's mouth (i.e. the upper bound of the number of viruses the patient may have emitted). The number of viruses was estimated by assuming that each millilitre of emitted particles contains 10^6 gene copies, which is characteristic of a normal emitter [18]. The number of viruses generated during a PFT was compared with the number of the viruses that the same person would emit in the same environment during normal breathing or speaking.

Results

Twenty-five patients were involved in the study, suffering from different diseases: two asthmatics, eight transplant patients, four patients with chronic obstructive pulmonary disease, one patient with cystic fibrosis, two patients with idiopathic pulmonary fibrosis, two patients with interstitial lung disease, one smoker suspected of asthma, two subjects suspected of tuberculosis, one patient with lung cancer, one patient with sarcoidosis, and one patient with pulmonary hypertension. The total number of measurements was 27, as two measurements were made on two of the patients (reversibility test). One participant (with sarcoidosis) was excluded because the increase in aerosol concentration (almost two orders of magnitude higher than the average increase) was generated by cloth handling by the patient. The measurements for the remaining 24 patients are summarized in Table I.

The 24 patients (12 males and 12 females) had a mean age of 57.7 [standard deviation (SD) 17.3] years and mean body mass index (BMI) of 24.1 (SD 5.8) kg/m^2. Mean forced expiratory volume in 1 s (FEV_1) was 2.0 (SD 0.9) L [70.5 (SD 29.4)%] and mean forced vital capacity was 2.8 (SD 1.1) L [81.0 (SD 22.8)%]. The mean duration of the examinations was 2.6 (SD 1.1) min. Mean ambient room humidity was 37.9 (SD 2.3)% and mean temperature was 23.4 (SD 0.9) °C. Throughout the measurement period, the mean ambient levels of total concentration corresponding to the monitored daily routine were [144 (SD 1.2)]$\times10^3$ L^{-1} and [142 (SD 1.1)]$\times10^3$ L^{-1} in the laboratory and in the body box, respectively. Higher fluctuations occurred after opening the door of the laboratory when a patient entered or left. As the box door was open between measurements, the trends of the concentration time series in the box without a patient systematically followed the trends in the laboratory ($r>0.92$). In addition, the two devices measured the same background concentration values inside the empty plethysmography box (Pearson test: $r>0.99$; Bland–Altman test: mean of the differences = 58 particles/L with no dependence of the difference on the magnitude of the concentration, linearity was confirmed, and the results of the two devices were on the equality line). According to the results of the measurements without a patient in the box, approximately 99% of the ambient particles were <1 μm in geometric diameter, and their mass fraction (PM1) represented 22.5% of the total mass of the sampled particles (<32 μm).

In eight cases (31% of the total number of measurements), the patient-specific total concentration values, calculated as the concentration of 0.25–32 μm particles measured by OPC-B averaged over the duration of patient stay in the cabin, were not significantly higher than the background concentration measured simultaneously by OPC-A. In 18 cases (69% of the total number of measurements), the total concentration increase was significant ($P=0.05$). Figure 2 demonstrates the total concentration values measured in parallel by the two devices in these 18 cases.

The mean total concentration increase was 1910 (SD 1018) particles/L. No statistically relevant correlation could be demonstrated between the type of disease and particle concentration enhancement due to the measurement. The correlation between FEV_1 and FVC and the increase in particle concentration was also weak. The concentration increase was usually higher for longer PFT durations, but the correlation was weak ($r=0.34$). In addition, at this sample size, the three types of examinations did not result in significantly different increases in particle concentrations. The left panel of Figure 3 depicts the average number of particles of different sizes generated by 16 patients (18 measurements). As the figure demonstrates, submicron particles dominate in the newly generated particles. However, the number of gene copies in infected cases is correlated with droplet volume rather than with droplet number [18,19]. Therefore, it is plausible to consider the volume size distribution as well. The right panel of Figure 3 demonstrates the frequency of different volume fractions in the size distribution of the generated particles. As can be seen, most of the generated volume (and mass) is contained in particles with diameter >1 μm. A comparison of the volume size distribution of the generated particles with the same type of size distribution of the background particles

Table I
Demographic data, breathing parameters, type of pulmonary function test (PFT) and duration, and particle concentration characteristics of the participating patients

Pat. ID	Disease	Sex (M/F)	Age (years)	BMI (kg/m^2)	FEV_1 (L)	FEV_1 (%)	FVC (L)	FVC (%)	PFT type	PFT duration (min)	Conc. increase (L^{-1})
1.	Asthma	M	48	27.7	3.4	84	5.4	103	Plethysmography	2.1	895
					3.8	91	5.6	107	Plethysmography	1.9	1887
2.	Transplant	F	37	20.8	2.0	72	2.2	71	Plethysmography	1.9	2064
3.	Asthma suspect	F	40	31.6	2.3	77	2.8	80	Plethysmography	2.2	–
					2.4	79	2.9	82	Plethysmography	2.0	–
4.	Transplant	F	52	26.7	2.0	89	2.8	103	Plethysmography	3.1	1335
5.	Transplant	M	63	21.5	2.6	86	3.3	86	Plethysmography	1.1	–
6.	Transplant	M	63	21.5	1.5	42	1.9	43	Spirometry	2.0	1577
7.	Transplant	F	38	16.4	3.0	109	3.9	118	Plethysmography	5.0	–
8.	COPD	M	63	28.0	1.2	35	3.5	80	Plethysmography	4.9	4019
9.	Transplant	F	44	17.2	2.4	92	2.8	91	Plethysmography	2.2	2197
10.	Transplant	M	49	14.5	0.8	22	1.8	44	Spirometry	2.0	2365
11.	Transplant	F	40	16.9	0.6	20	1.5	48	Spirometry	1.2	1242
12.	ILD	F	87	22.0	0.4	24	0.8	43	Spirometry	2.3	3411
13.	COPD	M	36	27.4	2.8	70	3.8	80	Plethysmography	1.1	1289
14.	COPD	M	48	19.1	0.8	24	2.3	60	Plethysmography	2.2	–
15.	Asthma	M	36	27.4	3.1	77	3.9	82	Plethysmography	2.1	3818
16.	ILD	F	81	22.1	1.4	80	1.9	85	Plethysmography + DLCO	3.2	1182
17.	COPD	M	70	23.0	1.7	54	3.7	90	Plethysmography	2.2	630
18.	CF	F	41	21.9	1.8	64	2.4	72	Plethysmography	2.1	–
19.	Pulmonary hypertension	M	78	34.5	1.7	66	2.6	72	Plethysmography + DLCO	2.5	1374
20.	Lung tumour	M	89	19.3	1.6	120	2.0	117	Plethysmography + DLCO	4.1	2056
21.	IPF	F	68	26.4	2.0	79	2.6	88	Plethysmography + DLCO	3.9	–
22.	TB suspected	F	75	22.5	1.9	112	2.3	113	Plethysmography	3.0	545
23.	IPF	F	64	35.4	1.1	50	1.3	50	Plethysmography + DLCO	4.8	2486
24.	TB suspected	M	75	33.7	3.2	114	3.7	99	Plethysmography + DLCO	3.2	–

COPD, chronic obstructive pulmonary disease; ILD, interstitial lung disease; CF, cystic fibrosis; IPF, idiopathic pulmonary fibrosis; DLCO, diffusing capacity of the lung for carbon monoxide; BMI, body mass index; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; M, male; F, female.

reveals a higher relative volume of large particles in the generated aerosol than in the background aerosol. This observation is in line with the outcome of the measurements of Larsson *et al.* [20], who suggested that this is due to the formation of larger particles during forced exhalation in the central airways as a result of the interaction between air and the respiratory tract lining fluid.

The average PM10 mass detected in patients with significant particle generation was 1.4 ng/L. Based on Equations (1)–(3), this quantity is equivalent to a droplet emission rate of 8.3×10^{-9} mL/min assuming that droplets do not evaporate. However, under the conditions monitored in the PFT laboratory, the evaporation of droplets (reduction of their volume) did take place. Based on the time resolution of the current measurements (6 s) and the time needed for the droplets to reach their final size by evaporation [<1 s, see Equation (5)], it can be considered that droplets had sufficient time to evaporate to their minimum size. In such circumstances, Equation (6) can be applied to obtain the initial volume of the droplets, which yields a value of 2×10^{-7} mL/min for the release rate of fresh droplets, equivalent to the emission of 0.20 gene copies/min in the case of a normal emitter (1 mL of droplet corresponds to 10^6 gene copies).

Discussion

A number of mechanisms (e.g. airflow-driven fragmentation of visco-elastic filaments in the upper airways, elastocapillary bursting of fluid films in the bronchioli) by which droplets are created and emitted from different anatomical regions of the respiratory tract are still under scientific debate [21,22]. The amount and size of the emitted droplets depend on the region of origin in the airways and also on the type of activity (breathing, speaking, singing, coughing, sneezing). It is scientifically plausible to hypothesize that within the same type of activity, the intensity of the activity is also a key factor. For instance, it has been demonstrated that the amplitude of vocalization (normal speaking vs loud speaking, quiet singing vs

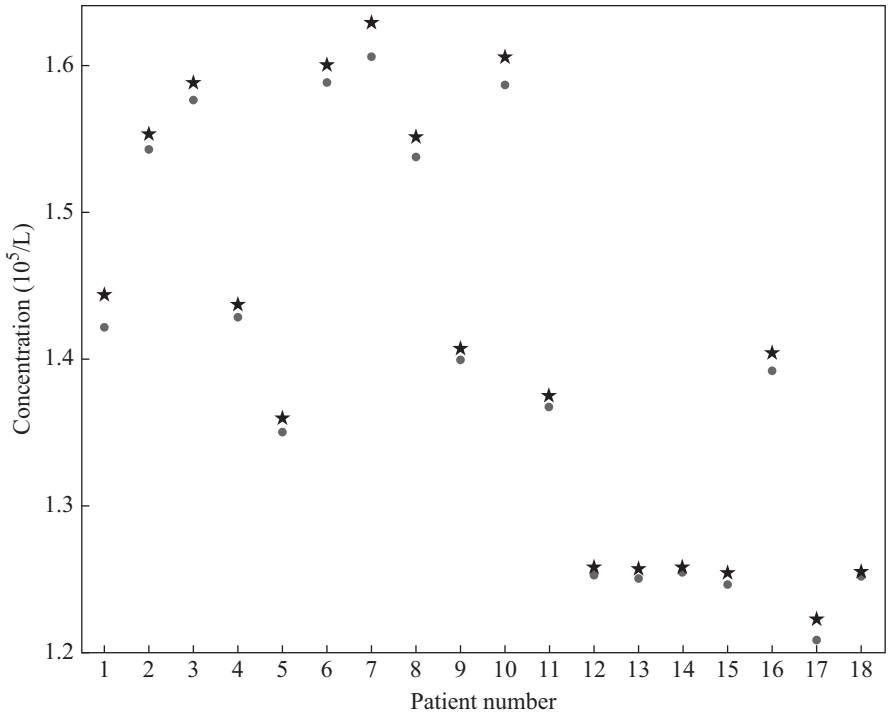

Figure 2. Total concentration of particles between 0.25 and 32 μm in the plethysmography box at 30 cm from the patient's mouth (stars, OPC-B, near-patient) in comparison with the background concentration (circles, OPC-A).

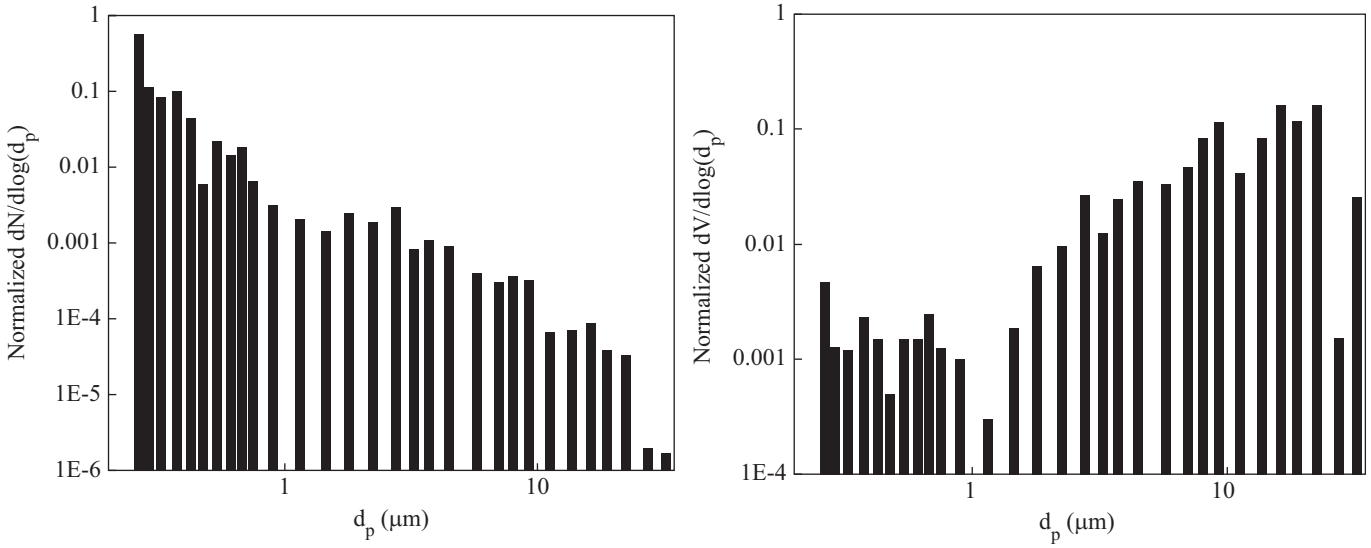

Figure 3. Number (left panel) and volume (right panel) size distribution of the generated particles in the case of 18 patients with a significant increase in particle number.

loud singing) affects the number of aerosol particles generated [1,9]. In a similar way, the mode of breathing (normal, deep, fast) may influence the size and amount of emitted droplets [20,23]. Forced breathing leads to higher air velocity and modified airway calibre, which affect the formation and emission of droplets by the abovementioned mechanisms. Based on this rationale, a PFT must be an aerosol-generating activity, and it is also likely that the number of emitted particles exceeds the number of particles emitted during quiet breathing. The present results seem to underpin this hypothesis, as a significant increase in particle concentration was detected in 69% of the measurements. The increase was slightly higher than that obtained by Sheikh *et al.* [8] but lower than that reported by Li *et al.* (2020) [7]. It is worth noting that four patients had a cough during the measurements, which may have further increased the number of particles generated. The present real-life measurements were completed at 15—20 min intervals based on the recommendations of the Hungarian Respiratory Society advising a maximum of four measurements per hour. Based on the observations of previous investigators

[7], this time interval may not have been sufficient to reach background levels, so the present results could contain an accumulation component. However, analysis of the measurement results showed that PFTs completed later did not generate a statistically relevant higher number of particles than the first tests. It is also important to note that the excess of particles was probably not the result of the forced expiration alone, but a combination of forced breathing, quiet breathing before the test, speaking in some cases, and even coughing for some patients. Nevertheless, these events are all at play during a PFT, so the data reflect the real-life situation.

The extent to which the surplus of particles due to PFTs can increase the risk of infection is an important question. When analysing this issue, it is worth considering that the increase in particle concentration was only significant for some of the patients and only at a short distance from the patient's mouth. In addition, the increase was small compared with the background concentration, and was less than its variation due to other patient-care-related activities. For instance, the measurements demonstrated that the increase in particle concentration could be of the order of 10^4 particles/L due to some activities in a hospital room, such as a medical visit or changing the bedding (data under publication). Similarly, the measurements during bronchoscopy indicated particle generation of the order of 10^3—10^4 particles/L (unpublished data). Flushing the toilet may generate up to 10^5 droplets/L of air [24], and a high SARS-CoV-2 load was detected in a toilet of a hospital [25].

Based on the model of Riediker and Monn [16] and their open online calculation tool, the number of gene copies released by a normal emitter during quiet breathing is 0.0085/min. Based on current results, this is approximately 25 times lower than the number of gene copies that the same infected person would emit during a PFT. The same model [16] predicts the release of 0.0855 gene copies/min for the same person while speaking normally. This means that the number of viruses emitted during a PFT is approximately one order of magnitude higher than the number of copies emitted by quiet breathing, and of the same order of magnitude as the copies released during normal speaking. Therefore, the viral load of a PFT is only slightly higher than the viral load of a routine medical examination with the patient speaking without a mask.

Obviously, the risk of infection can be decreased by a series of preventive measures. All the relevant information on the examination and all the possible instructions, except coaching during the test to optimize the patient's effort, should be given before the test while the patient is wearing a facemask. Regular ventilation (ventilation systems providing fresh air are advised instead of recirculators), use of air sterilizers with high filter efficiency and air turnover, frequent disinfection of the body box and the laboratory, use of disposable nose clips and mouthpieces/filters, longer turnaround time between testing, greater distance between the technician and the patient, and the use of facemasks (preferably FFP2) will all contribute to minimization of infection in the PFT laboratory during the COVID-19 pandemic, and beyond.

Limitations of the study

This study has a number of limitations. First, due to the operational complexity of the study, the number of patients was limited. As the study was observational, it was not possible to select many patients with the same disease, so there was a large span of diseases and heterogeneous underlying comorbidities. Therefore, it was not possible to have patient groups with large populations, which implies that the statistical power of the analysis is not very strong. Although the patients were not tested before the PFT, most of them were unlikely to have been infected by SARS-CoV-2 (they were asymptomatic). The viral load due to the PFT was calculated assuming the patient was a normal emitter, which was an approximation. It was also assumed that all the generated aerosol particles originate from the airways of patients, which may lead to overestimation of the viral load. In addition, the particle concentration was measured some distance from the patient's mouth, and the concentration of exhaled aerosol particles was determined theoretically based on these measurements, which may increase the uncertainty of the assessment.

In conclusion, in this observational study conducted under real-life conditions, the concentration of particles around the patients during PFTs was sampled successfully. The excess of particles due to lung function testing demonstrated that PFTs are indeed aerosol-generating procedures. The estimated number of viruses released by a hypothetical normal emitter revealed that the viral load of such a measurement is comparable to the load associated with the same person speaking without a facemask. The current results revealed that the excess risk due to PFTs is not negligible, especially taking into account that new variants (e.g. Delta variant) are more contagious. However, given that PFT measurements provide essential information on the status of the patient, stopping PFTs is not recommended. Evidently, all the activities related to PFTs must be carried out using preventive measures, considering all the possible safety measures.

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Conflict of interest statement
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