KNOWLEDGENETWORK
Kimberly-Clark Health Care Education

VAP Prevention: Excellence in Oral Care

"One of the most critical risk factors for ventilator-associated pneumonia is microbial colonization of the oropharynx."[1]

Etiological agents: Bacteria colonizing oropharynx confirmed as primary VAP causative agents

DNA clinical studies have confirmed that up to 90% of VAP is caused by pathogens colonizing the mouth. The CDC 2003 Guideline for the Prevention of Nosocomial Pneumonia states that in 76% of VAP cases, bacteria colonizing the mouth before pneumonia is diagnosed are the same as those causing the pneumonia.[2]

Pathology: Oral environment attracts respiratory pathogens awaiting access to lungs

Oral physiology altered: Within hours of admission, the oral physiology of the ICU patients begins to change beginning with a decrease in saliva production. The decrease in saliva reduces the volume of fluid available for adequately rinsing of oral surfaces. Tissues become seriously dry (xerostomia) often causing tissue inflammation and injury (mucositis-lesions). Chapped lips and oral lesions provide niches for microorganisms to hide and proliferate.[3] As saliva contains lactoferrin, cortisol, neopterin and IgA antibodies, diminishing levels prevent delivery of these important immunocological defenses. Saliva production is further decreased if the patient is sedated, on antihypertensives, sympathomimetics or anticholinergics, or is receiving chemotherapeutic treatments.[4,5] Reduced saliva causes a drop in pH to neutral levels making the environment less hostile to microbial invaders. Simultaneously, oral protease production is increased; an enzyme that normally starts protein digestion. When over produced, protease begins to digest fibronectin, a glycoprotein that coats cells in the oropharynx, covering receptor sites that allow pathogens to attach. As the fibronectin is digested, more and more receptor sites are exposed, ready to accept respiratory pathogens.[6]

Respiratory pathogens take up residence: Within 48 hours the microbial population of the mouth shifts to respiratory pathogens such as *Staphylococcus aureus, Streptococcus pneumoniae, Pseudomonas aeruginosa, Haemophilus influenza*, and *Acinetobacter baumannii* .[7,8] As the bacteria attach to teeth, they rapidly exude substances forming the biofilm matrixes we refer to as plaque. These extremely protective structures enable microorganisms to reproduce and spread rapidly above and below gingival margins and in the spaces between teeth.[9]

ET tube facilitates pathogen access: The presence of an endotracheal tube (ET tube) prevents normal coughing, normal swallowing and the protection of the trachea access by epiglottis closure. Further tissue desiccation occurs as the mouth is propped open 24 hours a day. As respiratory pathogens continue to multiply within their protective plaque structures, many make their way into the subglottic pool, and are positioned for migration around the ETT cuff, enabling them to drop into the lungs and initiate infection[10].

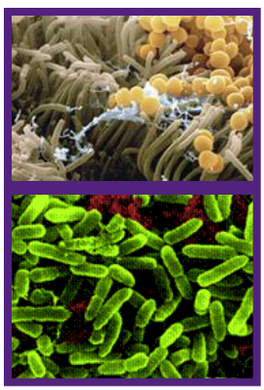
Place
Business Card
Here

Early onset of VAP pathogens:

- *Staphylococcus aureus* (Methicillin sensitive-MSSA)
- *Haemophilus influenza*
- *Streptococcus pneumonia*

Late onset VAP:

- *Staphylococcus aureus*
- Methicillin resistant-MRSA
- Pseudomonas aeruginosa
- Acinetobacter or Enterobacter



VAP Prevention: Excellence in Oral Care

Diligence in patient oral care is essential for VAP prevention[11,12,13]

- Educate staff on high probability that VAP is caused primarily by bacteria that colonize patients' mouths
- Post your standardized, effective oral care protocol, incorporating means to assist in and track compliance
- Provide standardized oral needs assessment tool from which care regimen can be established

- Maintain head elevation at 30-45° especially during feeding and oral care
- Start oral care a short time after intubation to discourage pathogen colonization and plaque formation
- Suction above ET tube cuff prior to oral care to reduce micro-aspiration risk

- Disrupt plaque formation using small soft-bristled toothbrush twice daily (swabs, gauze not adequate).[14] Include gums and tongue to stimulate tissues and prevent colonization even on the edentulous (toothless)
- Use effective antiseptic rinse such as chlorhexidine gluconate (CHG) or hydrogen peroxide
- Perform oral suctioning frequently.
- Prevent environmental contamination of used oral suction devices –utilizing product design, appropriate cover or holstering, disinfection, or use of single use products
- Frequently use water-soluble moisturizer to prevent desiccation/lesions. Avoid lemon-glycerine swabs as mucosal drying has been reported
- Lubricate lips with lip balm routinely to prevent chapped lips that provide niches for growth and infection

Primary Route of VAP Pathogenesis

Saliva flow decreases causing

- Mucosal tissues dry out = xerostomia
- Inflammation and lesions develop = mucositis
- pH drops to neutral = less hostile to microbial invaders

Saliva containing defenses not delivered

- lactoferrin
- cortisol
- neopterin
- IgA antibodies

Oral protease production increases

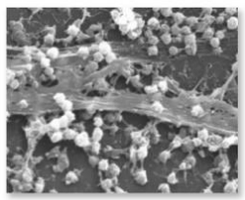
- digests fibronectin exposing microbial receptors on oropharyngeal cells

Microbial population shifts to respiratory pathogens

- *Staphylococcus aureus*
- *Streptococcus pneumoniae*
- *Pseudonmonas aeruginosa*
- *Haemohilus influenza*
- *Acinetobacter baumanii*

Plaque protects pathogens

- rapidly multiply and plaque thickens



Dental plaque early stages (hours)

Pathogens into pooled secretions above ET tube cuff

Micro-aspiration around cuff or through cuff folds → **lung defenses overcone** → **Pneumonia**

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Kimberly-Clark

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