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Pulmonary oedema, pneumonia and mortality in submersion victims; a retrospective study in 125 patients

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Abstract Objective: The identification of risk factors contributing to the development of pulmonary oedema, pneumonia and late mortality in submersion victims.

Design: A retrospective study of 125 submersion victims.

Setting: The medical intensive care unit in a university hospital.

Methods: Baseline examination on admission consisted of history, physical examination, arterial blood gas analysis and a chest radiograph. Patients were then classified into four groups: class I, baseline examination negative; class II, baseline examination positive, but mechanical ventilation not needed on admission; class III, mechanical ventilation required on admission; class IV, patients suffering from cardiopulmonary arrest. All patients who were not successfully resuscitated or who had expired within 24 h after admission were excluded for determination of the risk of pulmonary oedema and pneumonia.

Results: Class I patients did not develop pulmonary complications; neither pulmonary oedema nor pneumonia occurred in this group. In the remaining classes the incidence of pulmonary oedema was 72% and that of pneumonia, 14.7%.

Stepwise logistic regression showed that pulmonary oedema was related to the type of water (seawater, ditch water, swimming pool) victims were submerged in and to the neurological state both at the time of rescue and on admission. The development of pneumonia was related to the use of mechanical ventilation (the risk was 52%). Pneumonia was not related to neurological state at the time of rescue or on admission, to body temperature on admission, to the prophylactic administration of antibiotics or to the use of corticosteroids. Mortality was high in class IV patients, but low in all other patients. Early mortality was 18.4% while late mortality was 5.6%.

Conclusions: There is no need to hospitalise submersion victims when there are no signs or symptoms of aspiration upon arrival in the emergency room. All other patients should be admitted to an intensive care unit. The risk of pneumonia is high when mechanical ventilation is necessary. Mortality is high in patients with circulatory arrest on admission, but low in all other patients.

Key words Drowning · Near drowning · Pneumonia · Pulmonary oedema · Mortality · Artificial respiration

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Introduction

Leiden, a well-known Dutch city, is on the river Rhine. It is surrounded by many brooks, classic Dutch ditches and lakes. It lies approximately ten miles from the North Sea. Because of Leiden's location there are a great many submersion incidents. Every year roughly 12 submersion victims are admitted to the Medical Intensive Care Unit of the University Hospital, which has a concomitant regional function for resuscitation and intensive care. This patient population therefore includes the more severe cases.

Pulmonary oedema and pneumonia are the two major complications that threaten submersion victims [1]. Pulmonary oedema is often present on admission to the hospital but can have a delayed onset. It can develop in the first 24 h after submersion, possibly even in initially asymptomatic persons. Admission to a hospital and observation of all patients with a history of submersion, whether symptomatic or not, is therefore commonly recommended. However, clinical studies supporting this view are scarce. There are no studies available that correlate the development of pneumonia to the type of water aspirated or to the clinical state of the patient at rescue or admission. We therefore conducted a retrospective study of the submersion victims admitted to the intensive and respiratory care unit during the 10-year period from 1 January 1971 to 31 December 1988. We tried to answer the following questions: is it possible to screen successfully at the time of admission to identify those patients most susceptible to the development of pulmonary oedema or pneumonia; and which category of patients has a reduced or small survival rate?

Methods

We retrospectively analysed the data of 125 consecutive victims of submersion who were admitted to the Medical Intensive Care Unit (MICU) of the University Hospital Leiden, The Netherlands, between 1 January 1979 and 31 December 1988. These patients were traced from computerised surveys of the central hospital records as well as from the detailed annual reports and administrative data of the MICU. Patient records were reviewed for age, sex, water type, resuscitation data, signs of pulmonary derangement on admission, classification of neurological state (both at the rescue site and on admission) according to Modell [2], central body temperature on admission, the need for and duration of mechanical ventilation, the occurrence of pneumonia, bacteriologic data, prophylactic treatment with antibiotics and/or steroids, duration of hospital stay, and final outcome. Dose of steroids was calculated as dose-equivalent of prednisolone/kg estimated body weight. All available chest radiographs were examined for signs of pulmonary oedema and for the presence of infiltrates by a panel of two radiologists, who had no knowledge of the clinical data.

Patients were identified upon admission as class I (signs of aspiration absent: i.e. no coughing, no rales on auscultation, normal blood gases, normal chest radiograph), class II (signs of aspiration present, but at the time of admission no need for mechanical ventilation), class III (mechanical ventilation required at admission) or class IV (patients presenting in cardiopulmonary arrest). This classification is a modification of Simcock's scheme [3]. Simcock did not use the blood gas analysis results and chest radiograph for classification purposes.

Pulmonary oedema was defined as an abnormal extravascular accumulation of liquid in the pulmonary tissues and air spaces. It was diagnosed when alveolar or interstitial infiltrates were present on the chest radiograph.

In order to diagnose pneumonia it was obligatory to have a pulmonary infiltrate that persisted for at least 24 h on the chest radiograph [4-8]. In addition, these patients fulfilled at least three of the following four criteria: (a) acute pyrexia (temperatures exceeding 38.0°C), (b) new leucocytosis (leucocytes $> 10.0 \times 10^9/l$), (c) purulence of sputum (> 10 leucocytes/high power field in gram stain) or (d) pathogenic micro-organisms cultured from sputum, or seen in gram stain. The diagnosis of pneumonia was also made in cases of a persistent pulmonary infiltrate on the chest radiograph and a positive sputum culture and a positive blood or pleural fluid culture contaminated with the same micro-organism. Finally the diagnosis could be made at post mortem.

Owing to the retrospective nature of this study not all data were available from all patients. Where appropriate the actual number of patients studied and the statistical method used are quoted. For determination of the risk of pneumonia and pulmonary oedema, stepwise logistic regression analysis using SPSS-PC v 5.01 software was used. All patients who were beyond resuscitation and all patients who died within 24 h of admission were excluded from this analysis.

Results

During the study period 125 submersion victims were admitted, to the MICU. (See Table 1 for characteristics of all 125 patients.) No significant age difference or significant central body temperature differences were observed among any of the four classes on admission.

Of the 27 patients already in cardiac arrest on admission (Class IV) 19 could not be resuscitated and another four died within 12 h after admission. There was no age difference between these 23 resuscitation failures and the 102 other patients (Mann Whitney U test). Body temperature levels on admission were lower in these resuscitation failure patients than in the other patients ($n = 116$; data missing for 9 patients; Mann Whitney U Wilcoxon Rank Sum W test: $P < 0.01$).

The incidence of complications in the remaining 102 patients is shown in Table 2. The incidence of pulmonary oedema was 43% (44/102 patients). In all these 44 patients it was already present upon admission. Progression of pulmonary oedema on chest radiographs was obvious of six patients. Deterioration of pulmonary function necessitating the employment of

Table 1 Patient characteristics

Class	I	II	III	IV-A ^a	IV-B ^a
Total number	41	42	15	4	23
Male	27	30	11	3	19
Age (y)					
Mean	29.1	30.9	36.5	23.2	31.4
Minimum	2	1	2	1	2
Maximum	85	91	78	53	80
Water type					
Ditch water	34	25	10	3	14
Swimming pool	5	9	2	1	7
Sea	2	8	3	–	2
Temperature on admission (°C)					
Number of patients	40	41	14	4	17
Mean	35.1	34.8	33.6	35.4	32.2

^a The Class IV patients who were successfully resuscitated, are tabulated as IV-A, and the resuscitation failures, as class IV-B

Table 2 Number of patients (non-survivors)

Class	I	II	III	IV-A	Total
Total	41 (0)	42 (3 ^a)	15 (2)	4 (2)	102 (7)
Pulmonary oedema	0 (0)	27 (2)	13 (1)	4 (2)	44 (5)
Pneumonia	0 (0)	8 (2)	5 (1)	2 (1)	15 (4)
Mechanical ventilation	0 (0)	7 (2)	15 (2)	3 ^b (2)	25 (6)

^a After discharge one patient died due to a cerebrovascular accident

^b A 53-year-old man with ventricular fibrillation did not need mechanical ventilation after successful defibrillation

Table 3 Risk factors associated with pulmonary oedema as obtained by stepwise logistic regression analysis ($n = 90$)

Risk factor	Patients at risk	Incidence of pulmonary oedema	95% confidence interval	P-value
<i>Neurological state at rescue</i>				
Comatose	39	66.7%	49.8–80.9%	< 0.01
Awake or blunted	51	21.6%	11.3–35.3%	
Relative risk		3.1	1.8–5.5	
<i>Neurological state on admission</i>				
Blunted or comatose	30	63.3%	43.9–80.1%	0.02
Awake	60	30%	18.8–43.2%	
Relative risk		2.1	1.3–3.4	
<i>Water type</i>				
Sea or swimming pool	25	64%	42.5–82%	< 0.01
Ditch water	65	32.3%	21.2–32.3%	
Relative risk		2.0	1.3–3.1	

mechanical ventilation, occurred in 7 out of 42 class II patients, mostly within a few hours of admission. In two of these class II patients the progression of pulmonary oedema was apparent. In class I patients, pulmonary oedema did not occur (risk 0%, 95% confidence interval 0–7.6%). In other patients the incidence

of pulmonary oedema was 72%. In a stepwise logistic regression analysis ($n = 90$) it was found that the best predictors for pulmonary oedema were type of water, comatose state at rescue, and not being awake on admission (Table 3). Body temperature on admission had no additional predictive value.

Table 4 Bacteriology (results of cultures in 15 submersion victims with pneumonia)

	Sputum	Blood	Pleural fluid
<i>Escherichia coli</i>	7	1	2
<i>Aeromonas</i> spp.	4	1	1
<i>Klebsiella</i> spp.	3	1	1
<i>Pseudomonas</i> spp.	5	1	1
<i>Haemophilus influenzae</i>	3		
<i>Staphylococcus aureus</i>	3		1
<i>Streptococcus pneumoniae</i>	1		
<i>Branhamella</i> spp.	1		
<i>Candida</i> species	1		
<i>Bacteroides</i> spp.	1		
<i>Streptococcus</i> spp.	1	1	1
<i>Clostridium</i> spp.			1
<i>Peptostreptococcus</i>			1
<i>Propionibacterium</i> spp.			1

The incidence of pneumonia was 14.7% (15/102 patients). All 15 patients with pneumonia fulfilled the criterion of an abnormal chest radiograph. In 13 patients a new lesion developed, most frequently following initial clearing of the oedema. In one case, a pleural effusion was diagnosed, which later proved to be empyema. In another case there was persistent diffuse bilateral pulmonary oedema. Five other patients developed a pulmonary infiltrate, but these patients did not meet the criteria required for the diagnosis of pneumonia.

Fever and leucocytosis were almost invariably present in the patients with pneumonia, but were also found in a substantial number of the other patients. In 13 of the 15 pneumonia patients sputum was examined. Analysis showed, to our surprise, more than 10 leucocytes, high-power field in the gram stain in only eight patients, but in every case pathogenic microorganisms were found. In two patients sputum was not obtained for analysis. The first case was in a 3-year-old boy who, could not cooperate but responded favourably to antibiotic treatment. In the second case sputum analysis was not performed because treatment was withdrawn (in this case a post-mortem diagnosis of pneumonia was made). Three cases were complicated by a pleural

empyema, and five cases by bacteraemia. Gram negatives were the predominant microorganisms (Table 4). *Aeromonas* species were cultured from four patients. Anaerobes did play a role in a substantial proportion of our patients.

Stepwise logistic regression analysis ($n = 88$) showed that the relation of pneumonia to mechanical ventilation was highly significant (Table 5). Pneumonia developed in 11 patients while on the ventilator and in one patient after weaning. Pneumonia was not related to classification on admission, water type, body temperature on admission, neurological state at rescue or on admission, use of corticosteroids or prophylactic use of antibiotics. Antibiotic prophylaxis was given to 45 patients, corticosteroids to 44 patients. The mean loading dose of corticosteroids was 10.6 mg prednisolone/kg of body weight, and the mean maintenance dose was 2.5 mg prednisolone/kg of body weight per day. Mean duration of corticosteroid use was 1.8 days. Corticosteroids were given less often in the later years of this study. The data did not suggest a beneficial effect of corticosteroids or prophylactic antibiotics on duration of mechanical ventilation, length of stay in MICU or length of stay in the hospital.

Early mortality was 18.4% (23 out of 125 submersion victims). Of the remaining 102 patients, 5 died in the hospital: three deaths were attributed to pneumonia, and one to head injury. Another patient suffered from a persistent vegetative state. After discharge two patients died: one of a cerebrovascular accident, which caused the submersion incident and one of a persistent vegetative state. Thus, late mortality was 5.6% (7/125) and total mortality, 24% (30/125) (Table 2). The incidence of late mortality was too low to perform a stepwise logistic regression analysis.

Discussion

Pulmonary oedema and pneumonia are two major complications that threaten submersion victims [1]. Pulmonary oedema is often present on hospital admission, but can also have a delayed onset. It can develop in the first 24 h after a submersion incident, possibly even in initially asymptomatic persons. Admission to

Table 5 Risk factors associated with pneumonia as obtained by stepwise logistic regression analysis ($n = 88$)

Risk factor	Patients at risk	Incidence of pneumonia	95% confidence interval	<i>P</i> -value
Needing mechanical ventilation	21	52%	32–72%	
Not needing mechanical ventilation	67	3%	1–11%	
Relative risk		17.3	4.2–72.9	< 0.001

a hospital and observation of all patients with a history of submersion, whether symptomatic or not, is therefore commonly recommended. However, none of our class I patients developed pulmonary oedema (secondary drowning) or pneumonia. The calculated risk of pulmonary oedema in Class I patients was 0–7.6%. Secondary drowning represents evolution of lung injury caused by the submersion incident. Our findings indicate that on admission there are always some signs of that injury present unless aspiration did not occur, in which case secondary problems will not develop. When the reports that recommend the observation of every submersion victim are carefully analysed, it indeed seems exceptional that an asymptomatic patient with a normal chest radiograph and a normal blood gas analysis will develop severe pulmonary problems [3,9,10,11]. The dilemma of whether to hospitalise asymptomatic patients or send them home was also addressed by Pratt [12]. He studied 52 patients who had suffered a submersion incident in seawater, 31 of whom refused referral to a hospital. He obtained follow-up information by telephone from 26 of them. None of them needed medical care later, and all had returned to a normal state of health. In another study [13] a close correlation was found between clinical findings and the findings on the first chest radiograph, taken immediately after rescue. Prognosis was good when the first chest radiograph did not demonstrate any lesions. These reports support the view that patients without symptoms and signs of aspiration on baseline examination are at low risk of pulmonary oedema or pneumonia. We agree, therefore, with Stewart [14] that an asymptomatic patient with a normal chest radiograph and a normal blood gas analysis can be sent home after a thorough examination at the emergency department, unless concomitant problems necessitate hospitalisation.

The risk of pulmonary oedema was further analysed using stepwise logistic regression, entering variables that could be obtained on admission. Variables included were: water type, neurological state at rescue and on admission, and body temperature on admission. The use of corticosteroids was not included, because in all our cases with pulmonary oedema, this complication was already present on admission. There was an increased risk of pulmonary oedema following submersion in seawater or swimming pool water. This might be a selection bias, because extremely few asymptomatic victims of swimming pool submersion were brought to our hospital. Body temperature on admission was not related to pulmonary oedema. Patients who were comatose at rescue and patients who were not awake on admission more often had pulmonary

oedema, probably reflecting the greater risk of aspiration when consciousness is impaired.

In our series the incidence of pneumonia was 14.7%. Pneumonia was closely related to mechanical ventilation ($P < 0.001$). The calculated risk of pneumonia was 52% (95% confidence interval 32–72%) when mechanical ventilation was needed, but in the other cases only 3% were at risk (95% confidence interval 1–11%).

The precise diagnosis of pneumonia in patients receiving mechanical ventilation is often difficult. These patients have a serious underlying disease, increased oropharyngeal colonization with hospital flora, and numerous reasons for elevated body temperature and leucocytosis. Purulent sputum may follow intubation or leakage of oropharyngeal secretions around the artificial airway. Furthermore, chest radiographic changes consistent with pneumonia may be caused by pulmonary oedema, pulmonary infarction, atelectasis or ARDS. We used an established criteria for the diagnosis of pneumonia. Although we cannot exclude the possibility that pneumonia was incorrectly diagnosed, it is unlikely that true cases of pneumonia would have been missed by these criteria.

That pneumonia was related to the necessity of mechanical ventilation underscores the fact that the aspiration of sufficient quantities of water or gastric contents leading to severe lung damage is necessary for the development of pneumonia. Logistic regression analysis showed that other variables related to aspiration, such as classification on admission and neurological state at rescue or on admission, gave no additional information useful for the prediction of pneumonia.

We were surprised that pneumonia was not related to water type. In other studies [3,9,15], where good-quality ocean or swimming pool water was involved, the incidence of pneumonia was very low, contrasting remarkably with Dutch studies on submersion [16,17] and also with some studies from other countries [18]. Since river deltas consisting of wide, shallow, sluggish rivers and also brooks, lakes and coastal waters are heavily polluted and harbour a variety of aquatic birds who add to the pollution by their excreta, we reasoned that the degree of contamination by micro-organisms and particulate matter must be an additional risk factor for the development of pneumonia. Therefore, we expected to find a higher incidence of pneumonia in victims rescued from sea or ditch water than in victims rescued from swimming pools. However, only one victim pulled from a swimming pool developed pneumonia: a six-year-old boy, whose pneumonia was caused by endogenous flora. Of the other victims extracted from swimming pools only two needed mechanical

ventilation. Therefore, only the relation of pneumonia to mechanical ventilation was significant.

Prophylactic use of antibiotics did not prove beneficial in our study. However, owing to the retrospective nature of this study and the uncontrolled arbitrary use of antibiotics no definite conclusion can be drawn. Nevertheless, we believe that the prophylactic use of antibiotics should be restricted to victims who need mechanical ventilation and who have probably aspirated severely contaminated water. In other situations antibiotics should be withheld unless specific evidence for infection develops [1]. Pending the results of cultures, antibiotics should cover endogenous as well as exogenous flora. The bacteriology of both the marine and the freshwater environment has been described by Auerbach [19,20] and by Sims [21]. Gram-negative micro-organisms predominate, but anaerobes and staphylococci are also found. In submersion victims pneumonia is caused by these bacteria, and also by common endogenous pathogens such as *Haemophilus influenzae* and *Streptococcus pneumoniae*. [16,18,21–25]. There are some reports on fungal infections in near-drowning [26–29]. In our series (Table 3) gram-negative micro-organisms were predominant. There were 4 cases caused by *aeromonas* spp., ubiquitous inhabitants of water [30]. Bronchial secretions were not routinely cultured for anaerobes, but these micro-organisms undoubtedly played an important role, as can be seen in our patients with empyema. Infection was polymicrobial in most patients.

Hypothermia is described as a risk factor for infections [31]. In this study no relationship was observed between body temperature on admission and incidence of pneumonia, probably because there were only a few cases in this study of very low body temperatures.

Corticosteroid therapy was once suggested for use after near-drowning to influence the course of pulmonary oedema, but its use is now discouraged because it impairs the response to infection [1]. However, we did not find a negative effect of corticosteroids on the occurrence of pneumonia.

Mortality was 24% (30/125) in our series. For patients with circulatory arrest on arrival in the emer-

gency department the prognosis was poor, which is consistent with the findings of other studies [3,9,10,18,32,33]. If a patient arrived at hospital with intact circulation or if resuscitation had been successful, mortality was low. Because of the low mortality rate we were not able to determine what factors predicted the outcome in this group of patients. A disease severity scoring system, such as APACHE II [34], was not used in the MICU until 1990. Use of such a system would have put our mortality figures into a better perspective. However, mortality was low in patients who were not in circulatory arrest on admission. The classification scheme proved useful, but can be further simplified: patients without signs of aspiration on admission (class I) have a very low risk of pulmonary oedema, pneumonia and mortality and can be sent home unless another condition mandates hospitalisation. Patients with signs of aspiration but with an intact circulation (class II and III) are at high risk of pulmonary oedema and should be admitted to an intensive care unit. In this respect class II and class III patients can be grouped into one class. Patients with circulatory arrest on admission are a distinct group with a very high mortality rate.

Conclusions

Every submersion victim should be taken to hospital for examination. This must include history, a physical examination, an arterial blood gas analysis and a chest radiograph. Patients can be classified according to these results. There is no need for hospitalisation when this examination is negative (class I). Damage to the alveolocapillary membrane, resulting in the development of pulmonary oedema, will always produce signs and symptoms apparent on admission. Therefore, patients with signs of aspiration on admission are at risk for the development of pulmonary oedema. In particular, patients requiring mechanical ventilation are at risk of pneumonia. Patients presenting with circulatory arrest on admission show a high mortality rate, while the mortality rate is low in all other patients.

References

1. Modell JH (1993) Current concepts: drowning. *N Engl J Med* 328: 253–256
2. Modell JH, Graves SA, Kuck EJ (1980) Near-drowning: correlation of consciousness and survival. *Can Anaesth Soc J* 27: 211–215
3. Simcock AD (1986) Treatment of near-drowning—a review of 130 cases. *Anaesthesia* 41: 643–648
4. Andrews CP, Coalson JJ, Smith JD, Johanson WG (1981) Diagnosis of bacterial nosocomial pneumonia in acute, diffuse lung injury. *Chest* 80: 254–258
5. Salata RA, Lederman MM, Shlaes DM, Jacobs MR, Eckstein E, Twardy D et al. (1987) Diagnosis of nosocomial pneumonia in intubated, intensive care unit patients. *Am Rev Respir Dis* 135: 426–432

6. Rosenbaum HT, Thompson WL, Fuller RH (1964) Radiographic pulmonary changes in near-drowning. *Radiology* 83: 306-313
7. Hunter TB, Whitehouse WM (1974) Fresh water near-drowning: radiological aspects. *Radiology* 112: 51-56
8. Wunderlich P, Rupprecht E, Trefftz F, Thomsen H, Burkhardt J (1985) Chest radiographs of near-drowned children. *Pediatr Radiol* 15: 297-299
9. Simcock AD (1989) The resuscitation of submersion victims. *ACP* 2: 293-298
10. Martin TG (1984) Near-drowning and cold water immersion. *Ann Emerg Med* 13: 263-267
11. Model JH, Graves SA, Ketover A (1976) Clinical course of 91 consecutive near-drowning victims. *Chest* 70: 231-238
12. Pratt FD, Haynes BE (1986) Incidence of "secondary drowning" after salt water submersion. *Ann Emerg Med* 15: 1084-1087
13. Schiavon F, Nardini S, Bossi A, Bezegato L (1990) Radiologic picture of the thorax in acute asphyxia caused by near drowning. *Radiol Med* 80: 24-28
14. Stewart RD (1989) Submersion incidents: drowning and near-drowning. In: Auerbach PS, Geehr EC (eds) *Management of wilderness and environmental emergencies*. 2nd edn. Mosby, St Louis, pp 908-932
15. Gonzalez-Rothi RJ (1987) Near drowning: consensus and controversies in pulmonary and cerebral resuscitation. *Heart Lung* 16: 474-482
16. Klein JJ, Dallinga OT, Van Dijk W, De Jager AEJ, Meinesz AF, Snoek WJ, Sluiter HJ (1979) Bijna-verdrinking: een analyse van 60 patiënten. *Ned T Geneesk* 20: 162-167
17. Eggink WF, Bruinink HA (1977) Respiratory distress syndrome caused by near or secondary drowning and treatment by positive pressure ventilation. *Neth J Med* 20: 162-167
18. Oakes DD, Sherck JP, Maloney JR, Charters AC (1982) Prognosis and management of victims of near-drowning. *Trauma* 22: 544-549
19. Auerbach PS, Yajko DM, Nassos PS, Kizer KW, McCosker JE, Geehr EC, Hadley WK (1987) Bacteriology of the marine environment: implications for clinical therapy. *Ann Emerg Med* 16: 643-649
20. Auerbach PS, Yajko DM, Nassos PS, Kizer KW, Morris JA, Hadley WK (1987) Bacteriology of the freshwater environment: implications for clinical therapy. *Ann Emerg Med* 16: 1016-1022
21. Sims JK, Enomoto PI, Frankel RI, Wong LMF (1983) Marine bacteria complicating seawater near-drowning and marine wounds: a hypothesis. *Ann Emerg Med* 12: 212-216
22. Rosenthal SL, Zuger JH, Apollo E (1975) Respiratory colonization with *Pseudomonas putrefaciens* after near-drowning in salt water. *Am J Clin Pathol* 64: 382-384
23. Vernon DD, Banner W Jr, Cantwell GP, Holzman BH, Bolte RG, Dean JM (1990) *Streptococcus pneumoniae* bacteremia associated with near-drowning. *Crit Care Med* 18: 1175-1176
24. Kelly MT, Avery DM (1980) Lactose-positive *Vibrio* in seawater, a cause of pneumonia and septicemia in a drowning victim. *J Clin Microbiol* 11: 278-280
25. Wenger JD, Hollis DG, Weaver RE, Baker CN, Brown GR, Brenner DJ, Broome CV (1989) Infection caused by *Francisella philomiragia* (formerly *Yersinia philomiragia*). A newly recognized human pathogen. *Ann Intern Med* 110: 888-892
26. Fisher JF, Shadowy S, Teabeaut JR, Woodward Y, Michaelis GE, Newman ME, et al. (1982) Near drowning complicated by brain abscess due to *Letrillidium boydii*. *Arch Neurol* 39: 511-513
27. Vieira DF, Van Saene HKF, Miranda DR (1984) Invasive pulmonary aspergillosis after near-drowning. *Intensive Care Med* 10: 203-204
28. Kershaw P, Freeman R, Templeton D, DeGirolami PC, DeGirolami U, Tarsy D, et al. (1990) *Pseudallescheria boydii* brain abscess: association with near-drowning and efficacy of high dose, prolonged miconazole therapy in patients with multiple abscesses. *Medicine* 68: 218-214
29. Dworzack DL, Clark RB, Borkowski WJ, Smith DL, Dykstra M, Pugsley MP, et al. (1989) *Pseudallescheria boydii* brain abscess: association with near-drowning and efficacy of high dose, prolonged miconazole therapy in patients with multiple abscesses. *Medicine* 68: 218-214
30. McGowan JE, Del Rio C (1990) Other gram-negative bacilli. In: Mandell GL, Douglas RG, Bennett JE (eds) *Principles and practice of infectious diseases*, 3rd edn. Churchill Livingstone, New York, 1783-1784
31. Reuler JB (1978) Hypothermia: pathophysiology, clinical settings, and management. *Ann Intern Med* 89: 519-527
32. Nichter MA, Everett PB (1989) Childhood near-drowning: is cardiopulmonary resuscitation always indicated? *Crit Care Med* 17: 993-995
33. Biggart MJ, Bohn DJ (1990) Effect of hypothermia and cardiac arrest on outcome of near-drowning accidents in children. *J Pediatr* 117: 179-183
34. Knaus WA, Draper EA, Wagner DP, Zimmerman JE (1985) APACHE II: a severity of disease classification system. *Crit Care Med* 13: 818-829