Oxygenoterapie proč méně je někdy více

Kristýna Holomková, KNL, TUL, 2025





Speciální okolnosti

- otrava CO, pneumothorax
- zástava
- preoxygenace před zajištěním DC
- tonutí nedostatečná evidence (ERC)
- sepse nedostatek dat k vydání doporučení (SSC)
- těhotentsví klíčová je optimalizace oxygenace (ERC)

Box 2 | Examples of conditions that might benefit from higher or lower oxygen saturation thresholds

Lower target (such as SpO₂ 88-92%)

- Patients at risk of hypercapnic respiratory failure, for example:
 - Chronic obstructive pulmonary disease
 - Obesity hypoventilation
 - Neuromuscular respiratory diseases
 - Obstructive sleep apnoea
 - Decreased central respiratory drive (such as sedative overdose, stroke, encephalitis)

Higher target (such as SpO₂ approaching 100%)

- Carbon monoxide poisoning
- Cluster headaches
- Sickle cell crisis
- Pneumothorax

Co je to ta optimalizace oxygenace?

ESC European Societ of Cardiology

European Heart Journal (2023) 44, 3720–3826 https://doi.org/10.1093/eurheartj/ehad191 **ESC GUIDELINES**

Oxygen therapy for acutely ill medical patients: a clinical practice guideline

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WHAT YOU NEED TO KNOW

RAPID RECOMMENDATIONS

- It is a longstanding cultural norm to provide supplemental oxygen to sick patients regardless of their blood oxygen saturation
- A recent systematic review and meta-analysis has shown that too much supplemental oxygen increases mortality for medical patients in hospital
- For patients receiving oxygen therapy, aim for peripheral capillary oxygen saturation (SpO₂) of ≤96% (strong recommendation)
- For patients with acute myocardial infarction or stroke, do not initiate oxygen therapy in patients with SpO₂ ≥90% (for ≥93% strong recommendation, for 90-92% weak recommendation)
- A target SpO₂ range of 90-94% seems reasonable for most patients and 88-92% for patients at risk of hypercapnic respiratory failure; use the minimum amount of oxygen necessary

2023 ESC Guidelines for the management of acute coronary syndromes

4.2.2.1. Oxygen

Oxygen supplementation is recommended in ACS patients with hypoxaemia (oxygen saturations <90%). Oxygen supplementation in patients who are not hypoxic (oxygen saturations >90%) is not associated with clinical benefits and is therefore not recommended. 148,149

HypoxiaICOxygen is recommended in patients with hypoxaemia (SaO $_2$ <90%).</td>ICRoutine oxygen is not recommended in patients without hypoxaemia (SaO $_2$ >90%). 148,172IIIA

Co je to ta optimalizace oxygenace?



Oxygen therapy

This is a key component of hospital treatment of an exacerbation. Supplemental oxygen should be titrated to improve the patient's hypoxemia with a target saturation of 88-92%. (1389) Once oxygen is started, blood gases should be checked frequently, or as clinically indicated, to ensure satisfactory oxygenation without carbon dioxide retention and/or



Controlled oxygen therapy (if available)

Oxygen therapy should be titrated against pulse oximetry (if available) to maintain oxygen saturation at 93–95% (94–98% for children 6–11 years); note the potential for overestimation of oxygen saturation in people with dark skin color. In hospitalized asthma patients, controlled or titrated oxygen therapy is associated with lower mortality and better outcomes than high concentration (100%) oxygen therapy (Evidence A). 734-737 Oxygen should not be withheld if oximetry is not available, but the patient should be monitored for deterioration, somnolence or fatigue because of the risk of hypercapnia and respiratory failure. 734-737 If supplemental oxygen is administered, oxygen saturation should be maintained no higher than 96% in adults. 738

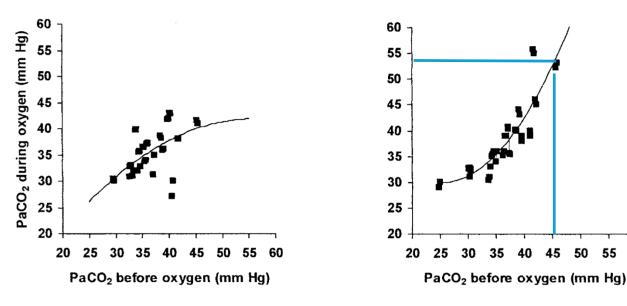


FIGURE 1. PaCo₂ during oxygen administration as a function of PaCo₂ before oxygen treatment. The variables correlated significantly in both groups (p < 0.01). Patients breathing 28% oxygen had a PaCo₂ fall (*left panel*); on the contrary, patients who received 100% oxygen showed an increase in PaCo₂, particularly those with PaCo₂ before oxygen treatment > 40 mm Hg (*right panel*).

BMJ

RESEARCH

Effect of high flow oxygen on mortality in chronic obstructive pulmonary disease patients in prehospital setting: randomised controlled trial

Michael A Austin, honorary associate, emergency medicine registrar, wilderness helicopter, intensive care paramedic, Karen E Wills, biostatistician, Leigh Blizzard, senior biostatistician, Eugene H Walters, professorial fellow, Richard Wood-Baker, honorary fellow, director

Interventions

Patients in the active arm received titrated oxygen treatment delivered by nasal prongs to achieve arterial oxygen saturations between 88% and 92%, with concurrent bronchodilator treatment administered by a nebuliser driven by compressed air (Walkie nebulisation air compressors, FlaemNova, Milan, Italy) and delivered via a facemask over the nasal prongs. The control arm received high flow oxygen treatment (8-10 l/min) administered by a non-rebreather face mask and bronchodilators delivered by nebulisation with oxygen at flows of 6-8 l/min. Pulse oximeters

were used to measure overen saturations and titrate

Table 2 | Baseline characteristics for all patients and subgroup with confirmed diagnosis of chronic obstructive pulmonary disease (COPD). Values are mean (SD) unless stated otherwise

Characteristic	Control (high flow oxygen)	Active (titrated oxygen)
All patients (n=405)		
No (%) male	114/226 (50)	83/179 (46)
Age (years)	69 (10.9) (n=202)	69 (11.8) (n=152)
Prehospital treatment time (minutes)	47 (19) (n=156)	47 (18) (n=144)
Pretreatment oxygen saturation (%)	86 (13.6) (n=189)	88 (9.8) (n=160)
Confirmed diagnosis of COPD (n=214)		
No (%) male	57/117 (49)	45/97 (46)
Age (years)	68.0 (10.2) (n=117)	67.9 (10.3) (n=97)
Per cent predicted FEV ₁	42.1 (16.4) (n=117)	43.3 (16.5) (n=97)
Smoking history (pack years)	45.5 (26.0) (n=87)	46.3 (22.1) (n=83)
Prehospital treatment time (minutes)	47 (17) (n=87)	50 (19) (n=80)
Pretreatment oxygen saturation (%)	84 (14) (n=101)	87 (10) (n=87)
FEV ₁ =forced expiratory volume in one se	cond.	

Austin MA, Wills KE, Blizzard L, Walters EH, Wood-Baker R. Effect of high flow oxygen on mortality in chronic obstructive pulmonary disease patients in prehospital setting: randomised controlled trial. BMJ. 2010 Oct 18;341:c5462. doi: 10.1136/bmj.c5462. PMID: 20959284; PMCID: PMC2957540.

WHAT IS ALREADY KNOWN ON THIS TOPIC

Audits have shown increased mortality, acidosis, and hypercarbia in patients with acute exacerbations of chronic obstructive pulmonary disease treated with high flow oxygen

High flow oxygen is still used routinely in prehospital and hospital areas for breathless patients with chronic obstructive pulmonary disease

A "more is better" oxygen culture is strong in prehospital management

WHAT THIS STUDY ADDS

Titrated oxygen treatment reduces mortality, acidosis, and hypercarbia in patients with acute exacerbation of chronic obstructive pulmonary disease treated before arrival at hospital

The risk of death was reduced by 78% by use of titrated oxygen rather than high flow oxygen, with a number needed to harm of 14

These findings provide strong evidence that titrated oxygen treatment should be used for hypoxic or breathless patients with chronic obstructive pulmonary disease in prehospital settings

Results In an intention to treat analysis, the risk of death was significantly lower in the titrated oxygen arm compared with the high flow oxygen arm for all patients (high flow oxygen n=226; titrated oxygen n=179) and for the subgroup of patients with confirmed chronic obstructive pulmonary disease (high flow n=117; titrated n=97). Overall mortality was 9% (21 deaths) in the high flow oxygen arm compared with 4% (7 deaths) in the titrated oxygen arm; mortality in the subgroup with confirmed chronic obstructive pulmonary disease was 9% (11 deaths) in the high flow arm compared with 2% (2) deaths) in the titrated oxygen arm. Titrated oxygen treatment reduced mortality compared with high flow oxygen by 58% for all patients (relative risk 0.42, 95% confidence interval 0.20 to 0.89; P=0.02) and by 78% for the patients with confirmed chronic obstructive pulmonary disease (0.22, 0.05 to 0.91; P=0.04). Patients with chronic obstructive pulmonary disease who received titrated oxygen according to the protocol were significantly less likely to have respiratory acidosis (mean difference in pH 0.12 (SE 0.05); P=0.01; n=28) or hypercapnia (mean difference in arterial carbon dioxide

The risk of death was reduced by 78% by use of titrated oxygen rather than high flow oxygen, with a number needed to harm of 14

Table 3 Intention to treat analysis. Values are numbers (percentages) unless stated otherwise

	Control (h	nigh flow oxygen)	Active (titra	ated oxygen)	Treatment	effect	P value		
Mortality									
All patients		Rando	misation			.89)*	0.02		
Confirmed COPD	Ţ.	Paramedics					0.04		
ncidence of ventilation	Control (high f	Control (high flow) (n=30) Active (titrated) (n=32)							
All patients	↓		breathlessnes story of COPD	S		.72)*	0.70		
Non-invasive ventilation	Control (high fl	ow) (n=226)		Active (titrated) (n=179)					
Invasive ventilation	Patients	excluded (n=109):	1	Patients exclude					
Confirmed COPD		function data (n=103) /C>0.7 (n=6)		No lung function da FEV ₁ /FVC>0.7 (n=7)		.54)*	0.34		
Non-invasive ventilation		Subgroup with confirmed diagnosis of COPD Patients included in ITT analysis (n=117) Patients included in ITT analysis (n=97)							
Invasive ventilation	Datients included in I								
Length of hospital stay (mea	Patients included in t	i i dildiysis (ii=117)	Patients	Included III II I allal	ysis (II=97)				
All patients		received non-protocol ment (n=25; 21%)		Patients receive treatment (r	ed non-protocol n=54: 56%))†	0.19		
Confirmed COPD	↓)†	0.37		
Arterial blood gases (<30 min	Patients included in 1	PP analysis (n=92)	Patients	included in TPP ana	lysis (n=43)				
Mean (SD) pH	Flow of participants throu	igh study COPD=chr	onic obstructi	ve nulmonary dise	ase: FFV.=forced	- 	0.11		
Mean (SD) carbon dioxide (m	Flow of participants through study. COPD=chronic obstructive pulmonary disease; FEV ₁ =force expiratory volume in one second; FVC=forced vital capacity; ITT=intention to treat;						0.06		
Mean (SD) bicarbonate (mmc	TPP=treatment per protoc	col				†	0.07		
Mean (SD) oxygen (mm Hg) (ar	terial only) 98.4	(46.1) (n=14)	79.3 (24	i.9) (n=9)	-19.1 (16	5.8)†	0.34		

Table 4 Treatm	ent per protoco	l. Values are numbers	(percentages) unless stated	otherwise
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	Control (high flow oxygen)	Active (titrated oxygen)	Treatment effect	P value
Mortality				
All patients	16/177 (9)	3/66 (5)	0.50 (0.16 to 1.54)*	0.23
Confirmed COPD	9/92 (10)	1/43 (2)	0.24 (0.04 to 1.57)*	0.14
Incidence of ventilation				
All patients	19/167 (11)	5/63 (8)	0.70 (0.25 to 1.97)*	0.50
Non-invasive ventilation	7	4		
Invasive ventilation	12	1		
Confirmed COPD	15/83 (18)	3/40 (8)	0.42 (0.14 to 1.20)*	0.11
Non-invasive ventilation	6	2		
Invasive ventilation	9	1		
Length of hospital stay (mean (SD) days)				
All patients	5.9 (5.5) (n=177)	6.0 (5.3) (n=66)	0.09 (0.78)†	0.87
Confirmed COPD	6.5 (6.0) (n=92)	6.2 (4.6) (n=43)	-0.29 (1.04)†	0.96
Arterial blood gases (<30 min) (confirmed C	OPD patients)			
Mean (SD) pH	7.29 (0.15) (n=18)	7.41 (0.09) (n=10)	0.12 (0.05)†	0.01
Mean (SD) carbon dioxide (mm Hg)	76.5 (50.2) (n=19) = 9,5	kPa 42.9 (14.2) (n=10) = 5,3	3 kPa -33.6 (16.3)†	0.02
Mean (SD) bicarbonate (mmol/l)	31.5 (9.9) (n=18)	26.0 (4.2) (n=10)	-5.5 (3.30)†	0.15
Mean (SD) oxygen (mm Hg) (arterial only)	98.4 (46.1) (n=14)	81.5 (30.9) (n=6)	-16.9 (20.7)†	0.46

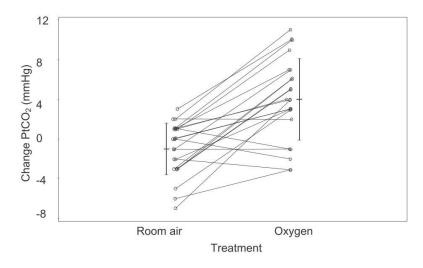


FIGURE 2. The change in $PtCO_2$ (mm Hg) from baseline following breathing 100% oxygen or room air. The vertical lines are the mean (central horizontal line) \pm 1 SD for 20 min $PtCO_2$ minus baseline. See Figure 1 legend for expansion of abbreviation.

CONCLUSION

Among people with mild, stable untreated OHS, breathing moderate concentrations of supplemental oxygen increased PavCO₂, sufficient to induce acidaemia during F_iO₂ 0.50. These findings highlight the need for caution during supplemental oxygen administration among people with OHS and support current clinical guidelines which recommend targeting an SpO₂ range and monitoring of ABGs during supplemental oxygen administration.

Hollier CA, Harmer AR, Maxwell LJ, Menadue C, Willson GN, Unger G, Flunt D, Black DA, Piper AJ. Moderate concentrations of supplemental oxygen worsen hypercapnia in obesity hypoventilation syndrome: a randomised crossover study. Thorax. 2014 Apr;69(4):346-53. doi: 10.1136/thoraxjnl-2013-204389. Epub 2013 Nov 19. PMID: 24253834.

Results: The study was terminated in three subjects breathing 100% oxygen due to a Ptco(2) increase ≥ 10 mm Hg, which occurred after 10:35, 13:20, and 15:51 min. Ptco(2) increased by 5.0 mm Hg (95% CI, 3.1-6.8; P < .001) with oxygen compared with room air. Minute ventilation decreased by 1.4 L/min (95% CI, 0.11-2.6 L/min; P = .03), and volume of dead space to tidal volume ratio increased by 0.067 (95% CI, 0.035-0.10; P < .001) with oxygen compared with room air.

Wijesinghe, M., Williams, M., Perrin, K., Weatherall, M., & Beasley, R. (2011). *The Effect of Supplemental Oxygen on Hypercapnia in Subjects With Obesity-Associated Hypoventilation. Chest, 139(5), 1018–1024*.doi:10.1378/chest.10-1280

Jak tedy poznám, komu to může uškodit?

- Anamnéza CHOPN
- Kuřáci 10 pack years/45 pack yrs



Box 2 | Examples of conditions that might benefit from higher or lower oxygen saturation thresholds

Lower target (such as SpO₂ 88-92%)

- Patients at risk of hypercapnic respiratory failure, for example:
 - Chronic obstructive pulmonary disease
 - Obesity hypoventilation
 - Neuromuscular respiratory diseases
 - Obstructive sleep apnoea
 - Decreased central respiratory drive (such as sedative overdose, stroke, encephalitis)

Higher target (such as SpO₂ approaching 100%)

- Carbon monoxide poisoning
- Cluster headaches
- Sickle cell crisis
- Pneumothorax

Jak tedy poznám, komu to může uškodit?

TABLE 1

Clinical features of patients with obesity hypoventilation syndrome based on an aggregated sample of 757 patients from 15 studies

Clinical features	Mean (range)
Age years	52 (42–61)
Male %	60 (49–90)
Body mass index kg⋅m ⁻²	44 (35–56)
Neck circumference cm	46.5 (45–47)
рН	7.38 (7.34–7.40)
Arterial P _{CO2} mmHg	53 (47–61) = 7 kPa
Arterial P _{O2} mmHg	56 (46–74) = 7,5 kPa
Serum bicarbonate mEq⋅L ⁻¹	32 (31–33)
Haemoglobin g⋅dL ⁻¹	15
Apnoea-hypopnoea index	66 (20–100)
\mathbf{S}_{pO_2} nadir during sleep %	65 (59–76)
Per cent sleep time S _{pO2} <90%	50 (46–56)

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BMI over 35 kg/m², the prevalence of OHS was 31%.

Jak tedy poznám, komu to může uškodit?

 Hypoventilace – bolest hrudníku, neurodegenerativní onemocnění, paréza bránice...

Bradypnoe

Porucha vědomí

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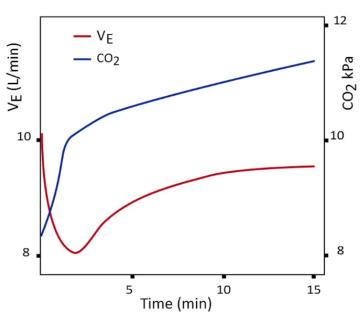
Higher target (such as SpO₂ approaching 100%)

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Jaké mechnismy na se tom podílejí?

Hyperoxemie nebo "normoxémie" Ztráta hypoxické Snížení Haldane plicní respiračního drive efekt vasokostrikce Snížení Perfuze špatně Snížení transportní kapacity ventilovaných minutové ventilace krve pro CO2 a H+ alveolů Intrapulmonální Hemodynamické Absorpční důsledky atelektázy zkraty

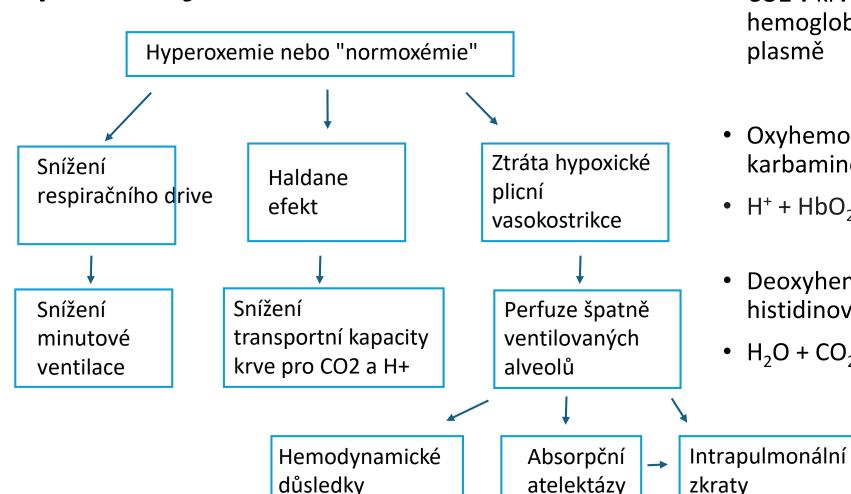
Vliv snížení respiračního drive



Effect of minute ventilation during oxygen-induced hypercapnia. During 15 minutes of high oxygen administration, an initial decrease in minute ventilation, which recovers substantially, is seen in patients with acute exacerbation of chronic obstructive pulmonary disease. However, the oxygen-induced hypercapnia does not recover. CO₂, carbon dioxide; V_E, minute ventilation. Based on data of Aubier and colleagues [4].

Abdo WF, Heunks LM. Oxygen-induced hypercapnia in COPD: myths and facts. Crit Care. 2012 Oct 29;16(5):323. doi: 10.1186/cc11475. PMID: 23106947; PMCID: PMC3682248

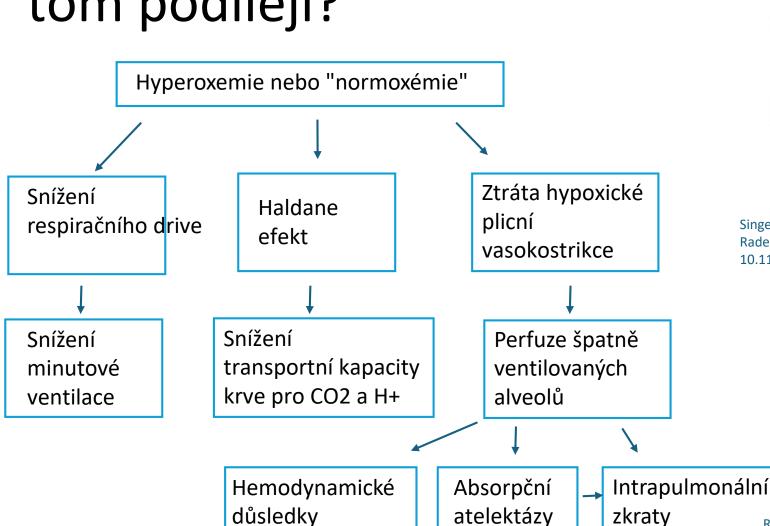
Jaké mechanismy na se tom podílejí?



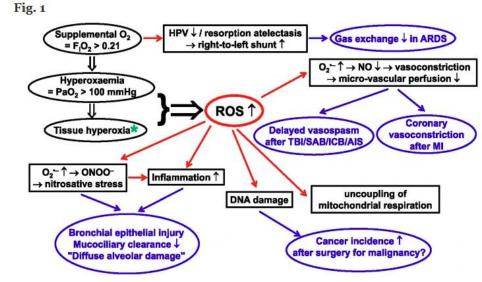
(Bohr-)Haldane efekt

- CO2 v krvi většina jako bikabonát, část na hemoglobinu, jen zlomek disolvovaný v plasmě
- Oxyhemoglobin má menší kapacitu jako karbaminohemoglobin
- $H^+ + HbO_2 \rightleftharpoons H^+Hb + O_2$
- Deoxyhemoglobin je efektivnější pufr (volná histidinová residua)
- $H_2O + CO_2 \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$

Jaké mechanismy na se tom podílejí?

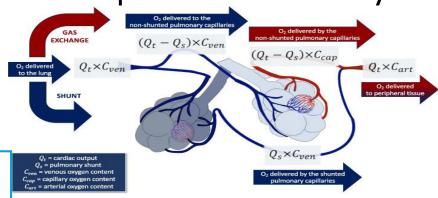


Ztráta hypoxické plicní vasokontrikce



Singer M, Young PJ, Laffey JG, Asfar P, Taccone FS, Skrifvars MB, Meyhoff CS, Radermacher P. Dangers of hyperoxia. Crit Care. 2021 Dec 19;25(1):440. doi: 10.1186/s13054-021-03815-y. PMID: 34924022; PMCID: PMC8686263.

Intrapulmonální zkraty



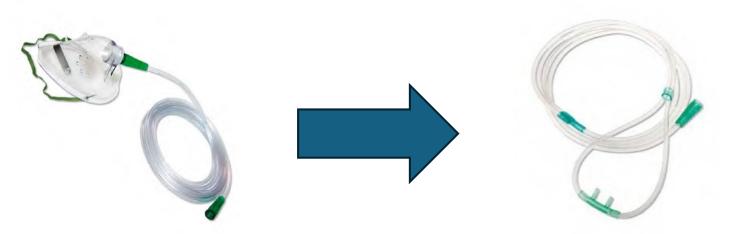
Rai mondi Cominesi D, Forcione M, Pozzi M, Giani M, Foti G, Rezoagli E, Cipulli F. Pulmonary shunt in critical care: a practical approach with clinical scenarios. J Anesth Analg Crit Care. 2024 Mar 6;4(1):18. doi: 10.1186/s44158-024-00147-5. PMID: 38449055; PMCID: PMC10916277.

Forma podávání kyslíku má taky vliv

Parameters during test periods.

Oxygen flow rate	BP mmHg	HR beats/min	f breaths/min	$rac{\mathbf{V}_{\mathrm{T}}}{\mathbf{I}}$	Ů l/min	Pao ₂ kPa	$ \begin{array}{c} \operatorname{Paco}_{2} \\ \operatorname{kPa} \end{array} $	BE mmol/l
5 l/min	112 ± 7	63 ± 10	12.5 ± 1.4	$0.38 \pm 0.09***$	4.80 ± 1.16***	38.4 ± 5.0***	4.8 ± 0.3	-1.1 ± 1.7
4 l/min	114 ± 8	61 ± 10	12.5 ± 2.8	$0.42 \pm 0.09 **$	$5.23 \pm 1.17***$	$36.9 \pm 5.8***$	4.7 ± 0.5	-1.3 ± 2.1
3 1/min	115 ± 7	64 ± 11	13.2 ± 2.6	$0.48 \pm 0.11*$	$6.30 \pm 1.46**$	32.0 ± 5.5 ***	4.8 ± 0.4	-0.6 ± 1.6
2 1/min	114 ± 7	61 ± 8	14.1 ± 2.8	$0.46 \pm 0.11*$	$6.30 \pm 1.39**$	27.6 ± 3.6***	5.0 ± 0.5	-0.3 ± 2.0
1 l/min	115 <u>+</u> 8	61 ± 10	13.2 ± 2.4	0.53 ± 0.15	7.00 ± 1.98	$23.5 \pm 2.9***$	4.7 ± 0.4	-1.8 ± 1.7
0 l/min	115 ± 9	64 ± 11	14.1 ± 3.1	0.54 ± 0.10	7.52 ± 1.89	13.3 ± 0.7	4.8 ± 0.3	-1.0 ± 1.2

All data are expressed as mean \pm s.d. Abbreviations as in Table 2. *=P<0.05, **=P<0.01 and ***=P<0.001 are used to indicate significantly different values compared to values obtained with an oxygen flow rate of 0 l/min.



The effect of oxygen flow rates on ventilation. Acta Anaesthesiol Scand. 1991 May;35(4):289-92. doi: 10.1111/j.1399-6576.1991.tb03291.x. PMID: 1906671.

Physiological paramete r	3	2	1 1	Score 0	1	2	3
Respiration rate (per minute)	≤8		9–11	12–20		21–24	≥25
SpO ₂ Scale 1 (%)	≤91	92–93	94-95	≥96			
SpO ₂ Scale 2 (%)	≤83	84–85	86–87	88–92 ≥93 on air	93–94 on oxygen	95–96 on oxygen	≥97 on oxygen
Air or oxygen?		Oxygen		Air			
Systolic blood pressure (mmHg)	≤90	91–100	101–110	111–219			≥220
Pulse (per minute)	≤40		41–50	51–90	91–110	111–130	≥131
Consciousness				Alert			CVPU
Temperature (°C)	≤35.0		35.1–36.0	36.1–38.0	38.1–39.0	≥39.1	