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Ventilator-associated pneumonia monitoring according to the INICC project at one centre

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Abstract

Background: Pneumonia is a common complication of hospitalisation in severely ill patients who need mechanical ventilation. The aim of this study was to assess the usefulness of the International Nosocomial Infection Control Consortium programme for the surveillance of ventilator-associated pneumonia (VAP).

Methods: A prospective study (1 Jan 2012–30 June 2014) was conducted in the 20-bed ICU. The device utilisation ratios for lung ventilation and the frequency (density and incidence) and aetiology of VAP were estimated in ICU patients.

Results: From a total of 1097 patients, VAP infections were diagnosed in 93. Thirty percent of patients with VAP died. The incidence index was 8.47 per 100 admissions to the ICU. VAP infections accounted for 46% of the overall count of device-associated healthcare-associated infections. Mechanical ventilation was used in 71 ± 8 patients during the 11 862 patient days and 8425 ventilation days. The rate of VAP per 1000 ventilator days was 11.15/9.34/10.23 in years 2012/2013/2014 (half a year), respectively. The main VAP pathogens were *Acinetobacter baumannii* (45%) and *Pseudomonas aeruginosa* (17%).

Conclusion: During the reported time span, the incidence of VAP was lower than in the INICC report (2007–2012), but it was tenfold higher than in the NHSN/CDC report (dated 2012). Because of the unchanged VAP level during the 2.5-year observation period, the root cause needs to be determined and action should be taken to resolve this issue.

Key words: hospital infections, ventilator-associated pneumonia

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Ventilator-associated pneumonia (VAP) is recognised in patients who are mechanically ventilated (for at least 48 h) using artificial airways (an endotracheal or tracheostomy tube) [1]. According to the binding definitions, the presence of the endotracheal/tracheostomy tube alone does not allow the physician to diagnose VAP. The most common cause of VAP is thought to be aspiration of microorganisms from the oral cavity [2]. In the empiric therapy of VAP, it is essential to characterise VAP according to the onset of the first symptoms into an early form (up to 5 days from endotracheal intubation, which is usually caused by commensal microorganisms of patients) or a late form (5 days after endotracheal intubation, which is most frequently caused by multi-drug resistant Gram-negative bacteria that are characteristic of a particular intensive care unit).

Pneumonia occurs in 7–20% of mechanically ventilated patients [2, 3]. The incidence of VAP compared with the total number of hospitalised patients is highly varied [2, 3]. According to the findings of American studies, the incidence reaches 2.5–22.8% [4, 5], whereas in a European study involving a smaller population, VAP was recognised in 9.3% of patients (from a total of 9080 hospitalised patients). According to the risk factors, the density of this nosocomial infection in the American ICUs ranges from 1.27 to 8.5/1000 days of mechanical ventilation [4, 7]. The density of VAP in Polish hospitals is estimated at 15.5–16.7 per 1000 days of mechan-

ical ventilation [8, 9]. According to a large study including 1735 patients treated in 27 European ICUs, the rate of VAP in various age groups were 13.7, 16.6 and 13/1000 ventilators days in patients aged 45–64, 65–74 and older than 75 years, respectively [10]. The mortality among VAP patients is 70% or is controversially low (1–1.5%) when estimated after the error correction resulting from the impact of concomitant diseases [11, 12]. A study performed in 88 689 individuals revealed that VAP caused statistically significant prolongation of mechanical ventilation (21.8 vs. 10.3 days, $P < 0.0001$), ICU therapy (20.5 vs. 11.6 days, $P < 0.0001$) and hospital treatment (32.6 vs. 19.5 days, $P < 0.0001$) [4]. The cost of therapy increased by about 40 000 USD for one patient with the diagnosis of VAP (American data), which is equally substantial [4, 6].

The best-known recommendations for reducing the incidence of respiratory infections were named the Institute for Healthcare Improvement (IHI) Ventilator Bundle. The recommendations include the following interventions: the use of the sedation protocol with everyday awakening, daily assessment of possible awakening or extubation, elevation of a bed head at the angle of 30–45°, application of chlorhexidine for oral care, use of proton pump inhibitors for preventing gastric ulcer, subglottic aspiration in those ventilated for more than 48 h, and prophylaxis of deep vein thrombosis [13, 14]. The introduction of preventive recommendations and monitoring of respiratory tract infections associated with mechanical ventilation is an element of surveillance in patients undergoing mechanical ventilation. These approaches decrease infection incidence, shorten hospitalisations, reduce treatment costs and improve patient safety [15, 16].

The aims of the present study were to estimate the incidence and to determine the aetiological factors of ventilator–associated pneumonia in patients treated in one ICU during a 30-month observation period. Moreover, the trend of VAP incidence was analysed and compared with the results of the most important international reports, i.e., the Center for Disease Control and Prevention/National Healthcare Safety Network (CDC/NHSN) and International Nosocomial Infection Control Consortium (INICC) [17, 18] and with our previous publications.

METHODS

The study was observational and prospective and was performed between 1.01.2012–30.06.2014 at the 20-bed ICU of the surgical and internal medicine profile. Analysis involved all patients who were hospitalised in the ICU for longer than 48 hours and who required invasive ventilation due to acute or chronic exacerbated respiratory failure. The infection surveillance charts included information on the use of mechanical ventilation or otherwise (to calculate the number of ventilation days) and the number of hospitalised patients (to calculate the number of patient days). The detailed data regarding identification of the patients, clinical data and data on the medical device used, risk factors for infections, diagnostic methods and data on the pneumonia cases diagnosed were also introduced to the monitoring programme of the Device-Associated Healthcare-Associated Infections (DA-HAIs). Moreover, the study presented the following basic elements of VAP monitoring according to the INICC: density of VAP/1000 ventilation days and incidence of VAP/100 ICU admissions, intensity of mechanical ventilation procedures (ventilator utilisation ratio — VU-R) and microbiological profiles of infections.

During VAP monitoring, the following were calculated once a month:

1. device utilisation ratio (DU-R), the ratio determining the percentage of mechanically ventilated patients (ventilator utilisation ratio — VU-R):

$$VU\text{-}R = \text{number of mechanical ventilation days} / \text{total number of person-days} \times 100;$$

2. rate of VAP (density):

$$\text{the rate of VAP} = \text{number of VAP patients} / \text{total number of ventilator days} \times 1000;$$

3. moreover, the incidence of VAP was calculated, i.e., the number of new VAP cases during one year (2010–2013) and six months (2014) per 100 ICU admissions:

$$\text{the incidence index} = \text{number of patients with VAP} / \text{number of patients admitted in a given period} \times 100.$$

CLINICAL AND MICROBIOLOGICAL DIAGNOSIS OF VAP

VAP was recognised based on the CDC/NHSN guidelines accepted by the INICC [19, 20]. VAP, denoted as PNU1 according to the classic CDC definition, was diagnosed in patients with endotracheal tubes undergoing mechanical ventilation for at least 48 hours, who also had radiologic changes of the lungs, at least one of the clinical symptoms of infection (body temperature > 38°C or < 36°C, leucocytosis ≥ 12 G L^{-1} or leucopoenia < 4 G L^{-1}) and at least two of the following criteria: 1) purulent secretion from the bronchial tree; 2) impaired parameters of gas exchange; 3) cough or respiratory disorders; 4) auscultation changes characteristic of pneumonia [19] and microbiological confirmation. Since 2013, the diagnosis of VAP was based on a new definition of probable VAP, which was defined as patients with respiratory tract infections with at least two days of stability of respiratory parameters during ventilator therapy for whom the

respiratory efficiency deteriorated and required increased positive endexpiratory preassure (PEEP) by at least 3 cm H_2O above the daily minimum and oxygen supply in the respiratory mixture (F_IO_2) at least 20% higher than the daily minimum. According to the new definition, impaired respiratory parameters have to be maintained for at least two calendar days [20]. The microbiological diagnosis of VAP in this form of infection considers the quantitative culture of bronchial tree secretion and presence of purulent secretion (> 25 neutrophils and < 10 epithelial cells in the visual field on microscopy). VAP was radiologically diagnosed; however, according to the current definition of probable VAP, radiologic lung changes are not necessary for diagnosis [20]. The bronchial tree secretion was subjected to the recommended microbiological diagnostic procedures for the presence of pathogens. Once VAP was suspected, bronchial secretion was collected using mini-bronchoalveolar lavage (mini-BAL) and diagnosed quantitatively. When the patient required therapeutic bronchofibroscopy during the course of pneumonia, bronchial secretion was collected using bronchoalveolar lavage (BAL) as well as diagnosed quantitatively. In cases of severe sepsis or septic shock, microbiological diagnostics also involved blood. In the selected cases, the diagnostic procedures for atypical pathogens, such as *Mycobacterium tuberculosis*, chlamydiae, mycoplasms, *Legionella spp.*, cytomegalovirus, and *Aspergillus spp.*, were collected and sent for tests according to the accepted standards. The drug-susceptibility of cultured bacterial or fundal strains were examined according to the accepted standards of microbiological diagnostic procedures, e.g., E-tests in cases of *Candida* yeast-like fungi using the dilution method in the Vitek 2 system (AST-YS07 chart). The following were accepted as significant for the diagnosing pneumonia: quantitative culture of bacterial or fungal strains collected by mini-BAL or BAL from the bronchial secretion > 10^4 CFU mL^{-1} [19].

RESULTS

Of 1097 patients treated in the ICU during 11 862 patient days, VAP was recognised in 93 and 30% of them died. The patient characteristics of patients are presented in Table 1. The incidence rate was 8.4/100 patients admitted to the ICU. Ventilator-associated pneumonia accounted for 46% of the total number of device-associated healthcare-associated infections DA-HAIs (n = 209). The total number of mechanical ventilation days in the observation period was 8425. The density of VAP/1000 ventilator days and ventilator utilisation ratio [(median/IQR) and (mean ± SD)] were 10.23 (8.4–13.68)/1000 ventilation days and 71.24 ± 8.1, respectively. The analysis of the VAP ratio density/incidence rates and ventilator utilisation ratios during the observation period as well as the number of ventilation days during the observation period are presented in Table 2.

The analysis of the VAP incidence in individual months of observation is presented in Figure 1. The main pathogens of VAP were *Acinetobacter baumannii* (45%), *Pseudomonas aeruginosa* (17%), and *Klebsiella pneumoniae* (14%). The *Acinetobacter baumannii* strains showed resistance to carbapenems in 94%/100% and to amikacin in 94%/100% in 2013 and 2014, respectively, as well as 100% susceptibility to colistin. The *Pseudomonas aeruginosa* strains were found resistant to the following: carbapenems in 43%/0%, amikacin in 43%/0%, ceftazidime in 57%/20%, ciprofloxacin in 57%/40%, and piperacillin with tazobactam in 15%/20%, in 2013 and 2014, respectively. All strains of *Klebsiella pneumoniae* demonstrated extended-spectrum beta-lactamase (ESBL) resistance and 100% susceptibility to carbapenems and amikacin. The aetiological factors of VAP are listed in Figure 2.

The comparative analysis of the VAP incidence in the ICU in various periods and results of international reports are presented in Table 3.

DISCUSSION

For the proper registration of DA-HAIs, the principles of recognition and diagnosis can be standardised and similar epidemiological terms can be used. With the presented programme of infection control, according to the INICC criteria, strictly defined definitions of pneumonia were applied with consideration for the diagnostic difficulties of VAP in patients treated in the ICU who have respiratory failure of various aetiologies. In our study, the VAP rate/1000 ventilator days was slightly lower compared to the results reported in another study that used similar criteria [23]. The use of various diagnostic methods according to the CDC or American College of Chest Physicians (ACCP) recommendations, even at a comparable number of positive bronchial secretion cultures (88% vs. 92%) contributed to significant differences in the diagnoses of infection incidence — 1.2 vs. 8.5/1000 ventilation days [7]. The application of liberal or more restrictive definitions, considering the deterioration of ventilation parameters, in a study of 8123 patients showed substantial differences in the frequency of infections (26.3 vs. 12.0 vs. 8.4 vs. 0.2/1000 ventilation days) in the same population of patients as well as a significant increase in the hospital mortality in the group diagnosed restrictively (OR 6.1 vs. 1.9) [23]. Patients with diagnosed "possible VAP" and deteriorating respiratory parameters (similar to the definition of probable VAP) as well as purulent bronchial secretions that were only evaluated microscopically [20] were not included in our study.

For diagnosing VAP, the recommended options include quantitative microbiological diagnostic methods of bronchial secretion (used by the authors), both BAL and mini-BAL, and qualitative assessment of tracheal secretions sampled

Table 1. Patient characteristics

Year	2012	2013	2014 (01–06)	TOTAL
Number of hospitalised patients	495	397	205	1097
Number of patient days	5327	4445	2090	11862
Females	193 (39%)	151 (38%)	77 (38%)	421 (38%)
Males	302 (61%)	246 (62%)	128 (62%)	676 (62%)
Surgical patients	396 (80%)	330 (83%)	125 (61%)	851 (78%)
Non-surgical patients	99 (20%)	67 (17%)	80 (39%)	246 (22%)
Patients with severe sepsis or septic shock	183 (37%)	184 (46%)	102 (50%)	469 (43%)

Table 2. Analysis of the density/incidence and mortality in VAP patients, utilisation of catheters and numbers of ventilation days. Data are presented as numerical values, percentages, median (IQR) and mean ± SD

	2012	2013	2014 (01–06)	TOTAL
Rate of VAP/1000 ventilator days	11.15 (9.58–13.68)	9.34 (8.4–11.34)	10.22 (9.6–11.69)	10.23 (8.4–13.68)
Number of ventilation days	3687	3208	1530	8425
Number of patients with VAP	43	33	17	93
Ventilator utilisation ratio (%)	68.69 ± 9.1	71.88 ± 5.4	73.14 ± 9.74	71.24 ± 8.1
Incidence rate of VAP/100 admitted to ICU	8.69	8.31	8.29	8.48
Number of deaths	11 (26%)	12 (36%)	5 (29%)	28 (30%)

VAP — ventilator-associated pneumonia; SD — standard deviation; ICU — intensive care unit

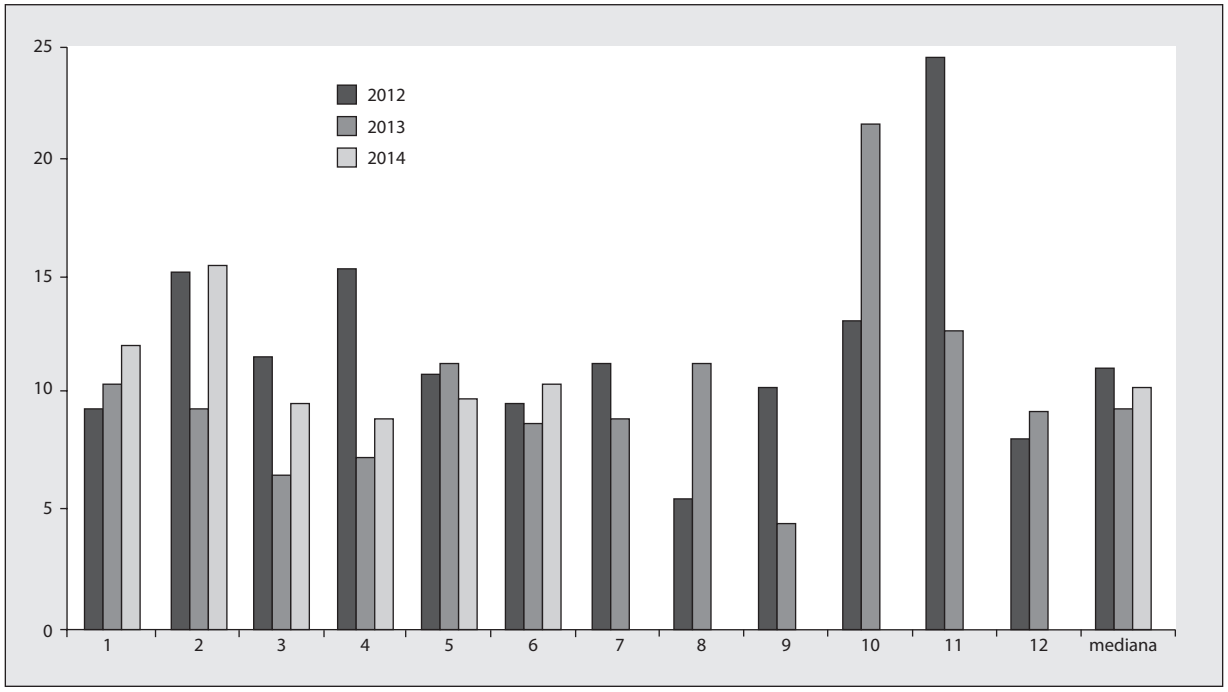

Figure 1. Analysis of ventilator-associated pneumonia (VAP) density in the individual months of observation

from the endotracheal aspirate (ETA). In one of the studies, the patients diagnosed by BAL had a lower number of confirmed VAP (6.1 vs. 25.5/1000 ventilation days) compared to those diagnosed with non-protective aspiration, which was characterised by 90% sensitivity and only 14% specificity [24]. The literature data indicate that VAP prophylaxis, in the form of the ventilation bundle, can result in notable effects, i.e., decreased incidence [3, 15, 23, 25]; however, studies confirming the lack of infections of this type in a given period are sparse [17, 26]. Moreover, the complete elimination of VAP has been questioned [27]. The first data regarding respiratory tract infections in individuals treated with a ventilator,

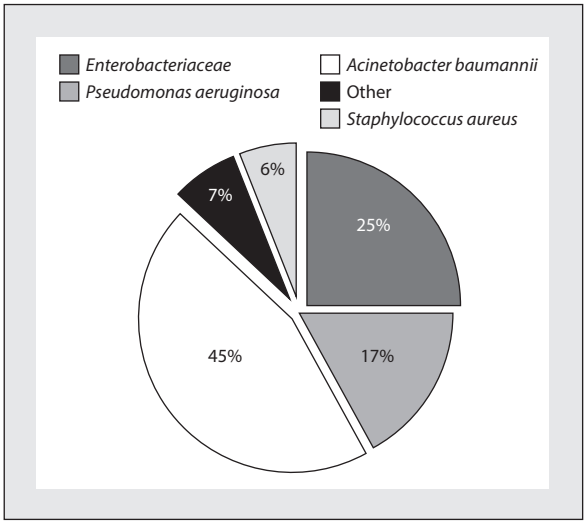

Figure 2. Aetiological factors of ventilator-associated pneumonia (VAP)

acquired in our ICU, come from the doctoral dissertation of the first author and focused on monitoring the changes in bacterial flora and hospital-acquired injections in the years 1995–1996. Respiratory tract infections were the most common clinical form of ICU-acquired infections, which were diagnosed in 38% and 31% of patients, whereas hospital ICU-acquired infections were diagnosed in 72% and 68% of the total number of the treated patients in 1995 and 1996, respectively [28]. The first registry of hospital infections in the authors' unit on monitoring infections using medical devices demonstrated that the ventilator utilisation rate was 70% with a VAP incidence of 29.34/1000 ventilation days [29].

Because of the formation of a team responsible for infection surveillance and introduction of DA-HAIs in the ICU-HELICS system on the initiative of the National Institute of Public Health, we were able to determine that the level of VAP incidence in 2007 over the 12-month observation period was 16 (18.6–11.7) /1000 ventilation days and that ventilation was used in 83% of the patients [21]. Multiple monitoring of DA-HAIs, performed by the same team within the INICC project, indicates that infection surveillance and institution of preventive procedures can result in infection reduction. With respect to VAP, its incidence decreased by 38% compared to the first published results of surveillance according to the INICC [22]. Although infection surveillance over many years showed a decreasing tendency in VAP density, the study over the 2.5 year periods did not reveal further decreases in the ventilator-associated pneumonia incidence. The control of infections, at the basic level, is currently insufficient and requires in-depth analysis of risk factors, preventive management at the ICU level, the introduction of new preventive measures by nursing personnel

Table 3. VAP density in our selected and international studies. Data are presented as a median (IQR) or mean (95%CI)

Study (years of surveillance)	Rate of VAP/1000 ventilator days
ICU, Wrocław (2012–06.2014)	10.23 (8.4–13.68)
ICU, Wrocław (2007) [21]	16 (11.7–18.6)
ICU, Wrocław (2007–2010) [22]	18.2 (15.5–21.6)
INICC report (2007–2012) [18]	16.5 (16.1–16.8)*
NHSN report (2012) [17]	1.1 (1.0–1.2)*

*data from medical/surgical intensive care units; VAP — ventilator-associated pneumonia; ICU — intensive care unit; CI — confidence interval; INICC — International Nosocomial Infection Control Consortium; NHSN — National Healthcare Safety Network

(nurse-to-patient ratio), education, staff motivation, practical/physical availability of preventive measures and more thorough observations of the trend.

The rate of VAP was decreased by 7.97/1000 ventilator days compared to that in our centre in the years 2007–2010 [22] and decreased by 6.27/1000 ventilation days compared to developing countries [18], while it was still higher than the 1.1 (1.0–1.2) in the American NHSN report of 2012 [17]. The most common aetiological factors of VAP, without considering the division in early and late VAP, include *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Klebsiella pneumoniae* [17]. The aetiological factors of VAP in our study, in which multiresistant Gram-negative bacteria predominate, do not differ from the study findings in other Polish hospitals [8,30].

The study limitations include a short observation period, lack of monitoring of the risk factors, lack of assessment of the "ventilation bundle" use and result from our choice of confining the study to monitoring of ventilator-associated pneumonia at the basic level.

In conclusion, the incidence of VAP in the observational period was lower than that in our previous publications and in the INICC report even though it was tenfold higher than in the NHSN/CDC report. As the incidence of ventilator-associated pneumonia has remained at a similar high level over the 2.5-year observation period, the causes should be elucidated and repair management promptly instituted.

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References:

1. *Rewa O, Muscedere J:* Ventilator-associated pneumonia: update on etiology, prevention,and management. Curr Infect Dis Rep 2011; 13: 287–295; doi: 10.1007/s11908-011-0177-9.
2. *Chastre J, Fagon JY:* Ventilator-associated pneumonia. Am J Crit Care Med 2002; 165: 867–903.
3. *Lambert M-L, Palomar M, Agodi A et al.:* Prevention of ventilator-associated pneumonia in intensive care units: an international online survey. Antimicrob Resist Infect Control 2013; 2: 9; doi: 10.1186/2047-2994-2-9.
4. Kollef MH, Hamilton CW, Ernst FR. Economic impact of ventilator-associated pneumonia in large matched cohort. Infect Control Hosp Epidemiol 2012; 33: 250–256; doi: 10.1086/664049.
5. *Safdar N, Dezfulian C, Collard HR et al.:* Clinical and economic consequences of ventilator-associated pneumonia: a systematic review. Crit Care Med 2005; 33: 2184–2193.
6. *Rello J, Ollendorf DA, Osler G et al.:* Epidemiology and outcomes of ventilator associated pneumonia in a large US database. Chest 2002; 122: 2115-–2121.
7. *Skrupky LP, McConnell K, Dallas J et al.:* A comparison of ventilator-associated pneumonia rates as identified according to the National Healthcare Safety Network and American College of Chest Physicians criteria. Crit Care Med 2012; 40: 281–284; doi: 10.1097/CCM.0b013e31822d7913.
8. Rutkowska K, Przybyła M, Misiołek H. Healthcare associated infection in the newly-opened intensive care unit. Anaesthesiol Intensive Ther 2013; 45: 62–66. doi: 10.5603/AIT.2013.0014.
9. Wałaszek M, Wolak Z, Dobroś W. Zakażenia szpitalne u pacjentów hospitalizowanych w latach 2005–2011. Szpital Wojewódzki im. Św. Lukasza w Tarnowie. Przegl Epidemiol 2012; 66: 617–621.
10. *Blot S, Koulenti D, Dimopoulos G et al.:* Prevalence, risk factors, and mortality for ventilator-associated pneumonia in middle-aged, old, and very old critically ill patients. Crit Care Med 2014; 42: 601–609; doi: 10.1097/01.ccm.0000435665.07446.50.
11. *Coppadoro A, Bittner E, Berra L:* Novel prevention strategies for ventilator-associated pneumonia Crit Care 2012; 16: 210; doi: 10.1186/cc11225.
12. *Bekaert M, Timsit JF, Vansteelandt S et al.:* Attributable mortality of ventilator associated pneumonia: a reappraisal using causal analysis. Am J Resp Crit Care Med 2011; 184: 1133–1139; doi: 10.1164/rccm.201105-0867OC.
13. *Berwick DM, Calkins DR, McCannon CJ et al.:* The 100000 Lives Campaign: setting a goal and deadline or improving health care quality. JAMA 2006; 295: 324–327.
14. Institute for Healthcare Improvement. http: //www.ihi.org/knowledge/Pages/Changes/ImplementtheVentilatorBandle.aspx. Published 2013; 28.09.2013.
15. *Rosenthal VD, Rodrigues C, Alvares-Moreno C et al.:* Effectiveness of a multidimensional approach for prevention of ventilator associated pneumonia in adult intensive care units from 14 developing countries of four continents : findings of the International Nosocomial Infection Control Consortium. Crit Care Med 2012; 40: 3121–3128; doi: 10.1097/CCM.0b013e3182657916.
16. *Berenholtz SM, Pham JC, Thompson DA et al.:* Collaborative cohort study of an intervention to reduce ventilator associated pneumonia in intensive care unit. Infect Control Hosp Epidemiol 2011; 32: 305–314; doi: 10.1086/658938.
17. *Dudeck MA, Weiner LM, Allen-Bridson K at al.:* National Healthcare Safety Network (NHSN) report, data summary for 2012, Device-associated module. Am J Infect Control 2013; 41: 1148–1166; doi: 10.1016/j.ajic.2013.09.002.
18. *Rosenthal VD, Maki DG, Metha Y et al.:* International Nosocomial Infection Control Consortium report, data summary of 43 countries for 2007–2012. Device-associated module. Am J Infect Control 2014; 42: 942–956; doi: 10.1016/j.ajic.2014.05.029.
19. *Horan TC, Andrus M, Dudeck MA et al.:* CDC/NHSN surveillance definition of healthcare-associated infection and criteria for specific types of infections in acute care setting Am J Infect Control 2008; 36: 309–332; doi: 10.1016/j.ajic.2008.03.002.
20. CDC/NHSN Surveillance Definition of Healthcare-Associated Infection and Criteria for Specific Types of Infections in Acute Care Setting. January 2013 avaliable: www.INICC.org
21. *Duszyńska W, Barteczko-Grajek B, Kübler A:* Doświadczenia własne z rejestracją zakażeń oddziałowych w systemie HELICS. Anestezjol Intens Ter 2008; 40: 17–21.
22. *Kübler A, Duszyńska W, Rosenthal VD et al.:* Device-associated infection rates and extra length of stay in an intensive care unit of a university hospital in Wroclaw, Poland: International Nosocomial Infection Control Consortium's (INICC) findings. J Crit Care 2012; 27, 105: e5–10; doi: 10.1016/j.jcrc.2011.05.018.
23. *Klompas M, Magill S, Robiscek A et al.:* for the CDC Prevention Epicenters Program. Objective surveillance definitions for ventilator-associated pneumonia. Crit Care Med 2012; 40: 1–8; doi: 10.1097/CCM.0b013e318260c6d9.
24. *Morris AC, Kefala K, Simpson AJ et al.:* Evaluation of the effect of diagnostic methodology on the reported incidence of ventilator-associated pneumonia. Thorax 2009; 64: 516–522; doi: 10.1136/thx.2008.110239.
25. *Al-Thagafy MS, El-Saed A, Arabi YM et al.:* Association of compliance of ventilator bundle with incidence of ventilator-associated pneumonia and ventilator utilization among critical patients over 4 years. Ann Thorac Med 2014; 9: 221–226; doi: 10.4103/1817-1737.140132.
26. *Sundar KM, Nielsen D, Sperry P et al.:* Comparison of ventilator-associated pneumonia (VAP) between different ICUs: implications of zero VAP rate. J Crit Care 2012; 27: 26–32; doi: 10.1016/j.jcrc.2011.05.019.
27. *Klompas M:* Ventilator associated pneumonia: is zero possible? Clin Infect Dis 2010; 51: 1123–1126; doi: 10.1086/656738.
28. *Duszyńska W:* Analiza zmian flory bakteryjnej u chorych leczonych w oddziałach intensywnej terapii. Praca doktorska, Wrocław 1998.
29. *Kübler A, Lysenko L, Zamirowska A et al.:* Wyniki rejestru zakażeń szpitalnych w oddziałach intensywnej terapii dla dorosłych Kliniki Anestezjologii I Intensywnej Terapii AM we Wrocławiu w 2002 r. Zakażenia 2005; 1: 71–74.
30. *Różańska A, Wójkowska-Mach J, Bulanda M et al.:* Surveillance of hospital acquired pneumonia in Polish hospitals. Przegl Epidemiol 2008; 63: 119–123.

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