Preventing Aspiration in the Nursing Home: The Role of Biofilm and Data from the ICU

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Two aspiration syndromes have been identified: Aspiration pneumonia is infectious caused by micro-aspiration of oral bacteria secondary to neurogenic dysphagia or sedation. Infectious bacteria may also be aspirated from the stomach. Aspiration pneumonitis classically follows large bolus aspiration of food, acid, or digestive enzymes and is initially noninfectious. Large bolus gastric aspiration events may have an acute/dramatic onset. This article discusses (1) prevention of recurrent aspiration events caused by 2 common motility disorders: neurogenic dysphagia and gastro esophageal reflux; (2) mechanical source control (debridement/drainage) of sites that may harbor large collections of bacteria protected from antibiotics in biofilm including dental plaque, coated tongue, and chronic sinusitis. **(J Am Med Dir Assoc 2010; 11: 70–77)**

Keywords: Aspiration pneumonia; prevention; nursing home

Many of the diseases and functional impairments that lead to nursing home (NH) placement also facilitate pulmonary aspiration. Mylotte et al[1] classified 75% of hospital admissions with suspected pneumonia from NHs as "aspiration" on the basis of sudden onset, witnessed vomiting, tube feeding, choking while eating, or dysphagia.

The title of this manuscript refers to the intensive care unit (ICU) as a source of information to be considered by NH clinicians. Why might this be a valid projection? NH and ICU patients at high risk of aspiration share many risk factors, including impaired mental status and swallowing, dependence for oral hygiene, immobility, polypharmacy, recurrent pneumonia, colonization pressure, and multiple courses of antibiotics with selection of resistant organisms. One major difference is that many ICU patients are intubated. The ICU is a high-tech environment with intense monitoring and frequent sampling of lower respiratory secretions. Unlike the NH, numerous descriptive and controlled studies performed in ICUs have led to data-driven clinical practice guidelines (CPGs) that might be adapted to the NH.

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DOI:10.1016/j.jamda.2009.03.020

CLINICAL ASPIRATION SYNDROMES

Two classic clinical aspiration syndromes have been identified, although overlap may exist. Aspiration pneumonia is infectious and caused by micro-aspiration of oral bacteria secondary to neurogenic dysphagia or sedation. However, gastric bacteria may also be aspirated and cause pulmonary infection.

Aspiration pneumonitis classically follows large bolus gastric aspiration of food, acid, or digestive enzymes and is initially noninfectious. Large bolus gastric aspiration events may have an acute/dramatic onset and occur when the resident is lying flat (with vomit on the pillow) or may be witnessed by staff after a meal. An unwitnessed acute onset can lead to diagnostic confusion with vascular events such as paroxysmal nocturnal dyspnea or pulmonary embolus. The resident's clinical condition may improve rapidly following a noninfectious gastric aspiration event. This possibility led an expert consensus panel to recommend withholding antibiotic therapy following such an event unless symptoms persisted beyond 24 hours. This recommendation has not been prospectively validated.[2,3] On the other hand, large bolus aspiration may be followed by progressive severe respiratory distress. Similar to viral lower respiratory infections, the damaged mucosa may be less resistant to subsequent bacterial inoculation.

Aspiration may present as an acute illness, a subacute illness with anaerobic abscess formation, or as chronic diffuse pulmonary lesions. Clinicians may overlook an aspiration mechanism in these indolent cases. If foreign material (food, medicine) is visualized in the bronchioles, aspiration

could have been caused by pharyngeal dysphagia or regurgitation from the stomach. Clinically, individuals often have risk factors for both. The clinician can make an educated guess based on which of the two possibilities is most prominent clinically. In one retrospective postmortem review of 5000 autopsies, Japanese investigators identified 4.8% of cases as aspiration pneumonia as well as 31 cases (0.6%) with recurrent aspiration of foreign particles. The latter cases included scattered miliary yellowish nodules similar to those seen in diffuse panbronchiolitis.[4] In a second report, pathologists in New York retrieved surgical or biopsy specimens in which vegetable or foreign material was observed in the bronchioles. The pathologic appearance was characterized as a suppurative and granulomatis reaction with multinucleated giant cells, foreign body granulomas and/or acute bronchopneumonia/bronchiolitis. The principal pathology identified in 88% was bronchiolitis obliterans-organizing pneumonia (BOOP). BOOP is a nonspecific reaction to a variety of toxic insults.[5] Acid reflux may contribute to the pathogenesis of idiopathic pulmonary fibrosis.[6]

Most clinicians can identify infiltrates in the basal segments of the lower lobes (especially the right) as potential aspiration infiltrates. This location is classically involved if aspiration occurred when the resident was sitting upright. In addition, aspiration events that occur in the recumbent position produce infiltrates in the posterior-mid lung fields, specifically the posterior segment of the upper lobes and apical segments of the lower lobes.[2,7]

SOURCE CONTROL-BIOFILM

Infection control includes 2 important goals: (1) The prevention of transmission of multidrug-resistant organisms (MDRO) or organisms with special virulence, such as epidemic *Streptococcus pyogenes*, capable of producing myonecrosis; and (2) the prevention of colonizing organisms from causing infection. An example includes the prevention and healing of wounds by avoiding ischemia (pressure), dead space, excess exudates, and necrotic tissue by maintenance debridement. This article emphasizes the latter approach in the prevention of pulmonary infection.

During my internal medicine training I viewed the topic of pneumonia through a microscope, with a complete emphasis on microbiology/bacteriology and choice of antibiotic. I will refer to this as "little stuff." More recently I have been impressed that the NH clinician must also pay attention to the "big stuff." This includes macroscopic, grossly visible aspiration events related to pharyngeal dysfunction, vomiting, or gastroesophageal reflux (GERD). In addition, many patients harbor large collections of bacteria in dental plaque, pharynx, stomach, or sinuses. Many of these sites may include biofilm, which protects bacteria from antibiotic eradication.

In 1999 scientists from the Centers for Disease Control and Prevention (CDC) estimated that 65% of human bacterial infections involve biofilm.[8] This field is rapidly expanding. Biofilm may adhere/colonize biological (mucosal) surfaces or foreign bodies.[9] It is composed of layers of slow-growing often polymicrobial bacteria embedded in gummy glycocalyx (exopolysaccarides).[10] The exopolysaccarides in biofilm are not visualized by conventional light microscopy.[11] Detection requires scanning electron or laser microscopy. Fluorescence in-situ hybridization is required to identify specific bacteria.[9,12,13] Potential biofilm sites in the respiratory tract include dental plaque, coated tongue, chronic sinusitis, and bronchiectasis.[8,9,11,12,14–17]

Biofilm may function like a primitive multicellular organism. Close proximity of bacteria in biofilm facilitates chemical "cross talk" that allows bacteria to sense high density and trigger production of virulence factors.[18] Neighboring B-lactamase–producing bacteria may protect bacteria sensitive to penicillin.[19] Close proximity may also facilitate transfer of resistance factors between bacterial species.[9,20]

Glycocalyx interferes with antibiotic penetration and slow growth makes bacteria relatively resistant to growth-dependent antibiotic killing and difficult to grow using standard techniques.[10,12,17] Killing bacteria in biofilm requires antibiotic/disinfectant concentrations 10 to 1000 times those needed to kill free-living bacteria (planktonic).[21] Antibiotic dosing based on usual culture and sensitivity data derived from free-living bacteria should not be expected to be effective against bacteria embedded in biofilm. A biofilm site may function like a fortress capable of launching attacks of free-floating bacteria and recurrent exacerbations.[9,10]

It is well known that when bacteria are exposed to sub optimal antibiotic concentrations, that selection of MDRO is facilitated. Thomas et al[22] studied the pharmacodynamic factors associated with the development of antibiotic resistance in 107 patients with ventilator-associated pneumonia (VAP). In addition to dental plaque, coated tongue, sinusitis, and bronchiectasis, endotracheal tubes support biofilm.[23] Twenty-five percent of the 128 bacteria causing pneumonia in this study, developed antibiotic resistance during therapy. Resistance developed during suboptimal antibiotic exposure: area under the curve (AUC)/MIC less than 100.[22] Related data indicate that when oral tetracycline or amoxicillin is given to patients with periodontitis (biofilm), approximately 40% of anaerobes become resistant to that antibiotic.[24,25] The possibility of selecting resistant bacteria and recurrent acute exacerbations from a biofilm cite (R + R; Resistance + Recurrance) might be diminished by not underdosing antibiotics and performing mechanical debridement and/or drainage.[26,27] Infectious disease clinicians refer to this as "Source Control."

PHARYNX

The most accessible site in need of debridement and "source control" is the oropharynx. Dental plaque is a form of biofilm that breeds anaerobes, *Staphylococcus aureus*, and gram-negative rods (GNRs) including *Pseudomonas*.[28,29] One cubic millimeter contains 100 million bacteria.[28] Periodontal pockets are stagnant sites that support plaque formation above and below the gum line.[28,30,31] These pockets are a rich source of bacterial and neutrophile enzymes including elastase capable of destroying gum tissue; creating deeper pockets filled with slime.[31] These inflammatory enzymes may also damage lung tissue if aspirated.[28,32] Another adverse effect of periodontal enzymes is the destruction of fibronectin,

a molecule that facilitates binding of nonpathogens in the pharynx. The destruction of fibronectin opens binding sites for GNRs.[33–35] The pharynx as well as dental plaque may become colonized with GNRs, *S aureus*, and/or *Pseudomonas*, especially when oral hygiene is neglected. This probably explains the shifted bacteriology of health care–associated pneumonia versus outpatient pneumonia. In addition, *Pseudomonas*, *S aureus*, and *Haemophilus influenzae*, are potential biofilm formers.[9,15] A related point is the possibility of aspirating *Legionella* or *Pseudomonas* originating from biofilm sites in plumbing.[36,37]

El Solh and colleagues[38] sampled dental plaque from 49 NH residents admitted to the ICU who had received no antibiotics for 60 days. *S aureus* was isolated in 15 (9 methicillin-resistant *S aureus*), GNRs in 14, and *Pseudomonas* in 4. The proportion with pathogens would probably be greater if residents with recent antibiotic exposure had been included. Thirteen pathogens were subsequently recovered from bronchoalveolar lavage (BAL) performed in residents who developed pneumonia. Nine pathogens were genetically identical to the plaque isolates including 5 *S aureus* and 2 *Pseudomonas*. Terpenning et al[39] found that the presence of *S aureus* in saliva was a significant risk factor for aspiration pneumonia in elderly veterans independent of teeth.

Residents colonized with GNRs, *S aureus*, or yeast have decreased pharyngeal clearance of radio-labeled albumin from the oropharynx (stasis).[33] Mechanical clearance of the mouth is facilitated by salivary flow and swallowing. Anticholinergic medication and dehydration decreased salivary production and slowed clearance of isotope during experimental studies.[43] Saliva contains immunoglobulin (Ig) A, IgG, and antibacterial agents such as lactoferrin, an iron-binding glycoprotein.[35] Dry mouth has been associated with dental decay, coated tongue (a form of biofilm), halitosis, and in one study VAP.[14,40–42]

Chronic sinusitis may support biofilm formation. Tissue specimens were obtained during surgery in 40 patients with chronic sinusitis; 20 contained biofilm. These patients were 3 times more likely than patients without biofilm to have significant symptoms and mucosal inflammation on endoscopy 8 months after surgery.[12,15] In another study, scanning electron microscopy was performed on tissue from sinus surgery. Biofilm was detected in 24 of 30.[13]

Bacterial sinusitis is acknowledged as a cause of chronic cough in a pulmonary CPG.[44,45] It is difficult to believe that bacteria aspirated from the sinuses could not be a source of pulmonary infection, although this assertion is not established. A prospective randomized study performed in the ICU found a link between maxillary sinusitis and pneumonia.[46] NH residents may have additional risk factors driving the development of sinusitis. Oropharyngeal dysphagia can produce regurgitation into the nasopharynx. Regurgitated material from GERD is also acknowledged in a CPG as a possible cause of sinusitis.[49] It is conceivable that nasopharyngeal regurgitation originating in the oropharynx or stomach could be an additional cause of chronic sinusitis in NH residents. In addition, nasogastric (NG) tubes may interfere with sinus drainage and have been identified as a risk factor for radiographic sinusitis.[47,48]

PREVENTION/ORAL CARE

Japanese investigators randomized 184 residents to an intense oral hygiene program and compared them to 182 controls. The intervention group had their teeth brushed and mucosa wiped (sometimes with an antiseptic) after each meal as well as weekly cleaning by a hygienist. Over 2 years, pneumonia occurred in 21 oral care subjects with 14 deaths compared with pneumonia in 34 controls with 30 deaths ($P < .05$). Edentate residents also benefited, underscoring the need to clean the dentures, tongue, and mucosa as well as the teeth.[50] The intervention applied in this study would admittedly require resources beyond those available in most facilities; however, the study does provide "Proof of Principle." A smaller, 24-month, randomized study that included only weekly hygienist cleaning reported similar results. A later 6-month study by the same group reported that dental hygiene significantly lowered the risk of influenza diagnosed by rapid test in elderly outpatients.[16] A promising clinical experience was recently reported from an American NH. A certified nursing assistant position was dedicated to provide oral care to residents with abnormal mental status and/or aspiration potential. The authors reported that plaque and debris usually covered two thirds of the surface of the teeth of residents who did not receive focused oral cleaning. A subsequent analysis revealed that pneumonia mortality in the high-risk intervention group was the same as that of a low-risk nonintervention group from the same facility.[51]

Incapacitated residents may not tolerate or allow caregivers to clean their teeth and mouth. This task is labor intensive and requires the use of gloves and barriers to prevent splatter. The use of a rechargeable battery-powered toothbrush might be expected to diminish caregiver fatigue and lessen the likelihood of oral trauma or gagging secondary to poorly controlled caregiver movement during manual brushing. If the device is not turned off before exiting the mouth the caregiver may/will be splattered. These devices have not been formally evaluated in the NH. In healthy subjects battery-powered toothbrushes have reduced plaque scores by 40% and staining by 50% compared with traditional manual brushing.[52]

Another promising labor saving "technology" is the use of xylitol or sorbitol, "sugar-free" sweeteners, in the form of gum or hard candy. These products stimulate salivation and swallowing. The value of salivation and clearance of saliva was discussed earlier in this article. In one 18-month study, the use of xylitol-sweetened hard candy decreased plaque and gingivitis scores by 20% in disabled children.[53] Xylitol prevented caries more effectively than sorbitol in elderly veterans; however, there was no difference between xylitol and sorbitol regarding gingival or plaque index.[54,55]

Short-term topical application of antibiotics or antiseptics has proven efficacy preventing pneumonia in hospitalized patients. Chlorhexidine mouthwash (0.12%) applied twice a day with a sponge decreased respiratory infection by 69% in 173 patients versus 180 controls undergoing cardiac surgery.[56] Chlorhexidine may have a bad taste and stain teeth. A meta-analysis of 7 randomized studies of intubated patients

treated with topically applied antiseptics revealed that the relative risk of pneumonia was reduced to 0.56. The best results were obtained in surgical and trauma patients. This finding raises the possibility that topical application of antiseptics may have a more limited effect if plaque is well established and not debrided.[32,57] Of course, caregivers cleaning an environmental body fluid spill are trained to remove visible body fluid contamination followed by application of an antiseptic.

In 2004 the CDC published "Guidelines for Preventing Health Care Associated (HCA) Pneumonia."[58] The topical application of chlorhexidine 0.12% was recommended perioperatively during cardiac surgery. In addition long-term care facilities were encouraged to implement an oral hygiene program that might include an antiseptic.[56,58] The mouth is a source of HCA pneumonia, "clean it"! Caregivers do not allow rotting organic debris to elicit an inflammatory reaction on the buttock. It's important to maintain the same standard for the mouth, which drains directly into the lungs, especially in the presence of oropharyngeal dysphagia.

PREVENTION/OROPHARYNGEAL DYSPHAGIA

When NH workers hear the word "aspiration," the first thing (perhaps only thing) that comes to mind is the speech language pathologist and oropharyngeal dysphagia. Although this is an important part of the aspiration spectrum, I hope this article serves to broaden the approach to this complicated topic. Many of the swallowing interventions used by speech pathologists in NH residents with dysphagia lack sufficient evidence. Admittedly there are other interventions applied to residents by physicians and occupational and physical therapists that are not evidence based.

A large randomized clinical trial that included 711 patients with dementia and/or Parkinson's disease who aspirated thin liquids was recently reported. During a radiographic video fluoroscopic evaluation, chin tuck alone demonstrated 32% efficacy preventing thin liquid aspiration compared with 37% for "nectar" (tomato juice) thick liquids and 47% for "honey" thick liquids.[66] The speed of bolus movement is reduced as liquids become thicker allowing some patients to better control the bolus. During a subsequent 3-month randomized intervention of 504 patients, pneumonia occurred in 15% of those randomized to "honey" thick liquids, 8% in the "nectar" thick group, and 9% in the chin tuck group. The results were not statistically significant. The trial was terminated on the basis of a futility analysis. Of those who developed pneumonia, median length of hospital stay was 18 days in those drinking "honey" thick liquids, 6 days in those assigned to chin tuck, and 4 days in those drinking "nectar" thick liquids. Thickened liquids were associated with dehydration in 6% compared with 2% in the group randomized to chin tuck. The authors state that in addition to the possibility of being unpalatable and diminishing quality of life, that thickened liquids require greater strength to propel and could be more difficult to clear if aspirated. The cost of 1 month of thickened liquids is approximately $200.[68,69] In addition, another author contends that the chin tuck maneuver could decrease the higher contraction pressures needed to swallow a thick bolus and be a problem in patients with muscle weakness such as amyotrophic lateral sclerosis.[67]

In 1999, the Centers for Medicare and Medicaid Services (CMS) reviewed the efficacy of swallowing evaluations applied during acute cerebrovascular accident (CVA). "Treatment may yield dramatic reductions in pneumonia rates based on historical studies. The magnitude is difficult to determine. However, the effects are substantial and must be taken as evidence of efficacy,"[70] This conclusion applies to a situation in which temporary restriction of oral intake may be followed by rapid recovery of swallowing function.

The application of speech pathology swallowing intervention in NH residents is controversial. In my opinion, considering consultation should be a standard of care especially in acute CVA, patients with deterioration during acute reversible medical illness, and patients who can follow instructions and perform therapeutic exercise. Therapeutic lingual exercise has been followed by reduced aspiration.[59]

The lessons learned during the previously cited randomized studies should be familiar to clinicians during trials of chin tuck or thickened liquids. Trials of a swallowing intervention might also focus on hydration, nutritional status, and the discomfort of aspiration events.

Compensatory swallowing interventions (chin tuck, thickened liquids) are not likely to improve swallowing secretions between meals, a time when a large proportion of infected secretions may be aspirated. Possible between-meal interventions include positioning on the side to drain the mouth (cardiopulmonary resuscitation [CPR] recovery position) and to avoid hyperextension of the neck (a significant risk factor in one study).[71] Oral suction may be useful in some residents. Five trials of subglottic suction in intubated patients showed a risk reduction to 0.51.[72]

A number of medical therapies have been proposed to prevent pharyngeal aspiration. Angiotensin-converting enzyme (ACE) inhibitors increase Substance P, a neurotransmitter implicated in cough and swallowing.[60,75] A randomized 4-year trial of the ACE inhibitor, perindopril, in patients with prior stroke or transient ischemic attack, failed to prevent pneumonia. However, when the Asian population (n = 2300) was analyzed separately, pneumonia was reduced 47%.[76] An excellent review of this topic was recently published.[75]

CLEARANCE OF ASPIRATED SECRETIONS: COUGH AND MOBILITY

Residents with oropharyngeal dysphagia may have associated deficits that interfere with clearing secretions following tracheal aspiration, including cough and mobility impairment. Patients with Parkinson's disease or those with aspiration pneumonia demonstrated decreased frequency of involuntary swallowing (based on observation and electro myogram), cough reflex sensitivity (irritant aerosol), and peak flow with cough.[60–63] The cough reflex may also be depressed following stroke or in the presence of diabetic autonomic neuropathy.[64] Like swallowing, cough is a complex motor act that requires a deep inspiration followed by a forced expiration initially against a closed glottis. It is likely that many individuals with oropharyngeal dysphagia also have impairment of the cough

mechanism. This impairment requires greater consideration.[64] In one interesting study performed in the ICU, a combined assessment of cough peak flow (peak flow meter), volume of secretions, and mental status predicted the success of extubation.[65] Residents could be prompted to cough and take deep breaths. Early mobilization in community-acquired pneumonia reduced length of hospital stay 1.1 days.[74] A meta-analysis of "mobilization" in the ICU was conducted. Fifteen randomized trials of oscillating beds showed a risk reduction in the incidence of pneumonia to 0.38.[73]

ASPIRATION OF GASTRIC CONTENTS

GERD/regurgitation is acknowledged by pulmonary and allergy CPGs as a possible cause of cough, asthmatic bronchitis and sinusitis.[44,49,77,78] In one study, 35% of deaths attributed to GERD were from aspiration.[79] However a strong association between GERD and pneumonia is controversial. Aspirated gastric contents may contain food, acid, enzymes, or colonizing microbes. Gastric bacterial colonization by oral flora (including GNRs and *S aureus*) is facilitated by achlorhydria and stasis (gastroparesis, bowel obstruction). The stomach is probably an incubator for oral bacteria.[37,80] Proton pump inhibitors (PPI), H2 blockers, and antacids do not prevent reflux or aspiration.[81,82] Rather, they prevent the aspiration of acid, possibly replaced by pH-neutral regurgitated material colonized with bacteria.[37,83–85] In a case-control study, current PPI use increased the risk of pneumonia in outpatients 1.9 times versus those who stopped the PPI.[86]

Feeding tubes support the growth of biofilm. Israeli investigators sampled pharyngeal and gastric secretions in 54 percutaneous endoscopic gastrostomy and 52 NG-fed residents. Respiratory pathogens were isolated from the pharynx of approximately 60% and the gastric juice of approximately 50% in both groups. However those with NG tubes were significantly more likely to have gastric colonization with *Pseudomonas*, *Proteus*, *Escherichia coli*, and B-hemolytic strep. The concordance between oral and gastric isolation of pathogens was higher in the NG group. Pathogens were more likely to be isolated in the presence of neutral gastric pH.[87]

The best data on the bacteriology of aspiration pneumonia in NH residents is that of El-Solh et al.[88] Protected BAL was performed within 4 hours of any antibiotic in 95 residents with risk factors for pharyngeal aspiration or regurgitation. In 41, clinicians failed to isolate bacteria ($>10^3$ colony forming units/mL). These residents may have been affected by non bacterial pneumonitis (acid, enzymes, food). Some cases of aspiration "pneumonia" are initially noninfectious. Clinicians should reevaluate the response to antibiotics and consider discontinuing therapy after a food/acid aspiration event if there is rapid, complete recovery.[2,3,89,90] If a significant proportion of cases are noninfectious with rapid recovery, clinicians could get the impression that ineffective antibiotics were effective.

Delayed gastric emptying can exacerbate reflux/regurgitation. Gastrointestinal (GI) motility is neglected. The enteric system is the largest collection of nerve cells outside the central nervous system. Many of the pathologies that cause neuropathy or dementia, such as diabetes mellitus (DM) and Parkinson's disease, probably effect gastrointestinal motility. Rectal distention, DM, uremia, cirrhosis, and inflammatory cytokines slow gastric emptying.[91–94] Delayed gastric emptying occurs frequently in critically ill ICU patients. Critical illness should be expected to exacerbate or precipitate reflux.[95] In my experience, fecal impaction is associated with pneumonia.

PREVENTION/GASTRIC ASPIRATION

Reflux may be difficult to diagnose in the elderly. Only 24% of 175 patients older than 85 years with endoscopic reflux esophagitis gave a history of heartburn or acid regurgitation.[96]

Elevating the head of the bed 30 to 45 degrees decreased the rate of pneumonia by 77% in intubated patients with NG tubes.[97] Elevating the head of the bed is a standard of care in the ICU[98]; however, this position increases pressure applied to the sacral skin.[99] ICU clinicians may avoid gastric distention by monitoring gastric residual volume with an NG tube.[90,100] In the NH, gastroparesis may present as nausea, vomiting, regurgitation, abdominal distension, or a large gastric air bubble on chest x-ray.

Japanese clinicians conducted small studies in NH residents to assess the effectiveness of interventions designed to reduce gastric regurgitation and aspiration. Twenty-eight bed-bound residents were placed in the sitting position for 2 hours after meals and compared with 34 controls. The intervention group sustained 13 febrile days per patient over the course of the 100-day study compared with 18 days in the control group ($P < .05$).[101] In a second study, PEG-fed residents with prior CVA were studied for 12 months. Thirty-eight received mosapride, a promotility agent, verses 37 controls. Forty-seven percent of the intervention group developed pneumonia compared with 81% in the control group ($P = .004$). In addition, mortality was 26% in the intervention group compared with 59% in controls ($P = .01$).[102]

The lungs, sinuses, and intestines have a shared entrance. Intensivists refer to this as the "aero-digestive tract." Perhaps humanity would be better served at the end of life if we had developed a dorsal blowhole like the dolphin to separate the respiratory tract from the biofilm-laden teeth and the GI tract?

PREVENTION/MEDICATION TAPER

The American Thoracic Society ICU Guideline recommends daily interruption or lightening of sedative agents to mobilize secretions.[98] Antipsychotics, sedatives, benzodiazepines, and anticholinergics have been identified as risk factors for aspiration pneumonia in elderly patients.[71,103–108] Obviously the risk/benefit of these medications must be considered before a monitored taper.

PULMONARY BIOFILM

Biofilm may form in the lungs in the presence of bronchiectasis in which bronchi are dilated, inflamed, and drain poorly. Causes include foreign body or gastric aspiration and necrotizing pneumonia, relatively common conditions in the NH.[109] Currently the diagnosis of bronchiectasis requires

Table 1. *Aspiration Pneumonia Prevention*

TAPER MEDS
Decrease salivation (slow bacterial clearance)
Impair swallowing
Facilitate reflux (relax lower esophageal sphincter, slow gastric emptying)
Promote gastric colonization (antacids, slow gastric emptying)
Depress / Weaken cough
Limit mobility
PREVENT ASPIRATION OF ORAL SECRETIONS / PROMOTE CLEARANCE
Swallowing interventions
Positioning
Suction
Mobilization
Treat bronchospasm
ORAL HYGIENE
Cleaning by hygienist
Electric brush?
Xylitol / Sorbitol /Antiseptic
ANTI-REFLUX THERAPY

high-resolution computed tomography (CT). In one series, 29% of 110 patients with suspected chronic obstructive pulmonary disease (COPD) (mean age 67) had bronchiectasis.[110] In addition, a subset of individuals with severe COPD developed chronic relapsing *Pseudomonas* infection. In one investigation, ongoing genetic drift toward resistance as well as biofilm formation was noted in isolates from these patients.[27] The frequency and extent of pulmonary biofilm in NH residents is unresolved. I would, however, encourage NH clinicians to consider chronic respiratory biofilm formation as a cause of relapsing *Pseudomonas*, *H influenzae*, or *S aureus* infection.[9]

CONCLUSION

Many residents are aspirating. Most have airway biofilm in dental plaque and possibly in sinusitis or bronchiectasis. Potential measures to prevent aspiration pneumonia are presented in Table 1. The selection of MDROs should be expected when oral antibiotics are administered repeatedly to individuals with recurrent aspiration and large collections of bacteria in biofilm without "Source Control."

Clinicians are encouraged to consider the "big stuff," including (1) large and/or recurrent aspiration events caused by 2 common motility disorders: oropharyngeal dysphagia and gastro esophageal reflux; (2) mechanical source control (debridement/drainage) of sites that may harbor large collections of bacteria protected from antibiotics in biofilm, including dental plaque, coated tongue, and possibly chronic sinusitis; and (3) Prevention of gastric bacterial colonization by maintaining emptying and acidity. However, aspirated gastric acid may also cause significant morbidity.

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