Oral Health and Respiratory Infection

Philippe Mojon, DMD, PhD

Abstract

The oral cavity has long been considered a potential reservoir for respiratory pathogens. The mechanisms of infection could be aspiration into the lung of oral pathogens capable of causing pneumonia, colonization of dental plaque by respiratory pathogens followed by aspiration, or facilitation by periodontal pathogens of colonization of the upper airway by pulmonary pathogens. Several anaerobic bacteria from the periodontal pocket have been isolated from infected lungs. In elderly patients living in chronic care facilities, the colonization of dental plaque by pulmonary pathogens is frequent. Notably, the overreaction of the inflammatory process that leads to destruction of connective tissue is present in both periodontal disease and emphysema. This overreaction may explain the association between periodontal disease and chronic obstructive pulmonary disease, the fourth leading cause of death in the United States. These findings underline the necessity for improving oral hygiene among patients who are at risk and those living in long-term care institutions.

Epidemiology of Pulmonary Disease: Significance of the Problem

Pneumonia is usually classified as community-acquired pneumonia (CAP) or nosocomial (hospital-acquired) pneumonia. The distinction is important because the pathogens implicated and the preventive measures taken are very different for the 2 types. CAP is a frequent illness, with an incidence rate estimated at about 8 cases per 1,000 inhabitants per year in industrialized countries. The mortality rate is about 7% in hospitalized patients. Streptococcus pneumoniae and Haemophilus influenzae are the main causative organisms (accounting for 40% to 60% of cases). Until now, the only authors who have looked at the association between acute respiratory diseases (including CAP) and oral health markers did not find any association between them.

Nosocomial pneumonia is the second most common infection (after infections of the urinary tract) in long-term care institutions. It accounts for approximately 10% to 15% of all hospital-acquired infections, and 20% to 50% of affected patients will die because of the infection. The etiologic agents are mainly gram-negative bacilli and staphylococci. The frequency of infection with anaerobic organisms is uncertain because of the technical difficulty associated with anaerobic culture and the possibility of contamination by anaerobic oral flora during sampling. Because of these difficulties, anaerobic bacteria are not cultured in routine analysis of pulmonary microbiological samples. In one frequently cited study, 35% of nosocomial infections were due to obligate anaerobes. Aspiration pneumonia refers to bacterial infection occurring in association with a condition predisposing to aspiration, such as
stroke, Parkinson’s disease or alcoholism. The bacteria involved are indigenous oral flora or they are acquired in hospital. As for aspiration pneumonia, the infections occurring during mechanical ventilation are closely associated with anaerobic bacteria.6

Because anaerobes are implicated in pneumonia, a role for the oral cavity in the pathogenesis of this disease has been suspected since the beginning of the 20th century. Yet it was only in the 1970s that the role of anaerobic bacteria in pulmonary infections came to be investigated more extensively.7

The oral flora may also be implicated in pulmonary diseases affecting airflow. The most prevalent is chronic obstructive pulmonary disease (COPD). In the United States, COPD affects 14 million people, and in 1991, it was the fourth leading cause of death, a toll that has been increasing over recent years.1 The main etiological factor is tobacco smoking, but bacteria (including oral bacteria) may play a key role in progression of the disease.

Given the high prevalence of lower respiratory tract infection (RTI), if even a small percentage of these cases are caused or facilitated by oral flora, the total number of pulmonary infections attributed to these organisms would be significant.

**Mechanism of Infection**

Two routes exist for oral micro-organisms to reach the lower respiratory tract: hematogenous spread and aspiration. Hematogenous spread of bacteria is an inevitable adverse effect of some dental treatments and may occur even after simple prophylactic procedures. Nonetheless, this route of infection seems rare, and only 2 well-documented case reports could be found in the literature. In both cases hematogenous spread was the most likely source of pulmonary infection with periodontal anaerobes.8,9 In contrast, aspiration of material from the upper airway occurs in 45% of healthy subjects during sleep and in 70% of subjects with impaired consciousness.10 It is probably the main cause of nosocomial infection along with aspiration of gastric contents. Three mechanisms of infection related to aspiration of material from the upper airway can be envisioned. First, periodontal disease or poor oral hygiene might result in a higher concentration of oral pathogens in the saliva. These pathogens would then be aspirated into the lung, overwhelming the immune defences. Second, under specific conditions, the dental plaque could harbour colonies of pulmonary pathogens and promote their growth. Finally, periodontal pathogens could facilitate the colonization of the upper airways by pulmonary pathogens.

**Microbiological Similarities Between Organisms Infecting the Lungs and Oral Flora**

The vast majority of pulmonary diseases are due to aerobic bacteria that are found in the oral flora but are not related to any oral diseases.11 In contrast, the list of anaerobes (facultative or obligate) that are implicated in the destruction of periodontal tissues and that have also been isolated from infected lungs is quite long.12 For example, Actinobacillus actinomycetemcomitans and Fusobacterium nucleatum have both been isolated from infected lungs9 whereas Pseudomonas aeruginosa, a known pulmonary pathogen, has been isolated from patients with “refractory” periodontitis.12 The pulmonary pathogenicity of Bacteroides gingivalis has been confirmed in an animal model simulating aspiration.13

It seems that commensal oral micro-organisms such as Streptococcus intermedius may also become opportunistic pathogens under specific circumstances. The colonization of the oral flora by respiratory pathogens has specifically been investigated in 4 studies. Lindemann and others14 isolated strains of P. aeruginosa from the tongues of 14 of 20 patients with cystic fibrosis. None of the 20 age-matched healthy patients had these bacteria, and none of the 40 plaque samples (from patients with cystic fibrosis and controls) were colonized by these strains either. However, in a study conducted in a critical care ward, Scannapieco and others15 isolated several known pulmonary pathogens (e.g., Klebsiella pneumoniae and Serratia marcescens) from dental plaque. In contrast, none of the plaque samples collected from patients in a dental clinic harboured these species. In a similar study, Fourrier and others16 found a high bacterial concordance between dental plaque, saliva and tracheal samples. In about 40% of their intensive care patients, dental plaque was colonized by aerobic respiratory pathogens. The colonization of dental plaque was highly predictive of concurrent or subsequent nosocomial infection. In the most recent study,17 plaque colonization in subjects from a chronic care facility was compared with plaque colonization in age-matched outpatients from a dental clinic. In both groups, about a quarter of the samples were colonized, but the concentration of bacteria was much higher among the subjects from the chronic care facility.

Edentulous subjects participated in 2 of these studies. It seems that some species, such as A. actinomycetemcomitans and Porphyromonas gingivalis, cannot be found after dental clearance,18 but other putative pulmonary pathogens such as Prevotella spp. are present in the oral microbiota of edentulous patients.19 If denture hygiene is inadequate, anaerobic bacteria count is inversely proportional to the cleanliness of the denture.20 Also worth mentioning is the fact that Candida albicans has been isolated from transtracheal aspirates from patients with pleurapulmonary infection6 and other pulmonary samples.21 Nevertheless, it seems that RTI is less frequent among edentulous patients.22,23
Mucosal Colonization and Periodontal Pathogens

Fibronectin, a protein that coats the oral mucosa, is probably involved in the ecology of the mucosal flora by providing binding sites for oral streptococci while inhibiting adhesion of more virulent bacteria. The competition for colonization seems to be modulated by the ability of bacteria to degrade fibronectin. The protease activity (including degradation of fibronectin) of periodontal pathogens has been extensively studied and is correlated with poor oral hygiene. Thus, fibronectinolytic activity of the crevicular fluid could play a role in pneumonia by promoting the adherence of anaerobic gram-negative bacteria to the epithelium of the upper airway. Scannapieco and Mylotte proposed a model to explain the colonization of the oral cavity by respiratory pathogens.

Linking Respiratory Infections and Oral Disorders

Epidemiological indications that oral microorganisms might be implicated in lung infections are still scant. Pneumonia is most frequent in elderly people, and this segment of the population would seem a logical target for such research. However, pneumonia in elderly people has specific features that make it difficult to study. For example, the traditional clinical signs used to diagnose pneumonia (cough, fever and abnormal auscultation) lack sensitivity and specificity. Chest radiography is the gold standard for diagnosis, but it is rarely available. Also, elderly patients often have comorbidities or risk factors that could confound the relationship between oral disorders and pneumonia. For example, poor nutritional status and medioc...
taken into account, the risk of COPD was 1.8 times greater among the 20% of subjects with the worst alveolar bone loss (the worst quintile).

These epidemiological findings seem to be supported by biological clues. According to Travis and others, pulmonary emphysema and periodontal disease share a similar mechanism of tissue destruction. Neutrophils are recruited to inflammatory sites either because of the presence of foreign materials (e.g., smoke particles) or because of chemotactic factors activated by bacteria. In both diseases, degranulation of neutrophils occurs during attempted phagocytosis (so-called “frustrated phagocytosis”), which releases proteolytic enzymes. Proteins from the connective tissue are degraded, which results in the destruction of the pulmonary alveolus or the periodontal attachment (Fig. 1).

For cystic fibrosis, the only information available is the work of Lindemann and others, as discussed earlier.

**Conclusion**

This literature review highlighted at least 12 studies that provide direct evidence of an association between pulmonary infection and oral diseases. The association seems to occur only in patients with severely compromised health, in frail elderly people and in patients with chronic pulmonary diseases. Accumulation of plaque on the teeth or dentures and some periodontal pathogens are the most likely culprits. Yet we cannot rule out that similar host susceptibility factors may lead to both periodontal and chronic pulmonary diseases. These results corroborate the suggestion made earlier that improving oral hygiene might reduce the risk of pneumonia among subjects who are at risk. A more rapid intervention would be the use of an oral disinfectant, but studies on the long-term use of such medication are lacking. The treatment of periodontal diseases (either by repeated prescription of antibiotics or by clinical interventions) might be another way to reduce the incidence of pneumonia. This literature review underlines the necessity for regular recalls among “at risk” patients and the introduction of specific

![Figure 1: Mechanism of tissue destruction in periodontal disease and emphysema (Adapted, with permission, from Travis)](image-url)
oral hygiene courses for caregivers in long-term care institutions. Nonetheless, a causal association has not been proven, and more studies, in particular intervention studies, are needed.

**Table 2  Main studies showing direct evidence of an association between pulmonary infection and oral diseases**

<table>
<thead>
<tr>
<th>Authors and others</th>
<th>Date</th>
<th>Sample size</th>
<th>Study design</th>
<th>Main respiratory outcome</th>
<th>Major finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lindemann and others</td>
<td>1985</td>
<td>40</td>
<td>C</td>
<td>Respiratory pathogens</td>
<td>Oral cavity is a potential reservoir for <em>Pseudomonas aeruginosa</em> in patients with cystic fibrosis</td>
</tr>
<tr>
<td>Pugin and others</td>
<td>1991</td>
<td>52</td>
<td>RCT</td>
<td>VAP</td>
<td>Oropharyngeal decontamination by topical antibiotics reduces aerobic gram-negative bacteria and VAP in an intensive care unit</td>
</tr>
<tr>
<td>Scannapieco and others</td>
<td>1992</td>
<td>59</td>
<td>C</td>
<td>Respiratory pathogens</td>
<td>Respiratory pathogens colonize dental plaque and oral mucosa</td>
</tr>
<tr>
<td>DeRiso and others</td>
<td>1996</td>
<td>353</td>
<td>RCT</td>
<td>Nosocomial RTI</td>
<td>0.12% chlorhexidine rinse reduces occurrence of nosocomial respiratory infection in patients undergoing heart surgery</td>
</tr>
<tr>
<td>Mojon and others</td>
<td>1997</td>
<td>302</td>
<td>C</td>
<td>RTI</td>
<td>RTIs are associated with greater plaque accumulation, specific oral health disorders and presence of teeth</td>
</tr>
<tr>
<td>Scannapieco and others</td>
<td>1998</td>
<td>386</td>
<td>C</td>
<td>Respiratory diseases</td>
<td>No relationship exists between acute respiratory diseases and oral condition; poor oral hygiene is associated with chronic diseases</td>
</tr>
<tr>
<td>Fourrier and others</td>
<td>1998</td>
<td>57</td>
<td>L</td>
<td>Nosocomial pneumonia</td>
<td>Respiratory pathogens in dental plaque are associated with nosocomial pneumonia</td>
</tr>
<tr>
<td>Hayes and others</td>
<td>1998</td>
<td>1,118</td>
<td>L</td>
<td>COPD</td>
<td>Alveolar bone loss is associated with greater risk of COPD</td>
</tr>
<tr>
<td>Russell and others</td>
<td>1999</td>
<td>58</td>
<td>C</td>
<td>Respiratory pathogens</td>
<td>Respiratory pathogens were not found in dental outpatients but were found in 14% of patients living in chronic care facilities</td>
</tr>
<tr>
<td>Yoneyama and others</td>
<td>1999</td>
<td>366</td>
<td>CT</td>
<td>Pneumonia</td>
<td>Oral care decreases risk of pneumonia in frail, dependent elderly people</td>
</tr>
<tr>
<td>Scannapieco and Ho</td>
<td>2001</td>
<td>13,792</td>
<td>C</td>
<td>COPD</td>
<td>Association exists between COPD and attachment loss</td>
</tr>
<tr>
<td>Terpenning and others</td>
<td>2001</td>
<td>358</td>
<td>L</td>
<td>Aspiration pneumonia</td>
<td>Caries, cariogenic pathogens and periodontal pathogens are significant risk factors</td>
</tr>
</tbody>
</table>

*C = cross-sectional  
RCT = randomized clinical trial  
VAP = ventilator-associated pneumonia  
RTI = respiratory tract infection  
L = longitudinal  
COPD = chronic obstructive pulmonary disease  
CT = non-randomized clinical trial*
References