



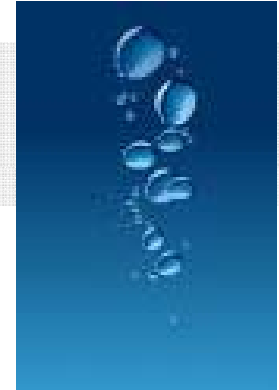
**Sauerstoff im Notfall
Ist weniger mehr?**

Sauerstoffgabe

Worum geht es?

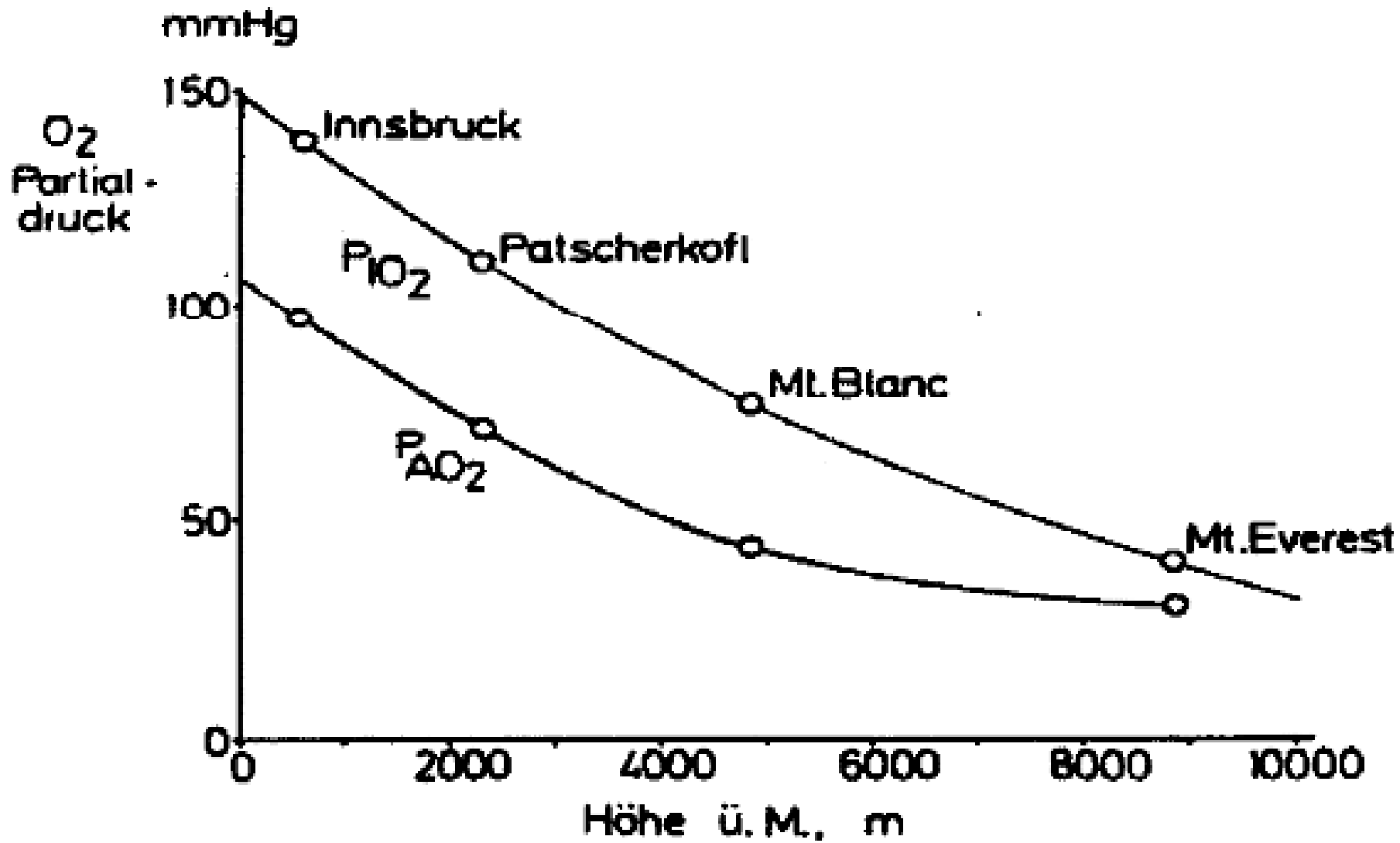
- Physiologische Grundlagen
 - Sauerstoffangebot
 - Sauerstofftransportkapazität
 - Sauerstoffversorgung
- Indikationen
- Grenzen / Kontraindikationen
- Wie soll ich es machen ?

Sauerstoff

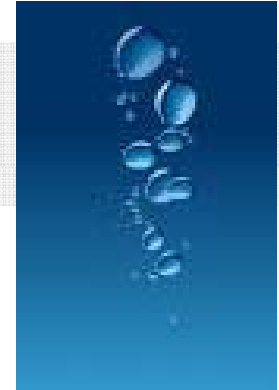


Luftdruck und Sauerstoffpartialdruck Auf Meereshöhe

- ca. 760 mmHg (100% Luft)
- ca. 150 mmHg (21% O₂)
- ca. 105 mmHg im arteriellen Blut
(Wasserdampfdruck, BTS, Shunt)



Sauerstoff



Sauerstoffgehalt des Blutes

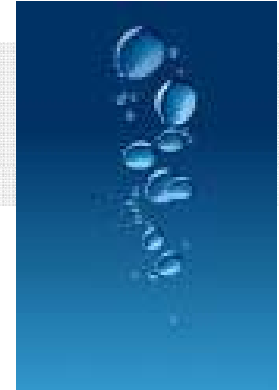
$$CaO_2 = (1,34 \times Hb \times SaO_2) + (0,003 \times paO_2)$$

Hb 15 g/dl – SaO₂ 98% - paO₂ 100 mmHg

maximal:

$$\begin{aligned} CaO_2 &= (1,34 \times 15 \times 0,98) + 0,003 \times 100 \\ &= \mathbf{19,998 \text{ ml}} \end{aligned}$$

Sauerstoff



Sauerstoffangebot

DO₂ [ml min⁻¹ m⁻²]

$$= 1,34 \times \text{Hb [g\%]} \times \text{SaO}_2 [\%] \times \text{CI [ml min}^{-1} \text{ m}^{-2}]$$

ERC-Leitlinien

..... Patienten benötigen keinen zusätzlichen Sauerstoff, solange sie nicht hypoxämisch sind. Einige der vorliegenden Daten deuten daraufhin, dass eine Sauerstoffgabe in hoher Konzentration für Patienten mit unkompliziertem Myokardinfarkt schädlich sein könnte [33, 34, 35]. **Der Zielwert der S_aO_2 soll 94–98% sein** bzw. 88–92%, wenn das Risiko der Atemdepression mit CO_2 -Retention besteht [36].

[33] Rawles JM, Kenmure AC

Controlled trial of oxygen in uncomplicated myocardial infarction. Br Med Bull **1976**;1:1121–1123

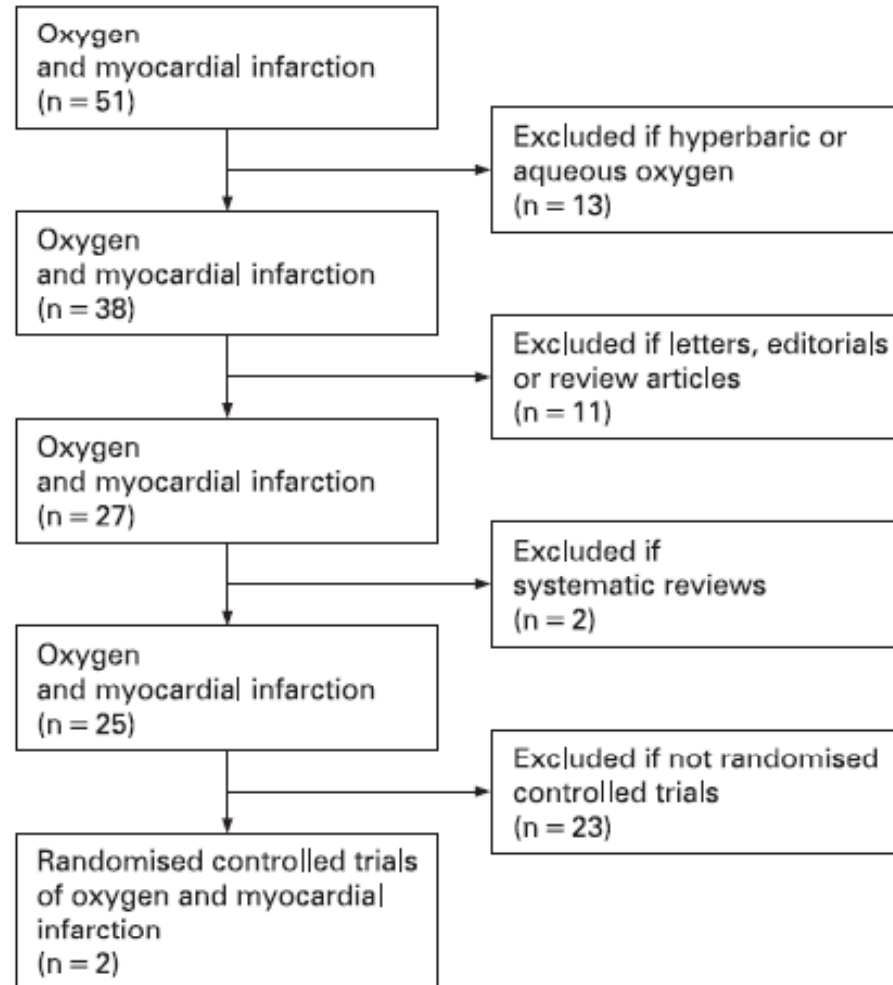
- 181 Patienten
- Myokardinfarkt
- randomisiert, doppelblind O₂ oder Luft für 24 h
- kein Unterschied in der Mortalität
- höhere Inzidenz einer Sinustachykardie in der O₂-Gruppe
- PaO₂ signifikant höher in der O₂-Gruppe
- *ASAT (GOT) signifikant höher in der O₂-Gruppe*

Fazit:

Keine Evidenz, dass eine generelle Gabe von O₂ beim unkomplizierten Myokardinfarkt einen Benefit hat.

[34] Wijesinghe M, Perrin K, Ranchord A et al.

Routine use of oxygen in the treatment of myocardial infarction: systematic review. *Heart* **2009**;95:198–202



[34] Wijesinghe M, Perrin K, Ranchord A et al.

Routine use of oxygen in the treatment of myocardial infarction: systematic review. Heart **2009**;95:198–202

Ergebnis:

- 2 Studien auswertbar
(randomisierte, kontrollierte Studien bezüglich O₂-Therapie beim unkomplizierten Infarkt)
- 1 Studie = Rawles JM, Kenmure AC. Br Med Bulletin 1976
- 1 Studie = keine verwendbaren Daten bzgl. Mortalität

Fazit:

The limited evidence that does exist suggests that the routine use of high-flow oxygen in uncomplicated MI may result in a greater infarct size and possibly increase the risk of mortality.

[35] Cabello JB, Burls A, Emparanza JI et al.

Oxygen therapy for acute myocardial infarction.
Cochrane Database Syst Rev **2010**;6:CD007160.

Objectives:

To review the evidence from randomised controlled trials to establish whether routine use of inhaled oxygen in acute myocardial infarction (AMI) improves patient-centred outcomes, in particular pain and death.

Selection Criteria:

Randomised controlled trials of people with suspected or proven AMI, less than 24 hours after onset, in which the intervention was inhaled oxygen (at normal pressure) compared to air and regardless of co-therapies provided these were the same in both arms of the trial.

[35] Cabello JB, Burls A, Emparanza JI et al.

Oxygen therapy for acute myocardial infarction.
Cochrane Database Syst Rev **2010**;6:CD007160.

Main Results:

3 trials involving 387 patients were included and 14 deaths occurred. The pooled RR of death was 2.88 (95% CI 0.88 to 9.39) in an intention-to-treat analysis and 3.03 (95% CI 0.93 to 9.83) in patients with confirmed AMI.

While suggestive of harm, the small number of deaths recorded meant that this could be a chance occurrence. Pain was measured by analgesic use. The pooled RR for the use of analgesics was 0.97 (95% CI 0.78 to 1.20).

Authors Conclusions: No conclusive evidence to support the routine use of inhaled oxygen in patients with acute AMI.

Farquhar H et al.

Systematic review of studies of the effect of hyperoxia on coronary blood flow. Am Heart J 2009 Sep;158(3):371-7.

Methods

Search of Medline, Cochrane Database of Systematic Reviews, EMBASE, and CINHAL. The primary outcome measure was coronary blood flow; secondary outcomes included coronary vascular resistance and myocardial oxygen consumption.

Results

6 studies from 4 publications with 6 healthy subjects and 61 subjects with cardiac disease. High-concentration O₂ resulted in Hyperoxia (PaO₂ 273-425 mm Hg) and caused

- **significant reduction in coronary blood flow** (-7.9% to -28.9%)
- **significant increase in coronary vascular resistance** (21.5- 40.9%, n = 4 studies)
- **significant reduction in myocardial oxygen consumption**
(mean change -15.3% to -26.9%, n = 3 studies)

Conclusions

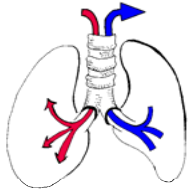
Hyperoxia causes a marked reduction in coronary blood flow and myocardial oxygen consumption. These physiologic effects may have the potential to cause harm and are relevant to the use of high-concentration oxygen therapy in the treatment of cardiac and other disorders.

ERC-Leitlinien

..... Patienten benötigen keinen zusätzlichen Sauerstoff, solange sie nicht hypoxämisch sind. Einige der vorliegenden Daten deuten daraufhin, dass eine Sauerstoffgabe in hoher Konzentration für Patienten mit unkompliziertem Myokardinfarkt schädlich sein könnte [33, 34, 35]. Der Zielwert der S_aO_2 soll 94–98% sein bzw. 88–92%, wenn das Risiko der Atemdepression mit CO_2 -Retention besteht [36].

One of the most pervasive myths surrounding the treatment of acute on chronic respiratory failure is that these patients rely on hypoxic drive to breathe.

Schmidt GA et al. (1989) JAMA 261: 3444



Atemantrieb bei COPD-Patienten:

	FiO₂ ≤ 0,4	FiO₂ = 0,7
Atemzugvolumen (ml)	389 ± 102	383 ± 101
Atemfrequenz (1/min)	24,0 ± 6,2	24,4 ± 7,2
Atemminutenvolumen (l/min)	8,7 ± 1,5	9,0 ± 2,3
paCO₂ (mmHg)	56,4 ± 6,2	56,7 ± 7,8
paO₂ (mmHg)	85,1 ± 17,6	226,8 ± 67,5

Crossley D, McGuire G, Barrow P, Houston P. Crit Care Med 1997, 25(9) 1522-6

[36] O'Driscoll BR, Howard LS, Davidson AG

BTS guideline for emergency oxygen use in adult patients.
Thorax **2008**;63(Suppl6):vi1-Vi68

9.4 Patients with known COPD

A proportion of breathless patients will have COPD (chronic bronchitis and emphysema). Unfortunately, a recent Cochrane review of oxygen therapy for COPD in the prehospital setting found no relevant studies.²⁸²

Recommendation (see table 3)

- ▶ Patients with COPD should initially be given oxygen via a Venturi 28% mask at a flow rate of 4 l/min or a 24% Venturi mask at a flow rate of 2 l/min. Some patients may benefit from higher flow rates via the Venturi mask (see recommendation 32). The target oxygen saturation should be 88–92% in most cases or an individualised saturation range based on the patient's blood gas measurements during previous exacerbations. **[Grade C]**



Oxygen therapy in the pre-hospital setting for acute exacerbations of chronic obstructive pulmonary disease [tion](#)

Michael A. Austin^{1,2}, Richard Wood-Baker²

Editorial Group: [Cochrane Airways Group](#)

Published Online: 21 JAN 2009

Abstract

Jump to...

Background

Chronic obstructive pulmonary disease (COPD), a leading cause of morbidity and mortality in the developed world, is characterised by acute deterioration in symptoms. During these exacerbations, people are prone to developing alveolar hypoventilation, which may be contributed to by the administration of high inspired oxygen concentrations.

Objectives

The objective of the review was to determine the effect of different inspired oxygen concentrations ('high flow' compared to 'controlled') in the pre-hospital setting on outcome for people with acute exacerbations of COPD.

Search strategy

We searched the Cochrane Airways Group Specialised Register (CENTRAL), MEDLINE, EMBASE and CINAHL and reference lists of articles. We also contacted authors of identified RCTs for details of other relevant, published and unpublished studies. The most recent search was conducted in August 2008.

Selection criteria

Randomised controlled trials comparing oxygen therapy at different concentrations or oxygen therapy versus placebo in the pre-hospital setting for treatment of acute exacerbations of COPD were eligible.

Data collection and analysis

Two review authors independently assessed trial quality and extracted data.

Main results

The search identified a total of 741 abstracts, of which 18 were selected as potentially relevant, only two of the 18 studies were randomised controlled trials and eligible for inclusion in the review, but were ongoing and had no data available for analysis.

Authors' conclusions

No relevant trials have been published to date, so there is no evidence to indicate whether different oxygen therapies in the pre-hospital setting have an effect on outcome for people with acute exacerbations of COPD. There is an urgent need for robust, well-designed randomised controlled trials to investigate the effect of oxygen therapies in the pre-hospital setting for people with acute exacerbations of COPD.

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

Austin MA, Wills KE, Blizzard L, Walters EH, Wood-Baker R.

BMJ 2010 Oct 18;341

ABSTRACT

Objectives To compare standard high flow oxygen treatment with titrated oxygen treatment for patients with an acute exacerbation of chronic obstructive pulmonary disease in the prehospital setting.

Design Cluster randomised controlled parallel group trial.

Setting Ambulance service in Hobart, Tasmania, Australia.

Participants 405 patients with a presumed acute exacerbation of chronic obstructive pulmonary disease who were treated by paramedics, transported, and admitted to the Royal Hobart Hospital during the trial period; 214 had a diagnosis of chronic obstructive pulmonary disease confirmed by lung function tests in the previous five years.

Interventions High flow oxygen treatment compared with titrated oxygen treatment in the prehospital (ambulance/paramedic) setting.

Main outcome measure Prehospital or in-hospital mortality.

Conclusions Titrated oxygen treatment significantly reduced mortality, hypercapnia, and respiratory acidosis compared with high flow oxygen in acute exacerbations of chronic obstructive pulmonary disease. These results

provide strong evidence to recommend the routine use of titrated oxygen treatment in patients with breathlessness and a history or clinical likelihood of chronic obstructive pulmonary disease in the prehospital setting.

Trial registration Australian New Zealand Clinical Trials Register ACTRN12609000236291.

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

Austin MA, Wills KE, Blizzard L, Walters EH, Wood-Baker R.

BMJ 2010 Oct 18;341

Methodik:

- high-flow O₂ vs. titrierte O₂ Gabe in der Präklinik
- Prä- oder innerklinische Mortalität

Ergebnisse:

- 405 Pat. mit V. a. COPD; 114/226 high-flow, 83/179 titriert,
- 208 fälschlich als COPD Patienten eingestuft
- Mortalität gesamt 9% (n=21, high-flow) vs. 4% (n=7, titriert)
- Mortalität COPD 9% (n=11, high-flow) vs. 2% (n=2, titriert)
- COPD signifikant seltener Azidose, Hyperkapnie

Table 4 | Treatment per protocol. Values are numbers (percentages) unless stated otherwise

	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 COPD 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)	42.9 (14.2) (n=10)	-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

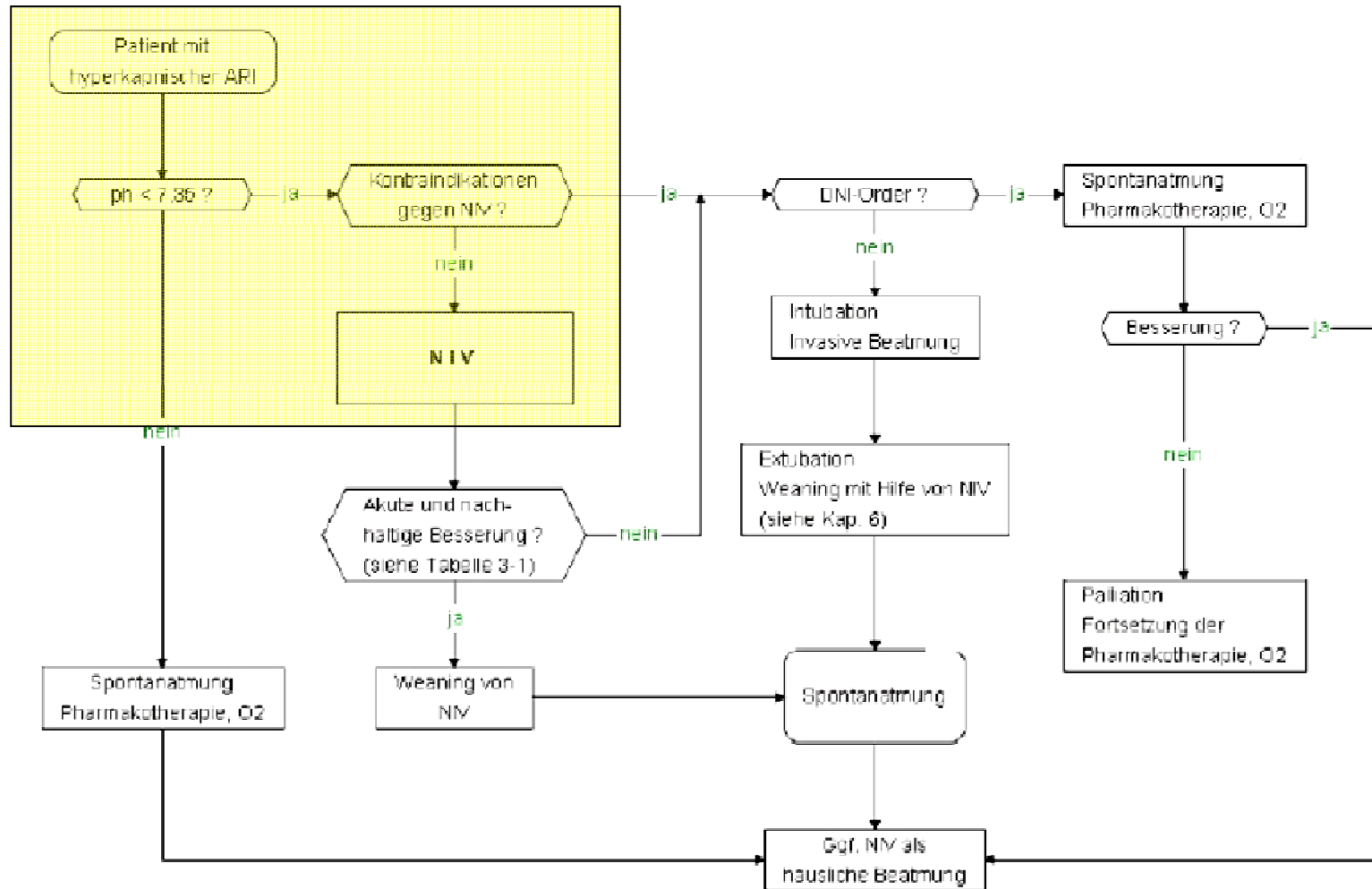
COPD=chronic obstructive pulmonary disease.

*Relative risk (95% CI).

†Mean difference (SE).

Hyperkapnische ARI

Indikation: $\text{pH} < 7.35$, $\text{paCO}_2 > 45 \text{ mmHg}$



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

Austin MA, Wills KE, Blizzard L,
Walters EH, Wood-Baker R.

BMJ 2010 Oct 18;341

Fazit ?

Titrierte O₂-Gabe reduziert bei AECOPD (nur 51% korrekt erkannt)

- signifikant die Mortalität um 58% für alle, 78% für COPD Pat.
- signifikant Azidose
- signifikant Hyperkapnie
- signifikant Azidose

Aussagen stützen sich auf die 29/243 Patienten bei denen eine BGA abgenommen wurde

> O₂ sollte bei Dyspnoe und V. a. COPD titriert verabreicht werden

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THORAX

October 2008

Thorax

AN INTERNATIONAL JOURNAL OF RESPIRATORY MEDICINE

Guideline for emergency
oxygen use in adult patients

British Thoracic Society
Emergency Oxygen Guideline Group

thorax.bmj.com



BMJ Journals

[36] O´Driscoll BR, Howard LS, Davidson AG

BTS guideline for emergency oxygen use in adult patients.
Thorax **2008**;63(Suppl6):vi1-Vi68

4 Kategorien der Sauerstoffgabe

- **Hochdosiert** = Ziel SpO₂ 94-98%
- **Moderat** = Ziel SpO₂ 94-98%
- **Kontrolliert** = SpO₂ 88-92%
- **Keine Gabe** = SpO₂ 94-98%

[36] O'Driscoll BR, Howard LS, Davidson AG

BTS guideline for emergency oxygen use in adult patients.
Thorax **2008**;63(Suppl6):vi1-Vi68

High

- **Reanimation** = max. O₂ bis normale Vitalparameter
- **Schweres Trauma** = 15 l/min
- **Anaphylaxie** = 15 l/min
- **Sepsis** = 15 l/min
- **Schock** = 15 l/min
- **Epileptischer Anfall** = 15 l/min während Anfall
- **Hypothermie** = 15 l/min

[36] O´Driscoll BR, Howard LS, Davidson AG

BTS guideline for emergency oxygen use in adult patients.
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Moderate

- **Akute Hypoxämie, SpO₂ <85% = 10-15 l/min**
- **Akute Hypoxämie, SpO₂ ≥85-93% = 5-10 l/min**

auch bei

- Lungenembolie ?!
- schwerer Anämie ?!

[36] O´Driscoll BR, Howard LS, Davidson AG

BTS guideline for emergency oxygen use in adult patients.
Thorax **2008**;63(Suppl6):vi1-Vi68

Controlled oder low-dose, Ziel 88-92%

- COPD

- schwere Adipositas (BMI >40 kg/m²)

CAVE: wenn SpO₂ <88% = 5-10 l/min

[36] O´Driscoll BR, Howard LS, Davidson AG

BTS guideline for emergency oxygen use in adult patients.
Thorax **2008**;63(Suppl6):vi1-Vi68

Kein O₂, wenn SpO₂ >94%

- Herzinfarkt
- HRST
- Schlaganfall
- ...
- GI-Blutung?

CAVE: wenn SpO₂ <85% = 15 l/min

wenn SpO₂ ≥85-93% = 5-10 l/min

Sauerstoff beim Neugeborenen

**Raumluft
wahrscheinlich
genau so effektiv
wie 100 % O₂**

Oxygen administration

Until recently, available guidelines recommended oxygen in increased concentration, as high as 100%, in the treatment of asphyxiated newborns. This attitude was strengthened by concerns about the risk of oxygen deprivation during and following asphyxia. Since then, the role of increased oxygen concentration in generating free oxygen radicals, oxidative stress, and reperfusion injury was more elucidated. Furthermore, experimental and clinical data have raised concerns about the potential adverse effects of 100% oxygen on respiratory physiology, cerebral circulation and potential tissue injury by oxygen free radicals. Also, randomised studies in asphyxiated newborns strongly suggest that air is as effective as 100% oxygen.

Sauerstoff beim Neugeborenen

Oxygen administration

At present, the standard approach to term newborns receiving resuscitation at birth with positive-pressure ventilation is to use room air instead of 100% oxygen. If, despite effective ventilation, there is no increase in heart rate, or oxygenation (measured by pulse oximetry) remains unacceptable, higher concentrations of oxygen should be used.

*Acceptable
pre-ductal SpO₂*

2 min: 60%

3 min: 70%

4 min: 80%

5 min: 85%

10 min: 90%

Daher Raumluft !

Es sei denn

Herzfrequenz steigt
nicht an,

SpO₂ unzureichend

Sauerstoff

Wieviel gibt der Rettungsdienst in Stadt und LK GÖ pro Jahr für O₂ aus?

A - 1000,- €

B - 5000,- €

D - 10000,- €

D - 20000,- €

Sauerstoff

Unser teuerstes Medikament

A - 1000,- €

B - 5000,- €

D - 10000,- €

D - 20000,- €

Sauerstoff

4 Gründe

- Präoxygenierung
 - Ziel Stickstoff auswaschen
- Hypoxämie (\neq Luftnot)
 - Ziel Vollsättigung des Hb
- Physikalische Löslichkeit
 - Ziel Anteil des nicht Hb gebunden O₂ erhöhen
- PVR reduzieren

Sauerstoff

Hochdosiert

- **Reanimation** = max. O₂ bis normale Vitalparameter
 - **Schweres Trauma** = 15 l/min
 - **Anaphylaxie** = 15 l/min
 - **Sepsis** = 15 l/min
 - **Schock** = 15 l/min
 - **Epileptischer Anfall** = 15 l/min während Anfall
 - **Hypothermie** = 15 l/min
- + *bei klinischem V. a. Anämie (GI-Blutung)*

Sauerstoff

Moderat

- Akute Hypoxämie, $\text{SpO}_2 < 85\%$ = 10-15 l/min
- Akute Hypoxämie, $\text{SpO}_2 \geq 85-93\%$ = 5-10 l/min

Kontrolliert oder low-dose, Ziel 88-92%

- COPD
- schwere Adipositas (BMI >40 kg/m²)

Kein O₂, wenn $\text{SpO}_2 > 94\%$

- Herzinfarkt
- HRST
- Schlaganfall