

Home ventilators for invasive ventilation of patients with COVID-19

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The coronavirus disease 2019 (COVID-19) pandemic has caused disruption in health systems all over the world. A considerable number of patients with COVID-19 need mechanical ventilation.^{1,2} It is therefore anticipated that there will be a surge in the number of patients requiring respiratory support and there may be a shortfall in the number of devices available.

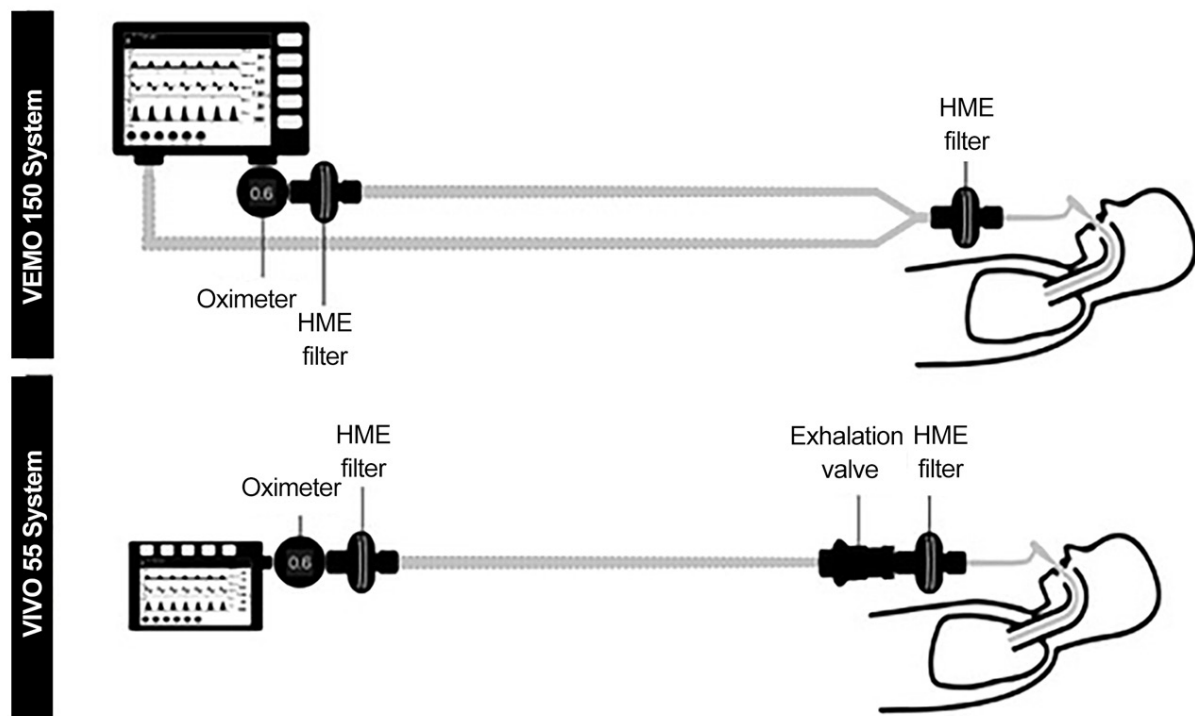
Increasing numbers of patients with chronic respiratory failure are being treated at home with small mechanical ventilators.³ Such ventilators are suitable for both airway stabilisation and ventilator support in patients with diseases ranging from chronic obstructive pulmonary disease to neuromuscular disease (eg, patients with spinal injury and patients with motor neuron disease). Given that the

number of such ventilators is considerable, the use of these machines to help ventilate patients with COVID-19 is appealing. In this case series, we report how ventilators designed for domiciliary support of patients with chronic respiratory failure may be used for invasive support in patients with COVID-19 needing mechanical ventilation.

Methods

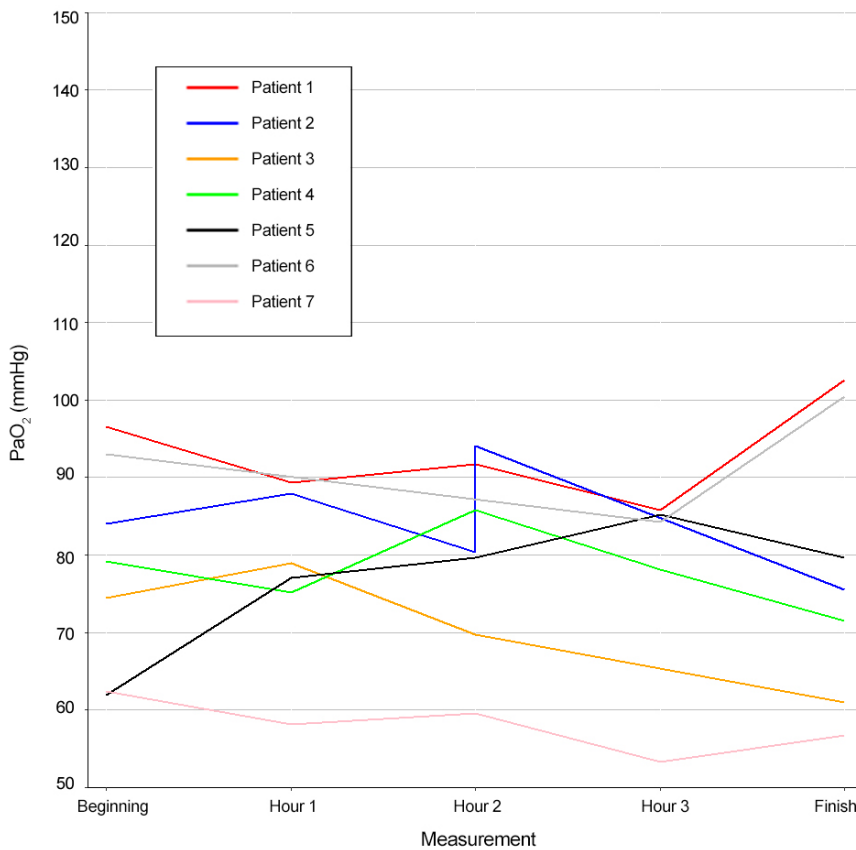
We studied a cohort of consecutive ventilated patients in COVID-19-dedicated intensive care units (ICUs) at San Raffaele Scientific Institute, Milan, Italy. In a proof-of-concept study, patients receiving invasive ventilatory support (at any stage of the disease) were transitioned for

Figure 1. Systems used for the two continuous positive airway pressure devices tested: VEMO 150 (EOVE, VitalAire, Italy) and VIVO 55 (Breas, MedicAir, Italy) ventilators



HME = heat and moisture exchanger.

Figure 2. Temporal changes in arterial partial pressure of oxygen (PaO₂)



Change in PaO₂ at the beginning using intensive care unit (ICU) ventilators, then hourly for 3 hours using ventilators designed for support of patients with chronic respiratory insufficiency, and at the end returning to ICU ventilators.

3 hours from the ICU ventilator to ventilators designed for support of patients with chronic respiratory insufficiency (the intervention period) and then back to ICU ventilators. Arterial blood gases were collected hourly during this period. In addition to descriptive analysis, we measured the coefficient of variation within each subject, calculated as the standard deviation divided by the mean.

Two devices were tested: the VEMO 150 (EOVE, VitalAire, Italy) and VIVO 55 (Breas, MedicAir, Italy) ventilators. The system used for each of the devices is described below. Supplementary oxygen was supplied at low pressure through the purpose-built inlet on both machines.

System for the VEMO 150

Outflow port → Oximeter → Heat and moisture exchanger (HME) → In tube → Y pieces → HME → Tracheal tube and back to the inflow port (Figure 1).

System for the VIVO 55

Outflow port → Oximeter → HME → In tube → Exhalation valve → HME → Tracheal tube (Figure 1).

Results

Over 10 days, from 28 March to 7 April 2020, seven patients were enrolled in this case series, including one patient with few measurements. Ventilatory variables are shown in Table 1.

The arterial partial pressure of oxygen (PaO₂) values at the beginning of the intervention ranged from 61.9 mmHg to 96.5 mmHg and, at the end, from 56.7 mmHg to 102.6 mmHg (Table 1 and Figure 2). From the beginning and during the intervention period, the variation in PaO₂ values ranged from 4.9% to 13.1%, with the highest increase being from 61.9 mmHg to 79.7 mmHg and the highest decrease from 96.5 mmHg to 85.8 mmHg.

No patient developed worse hypoxaemia during the intervention period. Ventilators designed for support of patients

with chronic respiratory insufficiency were able to offer a fraction of inspired oxygen (FiO₂) level from 40% to 86%, with oxygen flows ranging from 8 L/min to 20 L/min.

Similarly, Arterial partial pressure of carbon dioxide (Paco₂) (coefficient of variation, 3.0–9.3%) and pH (coefficient of variation, 0.0–0.3%) levels remained stable during the test, and a considerably increase in the Paco₂ level (from 58.4 mmHg to 72.5 mmHg) was observed in only one patient (Table 1). The patient developed fever and hypoxia unrelated to the intervention, which persisted even after return to the ICU ventilator.

Discussion

In this case series, seven critically ill patients with COVID-19 were ventilated with ventilators designed for support of patients with chronic respiratory insufficiency during a short period. As assessed by hourly arterial blood gases, PaO₂, Paco₂ and pH levels were kept constant

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Table 1. Clinical characteristics of the patients

	Patients						
	1	2	3	4	5	6	7
Clinical characteristics							
Age (years)	47	69	49	49	76	54	65
Gender	Male	Female	Male	Female	Male	Male	Female
Use of NMBA	Yes	No	Yes	No	No	Yes	No
Haemodynamic impairment	No	No	No	No	No	No	No
Tracheostomy	No	Yes	No	No	No	No	No
Ventilator*							
Beginning	Servo-i	Servo-i	EVITA 4	EVITA 4	Servo-i	EVITA 4	Servo-i
Hour 1	VEMO 150	VIVO 50	VEMO 150	VIVO 50	VIVO 50		VEMO150
Hour 2	VEMO 150	VIVO 50	VEMO 150	VIVO 50	VIVO 50		VEMO150
Hour 3	VEMO 150	VIVO 50		VIVO 50	VIVO 50	VEMO 150	VEMO150
Finish	Servo-i	Servo-i	EVITA 4	EVITA 4	Servo-i	EVITA 4	Servo-i
Ventilatory mode[†]							
Beginning	AutoFlow	PSV	AutoFlow	PSV	PSV	AutoFlow	PCV
Hour 1	VCV	PSV	VCV	PSV	PSV		PCV
Hour 2	VCV	PSV	VCV	PSV	PSV		PCV
Hour 3	VCV	PSV		PSV	PSV	VCV	PCV
Finish	AutoFlow	PSV	AutoFlow	PSV	PSV	AutoFlow	PCV
Tidal volume (mL)							
Beginning	480	400	380	340	620	440	420
Hour 1	480	420	380	350	540		410
Hour 2	480	405	380	360	500		380
Hour 3	480	410		370	550	440	420
Finish	480	400	380	380	640	440	420
Respiratory rate (mpm)							
Beginning	26	22	26	28	25	28	35
Hour 1	26	22	26	26	24		35
Hour 2	26	22	26	26	24		35
Hour 3	26	22		25	24	28	35
Finish	26	22	26	28	22	28	35
Pmax (cmH₂O)							
Beginning	26	20	33	14	18	31	34
Hour 1	26	20	33	14	18		34
Hour 2	27	20	32	14	18		34
Hour 3	26	20		14	18	26	34
Finish	27	20	33	14	18	30	34
PEEP (cmH₂O)							
Beginning	10	8	15	8	12	10	8
Hour 1	10	8	15	8	12		8

(continues)

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Table 1. Clinical characteristics of the patients (continued)

	Patients						
	1	2	3	4	5	6	7
Hour 2	10	8	15	8	12		8
Hour 3	10	8		8	12	10	8
Finish	10	8	15	8	12	10	8
FiO ₂ (%)							
Beginning	70%	40%	70%	50%	65%	60%	80%
Hour 1	70%	40%	70%	53%	68%		85%
Hour 2	70%	40%	72%	52%	68%		86%
Hour 3	70%	40%		52%	67%	48%	83%
Finish	70%	40%	100%	50%	65%	50%	80%
Oxygen flow (L/min)							
Beginning							
Hour 1	16	8	16	12	20	15	18
Hour 2	16	8	16	12	20		16
Hour 3	16	8		12	20	15	15
Finish							
Pao ₂ (mmHg)							
Beginning	96.5	84.0	74.4	79.2	61.9	93.0	62.4
Hour 1	89.4	87.9	79.0	75.2	77.0		58.1
Hour 2	91.7	80.4	69.7	85.8	79.7		59.6
Hour 3	85.8	94.1		78.1	85.2	84.3	53.3
Finish	102.6	75.5	61.0	71.5	79.6	100.5	56.7
Paco ₂ (mmHg)							
Beginning	58.1	44.3	58.4	65.4	35.5	52.3	65.8
Hour 1	55.5	37.5	62.9	64.4	34.5		63.7
Hour 2	46.5	43.9	72.5	67.6	36.0		57.0
Hour 3	50.4	44.3		70.6	37.1	59.2	59.9
Finish	57.0	37.3	64.4	65.3	38.6	53.2	45.1
pH							
Beginning	7.40	7.34	7.37	7.32	7.43	7.34	7.40
Hour 1	7.42	7.35	7.32	7.34	7.44		7.43
Hour 2	7.44	7.34	7.31	7.31	7.42		7.43
Hour 3	7.42	7.34		7.32	7.43	7.29	7.44
Finish	7.40	7.39	7.33	7.34	7.42	7.27	7.47
Bicarbonate (mmol/L)							
Beginning	32.5	22.9	32.9	32.9	23.4	27.6	40.5
Hour 1	33.4	20.6	31.9	34.2	23.0		41.5
Hour 2	29.9	22.5	35.5	33.1	23.1		37.9
Hour 3	30.3	23.1		35.5	23.8	27.8	40.1
Finish	32.5	22.1	33.4	34.9	23.4	23.9	39.1

FiO₂ = inspired fraction of oxygen; mpm = movements per minute; NMBA = neuromuscular blocking agent; Paco₂ = arterial partial pressure of carbon dioxide; Pao₂ = arterial partial pressure of oxygen; PEEP = positive end-expiratory pressure; Pmax = maximum airway pressure; PSV = pressure support ventilation; VCV = volume controlled ventilation. * EVITA 4 (Soma Tech), Servo-i (Getinge), VEMO 150 (EOVE), VIVO 55 (Breas). † AutoFlow (Dräger).

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during the intervention and only one patient developed worsening of the Paco_2 level in the setting of ventilation unrelated to the intervention but related to high fever and clinical deterioration.

This proof-of-concept study has several limitations. First, this was a small case series that included no controls. Second, it is unclear if a longer period of ventilation would result in similar findings, although the findings for the first 3 hours are encouraging. Third, ventilation was provided by two specific ventilators; whether a different ventilator would have been associated with different outcomes cannot be determined.

Conclusion

In this uncontrolled case series of seven critically ill patients with COVID-19 and under mechanical ventilation, the use of ventilators designed for support of patients with sleep apnoea and/or chronic respiratory insufficiency was safe and did not result in worsening of hypoxaemia or hypercapnia. The limited sample size and study design preclude a definitive statement about the potential effectiveness of this treatment.

Competing interests

None declared.

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