| 2013

Annual Update in Intensive Care and Emergency Medicine 2013

Edited by J.-L. Vincent

Springer

Editor

Prof. Jean-Louis Vincent
Erasme Hospital, Université libre de Bruxelles
Brussels, Belgium
jlvincen@ulb.ac.be

ISSN 2191-5709

ISBN 978-3-642-35108-2

ISBN 978-3-642-35109-9 (eBook)

DOI 10.1007/978-3-642-35109-9

Springer Heidelberg New York Dordrecht London

© Springer-Verlag Berlin Heidelberg 2013

This work is subject to copyright. All rights are reserved, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilm or in any other way, and storage in data banks. Duplication of this publication or parts thereof is permitted only under the provisions of the German Copyright Law of September 9, 1965, in its current version, and permission for use must always be obtained from Springer. Violations are liable to prosecution under the German Copyright Law.

The use of general descriptive names, registered names, trademarks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

Product liability: The publishers cannot guarantee the accuracy of any information about dosage and application contained in this book. In every individual case the user must check such information by consulting the relevant literature.

Cover design: WMXDesign GmbH, Heidelberg

Printed on acid-free paper

Springer is part of Springer Science+Business Media
www.springer.com

Prevention of Postoperative Pulmonary Problems Starts Intraoperatively

J. Poelaert, L. Szegedi, and S. Blot

Introduction

Pulmonary complications are a burden for the postoperative patient [1]. Atelectasis and pneumonia have been recognized as the most frequent pulmonary problems in critically ill surgical intensive care unit (ICU) patients [2]. However, it is sometimes difficult to differentiate clinically between atelectasis and pulmonary infection. About a century ago, collapse of the lung, related to insufficient inspiratory power was reported [3, 4]. Altered gas exchange was characteristic in these patients [5], and Bendixen et al. used the term "atelectasis" for the very first time in 1963 [6]. Atelectasis occurs with progressive loss of compliance, not least as a consequence of loss of laryngeal muscle tone and disappearance of intrinsic positive end-expiratory pressure (PEEP), after induction of anesthesia, paralysis and bypassing the oropharynx [7]. Atelectasis resolves only with some deep inflations of the lung (recruitment), adequate pain relief, physiotherapy and early mobilization of the patient.

Pulmonary infections are another problem, in particular for high-risk surgical patients [8]. Although preventive measures against ventilator-associated pneumonia (VAP) have a relatively high adherence rate of 72 %, there is consider-

J. Poelaert (✉)

Dept of Anesthesiology and Perioperative Medicine, University Hospital Brussels, Free University of Brussels, Brussels, Belgium
jan.poelaert@uzbrussel.be

L. Szegedi

Dept of Anesthesiology and Perioperative Medicine, University Hospital Brussels, Free University of Brussels, Brussels, Belgium

S. Blot

Dept of Anesthesiology and Perioperative Medicine, University Hospital Brussels, Free University of Brussels, Brussels, Belgium

able variability among countries [9] and disciplines. Moreover, knowledge of these preventive measures is not widespread among non-ICU physicians. Once present, VAP and postoperative pneumonia necessitate antibiotherapy, sometimes in association with prolonged need for mechanical ventilatory support. Both entities, atelectasis and pulmonary infections, increase considerably the length of stay in the hospital and thus costs. Adequate prevention could lead to a significant reduction in cost both at health care and social-economical levels.

In this review, these two clinical entities are critically examined, in particular in the light of preventive measures that could be undertaken in daily practice. We deliberately do not discuss states of acute lung injury (ALI), which can also be present in postoperative patients, often with detrimental consequences. Although some preventive measures can already be initiated intraoperatively, others can be pursued in the ICU setting.

Incidence of Postoperative Pulmonary Complications

The incidence of atelectasis is hard to assess and difficult to calculate. Only imaging can provide a hint of occurrence rates: In patients with normal lungs, 90 % of patients developed atelectatic regions in the most dependent segments, which were still present 1 h after intubation [7, 10]. In 50 % of patients these atelectasis regions remained present until 24 h after intubation. Moreover, they are related to constitutional characteristics [11].

In contrast, postoperative pneumonia and VAP have been studied extensively both in the general ICU and in postoperative cardiac surgical patients. The incidence of VAP after cardiac surgery encompasses 6.3 episodes per 1,000 days of mechanical ventilation [12]. In a meta-analysis, Smetana et al. assessed the risk factors for postoperative pneumonia in non-cardiothoracic surgical patients [13]; congestive heart failure, age, American Society of Anesthesiologists (ASA)-class > 2, and chronic obstructive pulmonary disease (COPD) appeared to be the most important risk factors. Box 1 shows the most important and relevant risk factors for postoperative pulmonary infection. Postoperative pneumonia was associated with a significant 30-day mortality of 21 % in patients undergoing non-cardiac surgery [14], and in a general surgical cohort, 30-day mortality was higher in patients with a postoperative pulmonary complication (19.5 %; 95 % confidence interval (CI), 12.5–26.5 %) than in those without (0.5 %; 95 % CI, 0.2–0.8 %) [2]. Postoperative pneumonia is also associated with larger hospital costs and longer lengths of stay compared with uncomplicated surgery [15]. Patients especially at risk of postoperative pneumonia and VAP are those undergoing high-risk surgery in the presence of extensive co-morbidities, those undergoing cardiac and cardiovascular surgery [16], open thoracic or thoracoplastic surgery [17], and major upper abdominal procedures and prolonged procedures [2].

knowledge of these
ians. Once present,
sometimes in asso-
. Both entities, ate-
ngth of stay in the
gnificant reduction

ed, in particular in
daily practice. We
which can also be
quences. Although
ely, others can be

Box 1

Risk factors for post-operative and ventilator-associated pneumonia

Major and Complicated Surgery	Non-Cardiothoracic Surgery
Cardiac surgery Thoracic surgery Major vascular surgery Major upper abdominal procedure Other high-risk surgeries Extensive co-morbidities	Congestive heart failure Age > 50 years old ASA-class > 2 Chronic obstructive pulmonary disease
ASA: American Society of Anesthesiologists	

Atelectasis

Pathophysiology

Atelectasis is the result of general anesthesia, ventilation and patient-related factors. In large series of postoperative patients ($n=24,000$), 0.9 % developed hypoxemia, not resolving with only supplemental oxygen [18]. Intraoperatively, atelectasis is responsible for a large part of hypoxemia. Upon administration of a hypnotic, opioid or neuromuscular blocker, muscular tone is abolished within the first few minutes [1]; during endotracheal suctioning, again, atelectasis may be induced through induction of negative intra-thoracic pressure [19]. Surgical manipulation causing compression and/or traction may be aggravated by type and length of intervention, age and body habitus, bleeding, etc. Cephalic displacement of the diaphragm during general anesthesia in the dorsal recumbent position, sometimes with increased intra-abdominal pressures, as well as the Trendelenburg position, intensifies hypoventilation and compression of the most dorsal lung segments (Box 2). Furthermore, loss of muscle tone and the cardiac weight itself may induce further pulmonary compression or inadequate expansion. Rapidly, the dependent lung segments fill up with fluid, hampering any rapid recruitment [20]. In addition to impaired oxygenation and compliance, microvascular leakage occurs as a witness of damaged endothelial function in anesthetized rats when atelectasis is present [21].

Atelectasis can be classified into three groups: Compression atelectasis occurs during general anesthesia and is caused by chest geometry and diaphragm position and motion [22]; in the case of absorption atelectasis, the greater the inspired oxygen fraction after induction, the faster the collapse of the lung regions [23]; loss-of-surfactant atelectasis collapse occurs within 5 min after a vital capacity maneuver at an inspired oxygen fraction (FiO_2) of 1.0 or immediately after removal of PEEP at FiO_2 of 0.5. Atelectasis in turn impedes surfactant function, making the lung more prone to collapse again after reopening [24].

Box 2

Factors predisposing to atelectasis

- Type and length of surgery
- Age
- Body habitus
- Bleeding
- Loss of muscle tone and cephalic displacement of the diaphragm during general anesthesia
- Increased intra-abdominal pressure
- Cardiac weight
- Increased FiO_2 and low alveolar ventilation
- Intra-tracheal suctioning
- Impaired surfactant

Absorption atelectasis is induced by increased FiO_2 and low alveolar ventilation; in addition, impaired surfactant will also provoke atelectasis (Box 2). In addition to hypoxemia, a fall in functional residual capacity (FRC) and decreased lung compliance, a significant increase in pulmonary resistance may occur, mainly by hypoxic pulmonary vasoconstriction, potentially hampering ejection by the right ventricle.

Diagnosis

Atelectasis is clinically suspected whenever impaired oxygenation and decreased lung compliance occur in a situation that could be related to atelectasis. This clinical diagnosis is particularly important intraoperatively, when active diagnosis is difficult. In situations where imaging is possible, bedside chest radiography appears the first choice.

Typically, signs of volume loss characterize the presence of atelectasis:

- displacement of thoracic structures: Interlobar fissure, hemidiaphragm, shift of mediastinal organs
- compensatory hyperinflation of certain lung segments
- typical triangular shape of collapsed lung segment
- obliteration of a lung segment may opacify adjoining parts of the mediastinum.

A more sensitive and specific diagnosis of atelectasis can be made with computed tomography (CT) scans. Already in 1986, Strandberg et al. described with CT scan the presence of atelectasis after induction of anesthesia [7, 10]. A CT scan depicts areas of increased density [11, 25] with an abrupt decrease in FRC and a lowered inflection point on the pressure-volume relationship. The densities correlate with collapsed lung alveoli [26]. Although CT scans indeed offer a near-

diaphragm during

low alveolar ventila-
telectasis (Box 2). In
(FRC) and decreased
ce may occur, mainly
ring ejection by the

ation and decreased
telectasis. This clini-
active diagnosis is
test radiography ap-

atelectasis:
diaphragm, shift of

of the mediastinum.

be made with com-
t al. described with
esia [7, 10]. A CT
ot decrease in FRC
iship. The densities
ndeed offer a near-

ly perfect tool for evaluation of the presence of atelectasis [25], this technique is often not very useful because of the need for transportation of an often-hypoxemic patient. All these methods provide regionally specific information, but only as a snapshot in time. Magnetic resonance imaging (MRI) currently has no additional value in the diagnosis of atelectasis. In contrast, ultrasound allows detection of many deviations in lung structure, pleural fluid, etc. In the ICU, this technique has shown value in detecting alveolar collapse [27]. However, intraoperatively ultrasound is often inadequate to visualize properly collapsed lung tissue. Electrical impedance tomography (EIT) is a new technology, which permits continuous visualization of the distribution of ventilation. Data are continuously displayed in the form of images, waveforms and parameters. Measures can be taken to individually tailor ventilator settings. Determining how different lung regions respond to therapeutic interventions over time is challenging without continuous regional information. The use of EIT in ICU settings is well-established; however, intraoperatively its value still has to be demonstrated. Regional ventilation distribution and recruitment has been assessed in experimental models of direct and indirect ALI as well as in normal lungs [28]. Recently, this technology was used to evaluate the effects of PEEP during pneumoperitoneum in laparoscopic surgery. PEEP led to improved aeration of the dorsal lung parts with consequent improved oxygenation and pulmonary compliance [29].

Prevention and Treatment

Experimental and clinical studies suggest recruitment of collapsed alveoli needing peak inspiratory pressures of >30 cm H₂O for >45 s to allow slow alveoli, with a low time constant, to reopen [30]. Table 1 shows the different studies and compares outcomes after recruitment maneuvers. Additional PEEP is obligatory to keep the alveoli open but only after a recruitment maneuver has been applied [19, 25, 30].

Table 1 Recruitment maneuvers and atelectasis in cardiac surgery: Review of the literature

Author	Specimen	Method	Result
Magnusson (1998) [64]	Pigs	ECC	Atelectasis in control Repeat within 6 h
Murphy (2001) [65]	Human	ECC	Earlier extubation
Tschernko (2002) [66]	Human	ECC	Decreased intrapulmonary shunting
Minkovich (2007) [67]	Human	ECC	After ECC and in the ICU
Shim (2009) [68]	Human	Off pump	Earlier extubation; ICU LOS no change

ECC: extracorporeal circulation; LOS: length of stay

In a small group of cardiac surgical patients, a recruitment maneuver induced a rapid decrease in cardiac output, concomitant with a fall in preload conditions [31]. In an animal experimental setting with lung injury, the same findings were described [32]. Furthermore, care should be taken to monitor right ventricular function because of potential overload during the maneuver [32]. Lower peak inspiratory pressures led to less impressive changes in hemodynamics, although equivalent results on oxygenation and compliance recovery [33]. Continuous perioperative monitoring of pressures, volumes and, if available, regional ventilation is necessary during general anesthesia to allow correct adjustment of ventilatory settings.

Postoperative Pneumonia and Ventilator-Associated Pneumonia

Pathophysiology

Postoperative pneumonia and VAP have been described in terms of aspiration of subglottic secretions and gastroesophageal reflux. Bacterial leakage (often 10^{10} bacteria/ml secretion [34]) in conjunction with impaired lung defense, results in tracheobronchial colonization and induction of ventilator-associated tracheobronchitis (VAT) and VAP. This aspiration risk is not a minor issue, as shown by Mahul et al., but can run up to volumes of 150 ml per day [34]; intraoperatively, even in the prone position, drainage from nose and mouth could be as high as 50 ml/h [35]. Risk factors for aspiration include not only the endotracheal tube (ETT), but also mechanical ventilation without PEEP, tracheal suctioning, the presence of a nasogastric tube with gastroesophageal reflux, and various patient-related factors (Box 3).

Box 3

Risk factors for aspiration

- Type of tracheal tube
- Mechanical ventilation without positive end-expiratory pressure
- Intra-tracheal suctioning
- Presence of the nasogastric tube
- Gastroesophageal reflux

Patients suffering from preoperative hypoxemia or a pulmonary infection less than one month before surgery show a significantly increased risk of postoperative pulmonary complications [2].

All types of mechanical ventilation are non-physiological and, in a vicious circle manner, cause further damage to the lungs, with either a biochemical injury (with loss of cytokines, complement, prostanooids, reactive oxygen species, leukotrienes, proteases), and/or a biophysical injury (shear, overdistension, cyclic stretch, increased intrathoracic pressure). Protective tools, such as barrel-shaped polyvinyl

Prev

chl
in
abc
for

Di

VA
the
Gr
Th
ob
che
tion
not

Pr

Pre
inf
culFig
pos
con
det

aneuver induced a
ad conditions [31].
findings were de-
entricular function
r peak inspiratory
though equivalent
ious perioperative
ntilation is neces-
sity settings.

d Pneumonia

s of aspiration of
e (often 10^{10} bac-
se, results in tra-
cheobronchitis
n by Mahul et al.,
vely, even in the
10 ml/h [35]. Risk
T), but also me-
ice of a nasogas-
factors (Box 3).

y infection less
of postoperative

in a vicious cir-
chemical injury
species, leukot-
i, cyclic stretch,
aped polyvinyl

chloride (PVC) cuffed ETTs, do not prevent aspiration as demonstrated recently in *in vitro* studies [35, 36]: with an ETT in a rigid cylinder, a dye solution poured above the barrel-shaped PVC cuff of the ETT descended along the channels formed within the PVC.

Diagnosis

VAP is diagnosed on the basis of several criteria. Essential clinical criteria include the presence of fever, purulent sputum and hypoxemia. In addition, a positive Gram-stain, leukocytosis and a positive chest X-ray should be present [37, 38]. The problem occurs after major surgery in patients in whom these criteria are not obvious or overt. Fabregas et al. suggested that the presence of infiltrates on the chest radiograph and two of three clinical criteria (leukocytosis, purulent secretions, fever) had a sensitivity of 69 % and a specificity of 75 % [39], rates that are not worse than those with the clinical pulmonary infection score.

Preventive Measures

Prevention is the cornerstone with respect to abolishing atelectasis and pulmonary infections induced through inadequate sealing and subsequent leakage along the cuff of the endotracheal tube (Fig. 1).

Fig. 1 Measures to prevent post-operative pulmonary complications (see text for details)

Preoperative Measures

In esophageal cancer surgery and in cardiac surgical patients, a clear decline in occurrence rate of postoperative pneumonia was observed with teeth brushing (40, 41). Rinsing of the buccal cavity with a 2 % solution of chlorhexidine reduces the frequency of postoperative pneumonia considerably [42]. In a recent meta-analysis, Labeau et al. showed that use of antiseptics resulted in a significant risk reduction of VAP (RR 0.67; 95 % CI 0.50–0.88; $p=0.004$) [43]. Chlorhexidine application was shown to be effective (RR 0.72; 95 % CI 0.55–0.94; $p=0.02$), in particular in a cardiac surgical subset (RR 0.41, 95 % CI 0.17–0.98).

Intraoperative Measures

The rates of VAP and of postoperative pneumonia are time-related: The longer the surgical procedure lasts, the greater the risk of aspiration and thus pulmonary infection. A surgical procedure of more than 2 h increases the risk 4-fold [44]. In this respect, comorbidities play an important role. The sooner the patient can breathe spontaneously and be weaned from the ventilator, the better. This implies that sedation levels in the ICU should be moderate and systematically adapted to the disease state of the patient; the anesthetic level also needs to be adapted in this manner. In addition to these important issues, some preventive measures can be undertaken to reduce intraoperative aspiration.

- Lubrication: In one-lung ventilated thoracic surgery patients, Sanjay et al. demonstrated that lubricated PVC cuffs of a double-lumen tube sealed the trachea nearly twice as long as non-lubricated tubes with a resultant decrease in pulmonary infection rates [45].
- Cuff pressure management: Since the introduction of the high-volume low-pressure tube, cuff pressures have been set considerably lower than with the high-pressure low-volume cuffed ETT. Tracheal damage has decreased considerably, but the risk of underinflation of the cuff has increased [46]. Even with an adequately inflated cuff, the rate of leakage must still be considered. Intervals of a cuff pressure <20 cm H₂O during the first 8 ventilation days significantly increased the risk of postoperative pneumonia (OR 4.23, CI 1.12–15.92). Intermittent control of cuff pressure, at least every four hours, to obtain a cuff pressure between 20 and 30 cm H₂O [47, 48], is one approach to this problem. Care should be taken to release the cuff pressure manometer from the valve of the cuff, so that minimal leakage occurs; in this way a slightly increased lower level of, e.g., 24 cm H₂O should be considered. Not controlling the cuff pressure will certainly lead to either underinflation or overinflation and to adequate cuff pressures in less than 20 % of cases [49].
- PEEP: Early onset VAP, occurring less than 5 days after intubation, was significantly less frequent in non-hypoxemic patients ventilated with 5–8 cm H₂O PEEP during lung surgery [50]. Prophylactic PEEP was found to be safe and to reduce VAP-rates, including late-onset VAP [51].
- Aspiration of subglottic secretions with a dedicated ETT, with a PVC cuff, may reduce the risk of aspiration [51]. In only 31 patients after cardiac surgery,

Bou
tor
mor
risk
cate
surg
sub
ons
eve
par
a re
cret

clu
nor
anc
tio
duc
im
[57
• Ch
cul
tio
sm
lur
an
str
of
[3
gi
di
fe
vi

tr
la
w
of
cu
• C
bi
cl
rr
te
a

lear decline in
h brushing (40,
ine reduces the
nt meta-analy-
ant risk reduc-
xidine applica-
; $p=0.02$), in

The longer the
us pulmonary
4-fold [44]. In
e patient can
. This implies
lly adapted to
dapted in this
asures can be

ay et al. dem-
d the trachea
ise in pulmo-

volume low-
han with the
ased consid-
]. Even with
dered. Inter-
days signifi-
l. 12–15.92).

to obtain a
to this prob-
er from the
slightly in-
controlling
nflation and

n, was sig-
5–8 cm H₂O
safe and to

□ cuff, may
ac surgery,

Bouza et al. demonstrated a significant difference in VAP-rate, days on ventilator and length of stay in the ICU in those patients needing ventilator support for more than 48 h. Combined use of PEEP and subglottic aspiration reduced the risk of VAP from 48 % in controls to 27 % in patients intubated with this dedicated ET tube [52]. Similarly, Mahul et al. demonstrated, in a mixed medical-surgical ICU population expected to require >72 h of ventilatory support, that subglottic secretion drainage resulted in a lower frequency of VAP or a later onset [34]. Valles et al. reported a similar reduction in VAP rate without, however, any difference in outcome [53]. Other investigators have described comparable results [12, 54, 55]. Estimating the cost of VAP treatment at €4,300 and a reduction in the VAP-rate of 30 % with a dedicated ETT with subglottic secretion drainage, a cost-benefit equilibrium is present.

The problems and debate associated with the single drainage lumen tube include the correct dorsal position of this lumen and the presence of an open, non-occluded lumen. Indeed, viscous secretions may occlude the sole lumen and suctioning against the tracheal wall may also occlude the lumen of the suction channel [56]. Occlusion was found in 43 % of investigated patients. Introduction of an ET tube with multiple drainage suction holes above the cuff could improve the preventive function of such tubes by less frequent occlusion rates [57], but larger *in vivo* studies are missing.

- Changed cuff design: Several *in vitro* studies have suggested that a modified cuff design could reduce the risk of aspiration and transition of subglottic secretions into the trachea. Indeed, Dullenkopf et al. demonstrated the presence of small channels in the PVC cuff, permitting transit of secretions into the tracheal lumen; the existence of these channels has been shown both with fluoroscopy and with dye solutions [58]. In an *in vitro* investigation, Zanella et al. demonstrated that a tapered-shaped, cuffed ET tube clearly delayed the risk of transit of dye solution up to 12 h on the condition that PEEP (5–8 cm H₂O) was present [36], in contrast to a barrel-shaped cuffed ET-tube, for which the security margin lasted only 2 and maximum 6 h. Finally, Dave et al. demonstrated in three different sized artificial tracheas that the tapered-shaped PVC cuff was more effective in preventing leakage [59]. To the best of our knowledge, published *in vivo* studies are not yet available.

Theoretically, it is conceivable that a tapered-shaped cuff indeed seals the tracheal lumen much better as this cuff fits perfectly into the lumen at a particular level [60]. The perfect fit is closely related to a particular level of the cuff at which no channels in the PVC are formed. Depending on the tracheal anatomy of a particular patient, this improved sealing can be at an upper or rather lower cuff level.

- Changed cuff material: In the past decade, ETTs with a polyurethane cuff have been developed. Because of the ultra-thin wall, polyurethane cuffs have smaller channels formed by the folds in the cuff. This characteristic has resulted in more favorable research outcomes in comparison with their PVC cuffed counterparts. In an *in vitro* study by Dave et al., it was demonstrated that PVC cuffs always leaked more and faster compared to polyurethane cuffs [59]. In a ran-

domized controlled trial in 134 cardiac surgical patients, it was demonstrated that use of polyurethane cuffed tubes resulted in a reduced prevalence of postoperative pneumonia. The risk of pneumonia was reduced by nearly 50%, albeit the prevalence of pneumonia in the control group was particularly high [48]. Similarly, Lucangelo et al. showed that a barrel shaped polyurethane cuffed ETT sealed the tracheal lumen better [61]. In children, Dullenkopf et al. demonstrated similar results [62].

Postoperative Measures

Intraoperative measures will certainly diminish considerably the risk of aspiration and, therefore, decrease the potential risk for initial bacterial descent towards the trachea. Preventive measures such as teeth brushing and antiseptic mouth rinsing should be restarted in the postoperative setting. Supine body position is a clear risk factor for pulmonary infections in ventilated patients; in a study comparing supine with semi-recumbent positioning in 86 intubated and mechanically ventilated patients, the rate of microbiologically confirmed pneumonia was significantly lower in the semirecumbent patients ($p=0.018$), and the odds ratio for development of nosocomial pneumonia was 6.8 (1.7–26.7) ($p=0.006$) for supine position [63]. Although the semi-recumbent position is not possible intraoperatively, whenever possible in terms of hemodynamic status, patients should be positioned in a semi-upright position (about 40°) once extubated in the operating room or postoperative care setting.

Conclusion

It is obvious that prevention of (postoperative) pulmonary complications should be a main goal in every perioperative setting. Preventive strategies start already before surgery and should be initiated on the ward with systematic application of oral hygiene. Intraoperatively, anesthesiologists should start more extensive preventive action including optimal cuff pressure and PEEP administration in addition to monitored and individually titrated mechanical ventilator parameters. Using a dedicated ETT is only supported by *in vitro* studies and should be related to the presumed duration of postoperative ventilator support at induction of the anesthetic.

References

1. Hedenstierna G (1989) Mechanisms of postoperative pulmonary dysfunction. *Acta Chir Scand Suppl* 550:152–158
2. Canet J, Gallart L, Gomar C et al (2010) Prediction of postoperative pulmonary complications in a population-based surgical cohort. *Anesthesiology* 113:1338–1350
3. Morrow DJ, Stimson PM (1947) Atelectasis in poliomyelitis. *Med Clin North Am* 31:609–625

4. Sutherland J (1998) Lung. *Proc R Soc Med* 91:1–10
5. Nunn JF (1998) with spo. *Br J Anaesth* 81:1–10
6. Bendixen PP (1998) during g. *Br J Anaesth* 81:1–10
7. Strandberg P (1998) anaesthe. *Br J Anaesth* 81:1–10
8. Varbanov I (1998) and post. *Br J Anaesth* 81:1–10
9. Ricard J (1998) control a. *Br J Anaesth* 81:1–10
10. Strandberg P (1998) pendent. *Br J Anaesth* 81:1–10
11. Strandberg P (1998) factors p. *Br J Anaesth* 81:1–10
12. Kollef M (1998) tion of su. *Br J Anaesth* 81:1–10
13. Smetana M (1998) for nonc. *Br J Anaesth* 81:1–10
14. Arozulla A (1998) a multife. *Br J Anaesth* 81:1–10
15. Dimick J (1998) tal costs. *Br J Anaesth* 81:1–10
16. Hortal J (1998) pneumo. *Br J Anaesth* 81:1–10
17. Liang J (1998) tions in. *Br J Anaesth* 81:1–10
18. Rose DF (1998) in the p. *Br J Anaesth* 81:1–10
19. Heinze I (1998) tional re. *Br J Anaesth* 81:1–10
20. Gattinoni L (1998) distress s. *Br J Anaesth* 81:1–10
21. Duggan M (1998) Atelecta. *Br J Anaesth* 81:1–10
22. Warner J (1998) patients. *Br J Anaesth* 81:1–10
23. Joyce C (1998) compute. *Br J Anaesth* 81:1–10

t was demonstrated prevalence of post- by nearly 50 %, al- is particularly high aped polyurethane n, Dullenkopf et al.

e risk of aspiration escent towards the ptic mouth rinsing ition is a clear risk comparing supine ally ventilated pa- significantly lower or development of ine position [63]. atively, whenever itioned in a semi- n or postoperative

cations should be art already before cation of oral hy- nsive preventive n in addition to ters. Using a de- lated to the pre- the anesthetic.

inction. Acta Chir

monary complica-

io

Clin North Am

4. Sutherland GA (1914) Anterior poliomyelitis; paralysis of abdominal muscles; collapse of lung. *Proc R Soc Med* 7:31–32
5. Nunn JF (1964) Factors influencing the arterial oxygen tension during halothane anaesthesia with spontaneous respiration. *Br J Anaesth* 36:327–341
6. Bendixen HH, Hedley-Whyte J, Laver MB (1963) Impaired oxygenation in surgical patients during general anesthesia with controlled ventilation. A concept of atelectasis. *N Engl J Med* 269:991–996
7. Strandberg A, Tokics L, Brismar B, Lundquist H, Hedenstierna G (1986) Atelectasis during anaesthesia and in the postoperative period. *Acta Anaesthesiol Scand* 30:154–158
8. Varban OA, McCoy TP, Westcott C (2011) A comparison of pre-operative comorbidities and post-operative outcomes among patients undergoing laparoscopic nissen fundoplication at high- and low-volume centers. *J Gastrointest Surg* 15:1121–1127
9. Ricard JD, Conti G, Boucherie M et al (2012) A European survey of nosocomial infection control and hospital-acquired pneumonia prevention practices. *J Infect* 65:285–291
10. Strandberg A, Hedenstierna G, Tokics L, Lundquist H, Brismar B (1986) Densities in dependent lung regions during anaesthesia: atelectasis or fluid accumulation? *Acta Anaesthesiol Scand* 30:256–259
11. Strandberg A, Tokics L, Brismar B, Lundquist H, Hedenstierna G (1987) Constitutional factors promoting development of atelectasis during anaesthesia. *Acta Anaesthesiol Scand* 31:21–24
12. Kollef MH, Skubas NJ, Sundt TM (1999) A randomized clinical trial of continuous aspiration of subglottic secretions in cardiac surgery patients. *Chest* 116:1339–1346
13. Smetana GW, Lawrence VA, Cornell JE (2006) Preoperative pulmonary risk stratification for noncardiothoracic surgery: systematic review for the American College of Physicians. *Ann Intern Med* 144:581–595
14. Arozullah AM, Khuri SF, Henderson WG, Daley J (2001) Development and validation of a multifactorial risk index for predicting postoperative pneumonia after major noncardiac surgery. *Ann Intern Med* 135:847–857
15. Dimick JB, Chen SL, Taheri PA, Henderson WG, Khuri SF, Campbell DA Jr (2004) Hospital costs associated with surgical complications: a report from the private-sector National Surgical Quality Improvement Program. *J Am Coll Surg* 199:531–537
16. Hortal J, Munoz P, Cuerpo G, Litvan H, Rosseel PM, Bouza E (2009) Ventilator-associated pneumonia in patients undergoing major heart surgery: an incidence study in Europe. *Crit Care* 13:R80
17. Liang J, Qiu G, Shen J et al (2010) Predictive factors of postoperative pulmonary complications in scoliotic patients with moderate or severe pulmonary dysfunction. *J Spinal Disord Tech* 23:388–392
18. Rose DK, Cohen MM, Wigglesworth DF, DeBoer DP (1994) Critical respiratory events in the postanesthesia care unit. Patient, surgical, and anesthetic factors. *Anesthesiology* 81:410–418
19. Heinze H, Eichler W, Karsten J, Sedemund-Adib B, Heringlake M, Meier T (2011) Functional residual capacity-guided alveolar recruitment strategy after endotracheal suctioning in cardiac surgery patients. *Crit Care Med* 39:1042–1049
20. Gattinoni L, Pelosi P, Suter P, Pedoto A, Vercesi P, Lissoni A (1998) Acute respiratory distress syndrome caused by pulmonary and extrapulmonary disease. Different syndromes? *Am J Respir Crit Care Med* 158:3–11
21. Duggan M, McCaul CL, McNamara PJ, Engelberts D, Ackerley C, Kavanagh BP (2003) Atelectasis causes vascular leak and lethal right ventricular failure in uninjured rat lungs. *Am J Respir Crit Care Med* 167:1633–1640
22. Warner DO, Warner MA, Barnes RD et al (1996) Perioperative respiratory complications in patients with asthma. *Anesthesiology* 85:460–467
23. Joyce CJ, Baker AB, Kennedy RR (1993) Gas uptake from an unventilated area of lung: computer model of absorption atelectasis. *J Appl Physiol* 74:1107–1116

24. Oyarzun MJ, Iturriaga R, Donoso P et al (1991) Factors affecting distribution of alveolar surfactant during resting ventilation. *Am J Physiol* 261:L210-L217
25. Reinius H, Jonsson L, Gustafsson S et al (2009) Prevention of atelectasis in morbidly obese patients during general anesthesia and paralysis: A computerized tomography study. *Anesthesiology* 111:979-987
26. Hedenstierna G, Lundquist H, Lundh B et al (1989) Pulmonary densities during anaesthesia. An experimental study on lung morphology and gas exchange. *Eur Respir J* 2:528-535
27. Karabinis A, Saranteas T, Karakitsos D et al (2008) The "cardiac-lung mass" artifact: an echocardiographic sign of lung atelectasis and/or pleural effusion. *Crit Care* 12:R122
28. Wrigge H, Zinserling J, Muders T et al (2008) Electrical impedance tomography compared with thoracic computed tomography during a slow inflation maneuver in experimental models of lung injury. *Crit Care Med* 36:903-909
29. Karsten J, Luepschen H, Grossherr M et al (2011) Effect of PEEP on regional ventilation during laparoscopic surgery monitored by electrical impedance tomography. *Acta Anaesthesiol Scand* 55:878-886
30. Futier E, Constantin J-M, Pelosi P et al (2010) Intraoperative recruitment maneuver reverses detrimental pneumoperitoneum-induced respiratory effects in healthy weight and obese patients undergoing laparoscopy. *Anesthesiology* 113:1310-1319
31. Nielsen J, Ostergaard M, Kjaergaard J et al (2005) Lung recruitment maneuver depresses central hemodynamics in patients following cardiac surgery. *Intensive Care Med* 31:1189-1194
32. Nielsen J, Nilsson M, Freden F et al (2006) Central hemodynamics during lung recruitment maneuvers at hypovolemia, normovolemia and hypervolemia. A study by echocardiography and continuous pulmonary artery flow measurements in lung-injured pigs. *Intensive Care Med* 32:585-594
33. Odenstedt H, Lindgren S, Olegard C et al (2005) Slow moderate pressure recruitment maneuver minimizes negative circulatory and lung mechanic side effects: evaluation of recruitment maneuvers using electric impedance tomography. *Intensive Care Med* 31:1706-1714
34. Mahul P, Auboyer C, Jospe R et al (1992) Prevention of nosocomial pneumonia in intubated patients: respective role of mechanical subglottic secretions drainage and stress ulcer prophylaxis. *Intensive Care Med* 18:20-25
35. Young PJ, Blunt MC (1999) Improving the shape and compliance characteristics of a high-volume, low-pressure cuff improves tracheal seal. *Br J Anaesth* 83:887-889
36. Zanella A, Scaravilli V, Isgrò S et al (2011) Fluid leakage across tracheal tube cuff, effect of different cuff material, shape, and positive expiratory pressure: a bench-top study. *Intensive Care Med* 37:343-347
37. Johanson WG Jr, Pierce AK, Sanford JP, Thomas GD (1972) Nosocomial respiratory infections with gram-negative bacilli. The significance of colonization of the respiratory tract. *Ann Intern Med* 77:701-706
38. Pugin J, Auckenthaler R, Mili N, Janssens JP, Lew PD, Suter PM (1991) Diagnosis of ventilator-associated pneumonia by bacteriologic analysis of bronchoscopic and nonbronchoscopic "blind" bronchoalveolar lavage fluid. *Am Rev Respir Dis* 143:1121-1129
39. Fabregas N, Ewig S, Torres A et al (1999) Clinical diagnosis of ventilator associated pneumonia revisited: comparative validation using immediate post-mortem lung biopsies. *Thorax* 54:867-873
40. Akutsu Y, Matsubara H, Shuto K et al (2010) Pre-operative dental brushing can reduce the risk of postoperative pneumonia in esophageal cancer patients. *Surgery* 147:497-502
41. Stonecypher K (2010) Ventilator-associated pneumonia: the importance of oral care in intubated adults. *Crit Care Nurs Q* 33:339-347
42. Panchabhai TS, Dangayach NS, Krishnan A, Kothari VM, Karnad DR (2009) Oropharyngeal cleansing with 0.2 % chlorhexidine for prevention of nosocomial pneumonia in critically ill patients: an open-label randomized trial with 0.01 % potassium permanganate as control. *Chest* 135:1150-1156

43.

44.

45.

46.

47.

48.

49.

50.

51.

52.

53.

54.

55.

56.

57.

58.

59.

60.

61.

62.

43. Labeau SO, Van de Vyver K, Brusselaers N, Vogelaers D, Blot SI (2011) Prevention of ventilator-associated pneumonia with oral antiseptics: a systematic review and meta-analysis. *Lancet Infect Dis* 11:845–854
44. Delgado-Rodriguez M, Medina-Cuadros M, Martinez-Gallego G, Sillero-Arenas M (1997) Usefulness of intrinsic surgical wound infection risk indices as predictors of postoperative pneumonia risk. *J Hosp Infect* 35:269–276
45. Sanjay PS, Miller SA, Corry PR, Russell GN, Pennefather SH (2006) The effect of gel lubrication on cuff leakage of double lumen tubes during thoracic surgery. *Anaesthesia* 61:133–137
46. Rello J, Sonora R, Jubert P, Artigas A, Rue M, Valles J (1996) Pneumonia in intubated patients: role of respiratory airway care. *Am J Respir Crit Care Med* 154:111–115
47. Valencia M, Ferrer M, Farre R et al (2007) Automatic control of tracheal tube cuff pressure in ventilated patients in semirecumbent position: a randomized trial. *Crit Care Med* 35:1543–1549
48. Poelaert J, Depuydt P, De Wolf A, Van de Velde S, Herck I, Blot S (2008) Polyurethane cuffed endotracheal tubes to prevent early postoperative pneumonia after cardiac surgery: a pilot study. *J Thorac Cardiovasc Surg* 135:771–776
49. Nseir S, Brisson H, Marquette CH et al (2009) Variations in endotracheal cuff pressure in intubated critically ill patients: prevalence and risk factors. *Eur J Anaesthesiol* 26:229–234
50. Ludwig C, Angenendt S, Martins R, Mayer V, Stoelben E (2011) Intermittent positive-pressure breathing after lung surgery. *Asian Cardiovasc Thorac Ann* 19:10–13
51. Lacherade JC, De Jonghe B, Guezennec P et al (2010) Intermittent subglottic secretion drainage and ventilator-associated pneumonia: A multicenter trial. *Am J Respir Crit Care Med* 182:910–917
52. Bouza E, Perez MJ, Munoz P, Rincon C, Barrio JM, Hortal J (2008) Continuous aspiration of subglottic secretions in the prevention of ventilator-associated pneumonia in the postoperative period of major heart surgery. *Chest* 134:938–946
53. Valles J, Artigas A, Rello J et al (1995) Continuous aspiration of subglottic secretions in preventing ventilator-associated pneumonia. *Ann Intern Med* 122:179–186
54. Lorente L, Lecuona M, Jimenez A, Mora ML, Sierra A (2007) Influence of an endotracheal tube with polyurethane cuff and subglottic secretion drainage on pneumonia. *Am J Respir Crit Care Med* 176:1079–1083
55. Smulders K, van der Hoeven H, Weers-Pothoff I, Vandenbroucke-Grauls C (2002) A randomized clinical trial of intermittent subglottic secretion drainage in patients receiving mechanical ventilation. *Chest* 121:858–862
56. Dragoumanis CK, Vretzakis GI, Papaioannou VE, Didilis VN, Vogiatzaki TD, Pneumatikos IA (2007) Investigating the failure to aspirate subglottic secretions with the Evac endotracheal tube. *Anesth Analg* 105:1083–1085
57. Doyle A, Fletcher A, Carter J, Blunt M, Young P (2011) The incidence of ventilator-associated pneumonia using the PneuX System with or without elective endotracheal tube exchange: A pilot study. *BMC Res Notes* 4:92
58. Dullenkopf A, Gerber A, Weiss M (2003) Fluid leakage past tracheal tube cuffs: evaluation of the new Microcuff endotracheal tube. *Intensive Care Med* 29:1849–1853
59. Dave MH, Frotzler A, Spielmann N, Madjdpour C, Weiss M (2010) Effect of tracheal tube cuff shape on fluid leakage across the cuff: an in vitro study. *Br J Anaesth* 105:538–543
60. Poelaert J (2011) Ventilator-associated pneumonia and cuff shape. *Am J Respir Crit Care Med* 184:485
61. Lucangelo U, Zin WA, Antonaglia V et al (2008) Effect of positive expiratory pressure and type of tracheal cuff on the incidence of aspiration in mechanically ventilated patients in an intensive care unit. *Crit Care Med* 36:409–413
62. Dullenkopf A, Gerber AC, Weiss M (2005) Nitrous oxide diffusion into tracheal tube cuffs – efficacy of a new prototype cuff pressure release valve. *Acta Anaesthesiol Scand* 49:1072–1076

63. Drakulovic MB, Torres A, Bauer TT, Nicolas JM, Nogue S, Ferrer M (1999) Supine body position as a risk factor for nosocomial pneumonia in mechanically ventilated patients: a randomised trial. *Lancet* 354:1851–1858
64. Magnusson L, Wicky S, Tyden H, Hedenstierna G (1998) Repeated vital capacity manoeuvres after cardiopulmonary bypass: effects on lung function in a pig model. *Br J Anaesth* 80:682–684
65. Murphy GS, Szokol JW, Curran RD, Votapka TV, Vender JS (2001) Influence of a vital capacity maneuver on pulmonary gas exchange after cardiopulmonary bypass. *J Cardiothorac Vasc Anesth* 15:336–340
66. Tschernko EM, Bambazek A, Wisser W et al (2002) Intrapulmonary shunt after cardiopulmonary bypass: the use of vital capacity maneuvers versus off-pump coronary artery bypass grafting. *J Thorac Cardiovasc Surg* 124:732–738
67. Minkovich L, Djaiani G, Katznelson R et al (2007) Effects of alveolar recruitment on arterial oxygenation in patients after cardiac surgery: a prospective, randomized, controlled clinical trial. *J Cardiothorac Vasc Anesth* 21:375–378
68. Shim J, Chun D, Choi Y, Lee J, Hong S, Kwak Y (2009) Effect of early vital capacity maneuver on respiratory variables during multivessel off-pump coronary artery bypass graft surgery. *Crit Care Med* 37:539–544

Period From

G. Marx ar

Introdu

Shoemaker major sur, optimizati least 26 o operative (DO₂) dec medium-to Several dynamic (AKI) [28 nary tract [31], and analyses a conducted ing abdom tive hemo significant

G. Marx (✉)
Klinik für O
Aachen, Pa
E-mail: gma

F. Michard
Critical Care

J.-L. Vincen
DOI 10.100