Prognostic factors for lower respiratory tract infections after brain-tumor surgery

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Nosocomial infection of the lower respiratory tract is a frequent and serious complication after major operations. A 32% incidence of lower respiratory tract infections was found after brain-tumor surgery in 289 patients, with a 21% incidence of pneumonia. In 186 of these patients (Group A), five factors were identified which were associated with an increased risk of postoperative lower respiratory tract infection. These were: age, tumor type, cardiac insufficiency, preoperative disturbances of consciousness, and preoperative corticosteroid treatment. Based on these factors, a risk score was developed which correlated well with the incidence of infection in this group of patients.

In a second group of patients (Group B), the derived risk score was applied and was found to possess a high degree of validity. As long as patients were intubated postoperatively, their freedom from infection decreased exponentially, with a half-life of 3.5 days.

KEY WORDS - brain neoplasm - dexamethasone - meningioma - pneumonia - respiratory tract infection - endotracheal intubation

Nosocomial infection of the lower respiratory tract is associated with a high mortality rate, in the range of 28% to 58%. It must therefore be regarded as a serious complication in any treatment which increases the risk of this infection. Prophylactic application of physiotherapy and/or drug treatment is only marginally effective.

To decrease the incidence of this complication, it is desirable to identify patients at risk as early as possible: in surgical patients this means prior to surgery. To achieve this, it is necessary to determine preoperatively those factors known to be of prognostic relevance in the development of such postoperative infections. Several indicators for postoperative infections of the lower respiratory tract (PILRT) have been mentioned in the literature: the patient's age, and weight, preexisting pulmonary diseases, smoking habits, alcohol intake pattern, Smoking habits, alcohol intake pattern, diabetes mellitus, and alcohol intake pattern, and/or the cardiovascular system, disturbance of consciousness, the presence of a malignant disease, or paralysis, pretreatment with immunosuppressive drugs, duration of the preoperative stay in the hospital, the patient's socioeconomic status, and the season of admission to the hospital.

The relative importance of these prognostic factors and their modes of interdependence seems to be based on the underlying diseases and their treatment. This effect is not clear in most published data because of the diagnostic heterogeneity of the populations investigated. Furthermore, the incidence of PILRT differs considerably depending on the populations studied. For patients in intensive care the incidence of nosocomial pneumonia ranges between 12% and 49% (Table 1). In the area of neurosurgery, information is available in one homogeneous population reported by Braun, et al. in their study of 66 patients with mechanical ventilation following severe brain injury, 15 (23%)
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developed pneumonia. These results were obtained independent of the extent of corticosteroid treatment. Thiopental treatment increased the risk of infection significantly, and other prognostic factors could not be identified. All other investigations of neurosurgical patients concern nosologically inhomogeneous populations.7,54,55

No specific data regarding PILRT are known for patients operated on for intracranial neoplasms. Therefore, such a population was investigated retrospectively in order to answer three questions: 1) how many patients develop PILRT after brain-tumor surgery? 2) which patients develop PILRT? 3) when does PILRT start in the postoperative course?

Clinical Material and Methods

This retrospective investigation includes 289 consecutive patients who were operated on for intracranial tumors (Table 2). They are subdivided into two groups. Group A included 186 patients operated on up to November 30, 1984. The clinical course of these patients was analyzed to identify relevant risk factors. Twenty-two of the patients had pituitary adenomas and were operated on transphenoidally. Group B included the remaining 103 patients, who were operated on after November 30, 1984, and served as a control group for the correctness of the risk assessment. Thirteen of these patients had a pituitary adenoma and were operated on transphenoidally. The date for separation of the two groups was chosen because the neurosurgical intensive care unit moved to another building on that day. Of the patients undergoing cranial surgery, only those who stayed in the intensive care unit for at least 24 hours were included in the study. Therefore, patients undergoing minor operations (for instance, biopsies) were excluded from the series.

Because it is difficult to classify PILRT according to the presence (pneumonia) or the absence (bronchitis) of parenchymal involvement in the inflammatory process, both forms of PILRT were considered. Thus, PILRT was diagnosed whenever the following criteria were met: 1) colonization of the lower respiratory tract by bacteria or yeasts, validated by bacteriological investigations.7,34,36,55

Table 2

Diagnosis of 289 patients with intracranial tumors, compared to review data collected by Youmans

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Group A</th>
<th>Group B</th>
<th>Youmans' Series*</th>
</tr>
</thead>
<tbody>
<tr>
<td>glioma</td>
<td>34%</td>
<td>43%</td>
<td>42%</td>
</tr>
<tr>
<td>meningoima</td>
<td>23%</td>
<td>23%</td>
<td>17%</td>
</tr>
<tr>
<td>pituitary adenoma</td>
<td>12%</td>
<td>13%</td>
<td>8%</td>
</tr>
<tr>
<td>metastasis</td>
<td>9%</td>
<td>10%</td>
<td>10%</td>
</tr>
<tr>
<td>neurinoma</td>
<td>4%</td>
<td>6%</td>
<td>7%</td>
</tr>
<tr>
<td>angina</td>
<td>4%</td>
<td>3%</td>
<td>3%</td>
</tr>
<tr>
<td>others</td>
<td>14%</td>
<td>2%</td>
<td>13%</td>
</tr>
<tr>
<td>total cases</td>
<td>186</td>
<td>103</td>
<td>25,588</td>
</tr>
</tbody>
</table>

Because it is difficult to classify PILRT according to the presence (pneumonia) or the absence (bronchitis) of parenchymal involvement in the inflammatory process, both forms of PILRT were considered. Thus, PILRT was diagnosed whenever the following criteria were met: 1) colonization of the lower respiratory tract by bacteria or yeasts, validated by bacteriological investigation of bronchial secretions collected by sterile suc-

Table 3

Distribution of diagnosis and preoperative characteristics in 186

Group A patients*

<table>
<thead>
<tr>
<th>Parameters</th>
<th>All Cases</th>
<th>Cases With PILRT</th>
<th>Cases With No PILRT</th>
<th>p Value (chi-square)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1 tumor pathology</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>meningoima</td>
<td>44</td>
<td>23</td>
<td>21</td>
<td>0.01</td>
</tr>
<tr>
<td>glioma</td>
<td>63</td>
<td>20</td>
<td>43</td>
<td>NS</td>
</tr>
<tr>
<td>pituitary adenoma</td>
<td>22</td>
<td>3</td>
<td>19</td>
<td>0.04</td>
</tr>
<tr>
<td>others</td>
<td>57</td>
<td>16</td>
<td>41</td>
<td>NS</td>
</tr>
<tr>
<td>1.2 infratentorial tumor</td>
<td>36</td>
<td>14</td>
<td>22</td>
<td>NS</td>
</tr>
<tr>
<td>1.3 disturbance of consciousness</td>
<td>31</td>
<td>17</td>
<td>14</td>
<td>0.005</td>
</tr>
<tr>
<td>1.4 reduction of drive</td>
<td>16</td>
<td>6</td>
<td>10</td>
<td>NS</td>
</tr>
<tr>
<td>1.5 cerebral seizures</td>
<td>22</td>
<td>10</td>
<td>12</td>
<td>NS</td>
</tr>
<tr>
<td>2.1 cardiac failure</td>
<td>21</td>
<td>13</td>
<td>8</td>
<td>0.002</td>
</tr>
<tr>
<td>2.2 hypertension</td>
<td>22</td>
<td>8</td>
<td>14</td>
<td>NS</td>
</tr>
<tr>
<td>2.3 diabetes mellitus</td>
<td>26</td>
<td>10</td>
<td>16</td>
<td>NS</td>
</tr>
<tr>
<td>2.4 pulmonary disease</td>
<td>35</td>
<td>16</td>
<td>19</td>
<td>NS</td>
</tr>
<tr>
<td>2.5 anemia</td>
<td>31</td>
<td>8</td>
<td>23</td>
<td>NS</td>
</tr>
<tr>
<td>2.6 adiposity</td>
<td>71</td>
<td>22</td>
<td>49</td>
<td>NS</td>
</tr>
<tr>
<td>3.1 corticosteroid therapy</td>
<td>105</td>
<td>43</td>
<td>62</td>
<td>0.01</td>
</tr>
<tr>
<td>3.2 age ≥ 60 yrs</td>
<td>67</td>
<td>30</td>
<td>37</td>
<td>0.02</td>
</tr>
<tr>
<td>3.3 male sex</td>
<td>84</td>
<td>25</td>
<td>59</td>
<td>NS</td>
</tr>
<tr>
<td>totals</td>
<td>186</td>
<td>62</td>
<td>124</td>
<td></td>
</tr>
</tbody>
</table>

* PILRT = postoperative infection of the lower respiratory tract; NS = not significant.
pressure under antihypertensive therapy; 3) diabetes mellitus (hyperglycemia preceding corticosteroid therapy); 4) diseases of the respiratory tract (chronic bronchitis, asthma bronchiale, emphysema, neoplasm); 5) anemia (hemoglobin < 12 gm/100 ml for women and < 14 gm/100 ml for men); and 6) adiposity (body weight (kg) more than 10% above Broca’s formula of body height in cm – 100 cm). Other parameters included: 1) whether corticosteroid therapy was given prior to operation; 2) the patient’s age (< 60 years or ≥ 60 years); and 3) the patient’s sex.

These 14 parameters were analyzed in the 186 Group A patients to define an expected incidence. Group A patients were then segregated into those who developed PILRT and those who did not, and the incidence of the 14 parameters was calculated separately in each group. We then tested whether this distribution was different from the expected incidence (chi-square test, probability of error below 5%). When a parameter was more common in patients developing PILRT than in those not developing PILRT, it was considered associated with an increased risk and was labeled as a “risk factor.” The interdependency of these risk factors was examined in a “matched pairs” trial. From the risk factors, a “risk score” was composed based on tests of various models. Finally, the best-fitting model of Group A was applied to Group B, in order to test its prognostic power.

Results

Incidence of Lower Respiratory Tract Infection

Sixty-two (33%) of the 186 Group A patients developed PILRT (the 95% confidence interval ranged from 26% to 39%), and 42 (23%) had pneumonia. Among the 103 Group B patients, 32 (31%) developed PILRT, and 18 (17%) had pneumonia.

Risk Factors

From the incidence of PILRT in Group A patients, we derived the null hypothesis: the preoperative signs and symptoms being tested for their importance as risk factors should be distributed between the 62 patients developing PILRT and the 124 not developing PILRT in a proportion of 62:124 (1:2). Five of the 14 signs and symptoms were not distributed in this manner according to chi-square testing (Table 3); therefore, the null hypothesis was rejected for these five items and they were labeled “risk factors.” These consisted of: 1) diagnosis of tumor: there was a reduced risk for transsphenoidally operated pituitary adenomas (p = 0.04) and an increased risk for meningiomas (p = 0.01); 2) age: there was an increased risk for the patients aged 60 years or older (p = 0.02); 3) cardiac failure: there was an increased risk for preoperative cardiac failure (p = 0.003); 4) corticosteroid therapy: there was an increased risk for preoperative corticosteroid therapy (p = 0.01); and 5) level of consciousness: there was an increased risk for preoperative disturbances of consciousness (p = 0.005). Two of the other preoperative characteristics showed a tendency to increase the risk of PILRT but did not reach the level of significance: namely, preexisting respiratory disease (p = 0.09) and preoperative cerebral seizures (p = 0.20). The distribution of the other seven parameters corresponded to the expected distribution (0.35 < p < 0.75).

To test the interdependency of the five risk factors in Group A patients, a matched-pairs analysis was performed and it was found that each risk factor influenced the incidence of PILRT independently of the existence of the other four factors (Table 4). From these five risk factors, a risk score was modeled, assigning to every patient a number of risk points in the following manner. For the diagnosis of tumor, transsphenoidally operated pituitary adenoma received 0 points, meningioma received 2 points, and every other kind of tumor received 1 point. In addition, 1 point was given for the existence of each of the other four risk factors. Thus, the score ranged from 0 to 6 points.

Group A was reduced by eight patients who died prior to the 3rd postoperative day (and therefore had almost no chance of developing PILRT). The remaining 178 patients were grouped according to their risk scores. The incidence of PILRT was calculated for each subgroup. For 0 points there was a 10% PILRT incidence (10 cases), for 1 point 14% (43 cases), for 2 points 24% (51 cases), for 3 points 51% (45 cases), and for 4 points 61% (23 cases). The subgroups with 5 points (five cases) and 6 points (one case) were not of sufficient size to allow a statistical analysis. A correlation was found between the risk score and the PILRT incidence; in the range from 0 to 4 points, this was approximately linear, with a coefficient of correlation according to Pearson’s coefficient of r = 0.975 (t5 = 10.60, df = 4, p = 0.001) and a regression line of y = 13.9 x + 4.2, where x represents points of risk score and y represents the percent of PILRT incidence (Fig. 1).

Other models were tested with different weightings of the risk factors, and the procedure was repeated including the two signs that failed to exhibit statistical significance.

The correlation was found to be linear with a coefficient of correlation according to Pearson’s coefficient of r = 0.975 (t5 = 10.60, df = 4, p = 0.001) and a regression line of y = 13.9 x + 4.2, where x represents points of risk score and y represents the percent of PILRT incidence (Fig. 1).

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### TABLE 4

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>No. of Matched Pairs</th>
<th>Incidence of PILRT in Patients with Risk Factor</th>
<th>Incidence of PILRT in Matched Controls</th>
<th>Ratio†</th>
</tr>
</thead>
<tbody>
<tr>
<td>pituitary adenoma</td>
<td>22</td>
<td>14%</td>
<td>32%</td>
<td>0.44:1</td>
</tr>
<tr>
<td>meningioma</td>
<td>41</td>
<td>49%</td>
<td>31%</td>
<td>1.58:1</td>
</tr>
<tr>
<td>age &gt; 60 years</td>
<td>57</td>
<td>39%</td>
<td>28%</td>
<td>1.39:1</td>
</tr>
<tr>
<td>cardiac failure</td>
<td>21</td>
<td>57%</td>
<td>41%</td>
<td>1.39:1</td>
</tr>
<tr>
<td>corticosteroid therapy</td>
<td>59</td>
<td>41%</td>
<td>30%</td>
<td>1.37:1</td>
</tr>
<tr>
<td>disturbance of conc-</td>
<td>31</td>
<td>55%</td>
<td>44%</td>
<td>1.25:1</td>
</tr>
</tbody>
</table>

* PILRT = postoperative infection of the lower respiratory tract.
† Ratio of incidence of PILRT in patients with risk factor matched controls.

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significance but showed a trend toward increasing the risk (cerebral seizures and preexisting diseases of the respiratory tract). In no model, however, did the correlation coefficient increase above the value mentioned above for the simple model described. In other words, the identified risk factors are roughly similar in their prognostic power and together they are responsible for about 95% of variance of the PILRT incidence. The risk score was then applied to the Group B patients and was found to be of high prognostic value (Table 5).

Time of PILRT Development

In 24 Group A patients who were intubated for more than 12 days we identified the interval between operation and the first appearance of PILRT. For every day after the operation the number of PILRT-free patients in this subgroup was recorded (Fig. 2). There was an exponential decrease in this number according to the formula:

\[ N_t = N_0 \times e^{-\lambda(t-1)}, \]

where \( N_t \) denotes the fraction of patients PILRT-free on Day \( t \) (day of surgery; \( t = 0 \)), \( N_0 \) denotes the size of the population (24 cases), \( e \) denotes Euler number (the base of the natural logarithm), \( t \) denotes the number of days after operation, and \( \lambda \) denotes the constant of proportion. In our population, \( \lambda = 0.198 \). Thus, the "half-life of freedom from PILRT" was

\[ T_{1/2} = \frac{\ln 2}{\lambda} = 3.5 \text{ days} \]

following the 1st postoperative day; that is, the number of patients who are free of PILRT is reduced every 3.5 days to one-half of the previous number. This law was valid for patients with medium duration of postoperative intubation (4 to 11 days) as well, but only for the 1st week after surgery (Fig. 2).

Discussion

A positive diagnosis of nosocomial pneumonia in intubated patients is not easy. It is usually based on four signs and symptoms: colonization of the bronchial tree; fever; leukocytosis; and the appearance of infiltrates on x-ray films of the lungs as an indicator of parenchymal reaction. Some authors include additional signs and symptoms for diagnosis: pathological sounds in auscultation; results of blood gas analysis; bacteriological investigation of pleura punctate or blood culture; purulence of the bronchial secretion; or the success of antibiotic therapy. Each of these signs and symptoms is nonspecific in patients with cerebral lesions who are in the intensive care unit after brain operations: elevated body temperature and leukocytosis appear to be part of the postoperative picture even without any infection. Blood in the subarachnoid space increases body temperature. The appearance of bacteria or yeasts in the bronchial secretion shows colonization, but does not prove infection. Radiological investigation of patients under intensive

### TABLE 5

<table>
<thead>
<tr>
<th>Risk Score</th>
<th>Calculated Incidence† (CI)</th>
<th>No. of Cases (N)</th>
<th>Patients Developing PILRT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Expected (CI × N)</td>
</tr>
<tr>
<td>0</td>
<td>4%</td>
<td>8</td>
<td>(0.3) = 0</td>
</tr>
<tr>
<td>1</td>
<td>18%</td>
<td>20</td>
<td>(3.6) = 4</td>
</tr>
<tr>
<td>2</td>
<td>32%</td>
<td>40</td>
<td>(12.8) = 13</td>
</tr>
<tr>
<td>3</td>
<td>46%</td>
<td>19</td>
<td>(8.7) = 9</td>
</tr>
<tr>
<td>4</td>
<td>60%</td>
<td>11</td>
<td>(6.6) = 7</td>
</tr>
<tr>
<td>5</td>
<td>74%</td>
<td>5</td>
<td>(3.7) = 4</td>
</tr>
</tbody>
</table>

* PILRT = postoperative infection of the lower respiratory tract.
† CI = 4.2 + 13.9 × risk score derived from Group A analysis (see text).

FIG. 1. Correlation between risk score (RS) and incidence of postoperative infection of the lower respiratory tract (% PILRT). See text for statistical discussion.

FIG. 2. Incidence of patients free of postoperative infection of the lower respiratory tract (% non-PILRT) during the 13 days after operation. Open circles: 24 patients with long-lasting intubation (more than 11 days); crosses: 45 patients with medium-lasting intubation (4 to 11 days); closed circles: expected results calculated from Equation 1 (see text).
care (patients are lying down so pictures are only available in one plane) is blemished by up to 30% false-positive and false-negative results.\textsuperscript{8,16,49,50} Furthermore, from a pathological point of view there seems to be a continuous transition between an infection being limited to the mucous membrane of the bronchial tree (bronchitis) and the additional involvement of lung parenchyma (pneumonia).\textsuperscript{11,20,34,37} For this reason, radiological evidence of pulmonary infiltrates is an artificial subdivision of a continuous pathological process. The application of invasive diagnostic methods, which possibly offer better results (for example, lung biopsy\textsuperscript{49} or transtracheal aspiration\textsuperscript{3}), is not without risk to the patient. We therefore decided not to differentiate between bronchitis and pneumonia, and considered PILRT as a whole. Differentiation was performed only suplementarily.

The incidence of PILRT by this definition was 33% in Group A. In Group B it lay well within the 95% confidence interval of this number. Thus, the move of the neurosurgical intensive care unit to a new building had no apparent effect on the results. The section of signs and symptoms to be examined for their prognostic power was based on the findings of previous studies.\textsuperscript{10,18,22,29,30,36,38,41,47,54,58,59} Specific neurosurgical factors were considered as well as the necessity for identification prior to operation. Our selection of factors was limited by the method of retrospective analysis and the relatively small size of the population. Some rare characteristics (for instance, gout) could not be considered whereas others were summarized (for instance, different kinds of preexisting diseases of the respiratory tract). The dichotomy of these characteristics (present or absent) is an artificial simplification; nevertheless, the prognostic power of the resulting risk score is high. The five risk factors identified (type of tumor, patient's age, disturbance of consciousness, cardiac insufficiency, corticosteroid therapy) account for about 95% of the variance in the incidence of PILRT in our population. As expected, there is no deterministic interrelationship between the risk factors and the appearance of the PILRT; rather, a statistical correlation was confirmed: the higher the risk score of a patient, the higher the probability that he would develop PILRT. The correlation was nearly linear and gave a prognosis of considerable reliability.

From a neurosurgical point of view, the increased PILRT risk for meningioma patients is remarkable. It could not be explained by the higher age of the patients alone (mean age of meningioma patients 58.0 years, gliomas 52.3 years, pituitary adenomas 53.0 years) nor by a higher incidence of other risk factors in these patients, as could be shown by matched-pairs analysis. Possibly, the special dynamics of focal brain edema and intracranial pressure after meningioma operations\textsuperscript{28} are critical in these patients. For patients undergoing transsphenoidal surgery for pituitary adenomas the low risk of PILRT is probably associated with the extracranial approach.

It has been shown that corticosteroid therapy increases the risk of infection in neurosurgical patients following severe brain injury\textsuperscript{4,41} and supratentorial hemorrhage.\textsuperscript{42} This corresponds to our findings in patients undergoing surgery for intracranial tumors. In our patients, however, corticosteroid therapy was given according to clinical needs. Possibly, patients treated with dexamethasone were sicker than those not being thus treated, so in these cases no conclusions can be drawn about causality.

Some characteristics that were identified as risk factors in other study populations did not prove to be relevant in our population. This was true for patient's sex,\textsuperscript{18,25,59} body weight,\textsuperscript{26,27,59} and the presence of diabetes mellitus.\textsuperscript{38} We are not able to comment on whether the application of histamine β-antagonists influences the incidence of PILRT, inasmuch as almost every patient in our population (except patients with transsphenoidal surgery for pituitary adenoma) received this treatment.

The correlation between the incidence of PILRT and the duration of postoperative intubation is well documented.\textsuperscript{12,31,32,38,52} In our population, patients who were extubated before the 3rd postoperative day showed a PILRT incidence of only 3%. For those intubated for a longer period there was an exponential decrease in the number of PILRT-free patients: every 3.5 days the number dropped to half the previous number. Mandelli, et al.,\textsuperscript{39} recently published data nearly identical to ours. Considering only PILRT which showed infiltration of pulmonary parenchyma, we found an incidence of 23% in Group A and 17% in Group B. This figure is very similar to the 23% mentioned for patients after severe brain injury by Braun, et al.,\textsuperscript{5} and also to the data of Mandelli, et al., who reported a 17% incidence in a mixed intensive-care unit series, and of Stevens, et al.,\textsuperscript{46} who reported a 22% incidence among surgical and anesthesiological patients receiving intensive care.

Our results indicate that useful predictions can be made on the risk of PILRT in brain-tumor patients even before the operation.

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