A SPIRATION PNEUMONIA IS BEST CONSIDERED NOT AS A DISTINCT ENTITY but as part of a continuum that also includes community- and hospital-acquired pneumonias. It is estimated that aspiration pneumonia accounts for 5 to 15% of cases of community-acquired pneumonia, but figures for hospital-acquired pneumonia are unavailable. Robust diagnostic criteria for aspiration pneumonia are lacking, and as a result, studies of this disorder include heterogeneous patient populations.

Aspiration of small amounts of oropharyngeal secretions is normal in healthy persons during sleep, yet microaspiration is also the major pathogenetic mechanism of most pneumonias. Large-volume aspiration (macroaspiration) of colonized oropharyngeal or upper gastrointestinal contents is the sine qua non of aspiration pneumonia. Variables affecting patient presentation and disease management include bacterial virulence, the risk of repeated events, and the site of acquisition (nursing home, hospital, or community). According to this spectrum, patients labeled as having aspiration pneumonia usually represent a clinical phenotype with risk factors for macroaspiration and involvement of characteristic anatomical pulmonary locations. Aspiration syndromes may involve the airways or pulmonary parenchyma, resulting in a variety of clinical presentations.

This review focuses on aspiration involving the lung parenchyma, primarily aspiration pneumonia and chemical pneumonitis. Aspiration of noninfectious material such as blood or a foreign body is also important. Aspiration pneumonia is an infection caused by specific microorganisms, whereas chemical pneumonitis is an inflammatory reaction to irritative gastric contents. Our understanding of the interaction between bacteria and the lung has improved. We examine this improvement, along with changing concepts of the microbiology and pathogenesis of aspiration pneumonia. We also examine the clinical features, diagnosis, treatment, and prevention of both aspiration pneumonia and chemical pneumonitis, as well as the risk factors.

CHANGING MICROBIOLOGIC AND PATHOGENETIC CONCEPTS OF ASPIRATION PNEUMONIA

Our understanding of normal lower-airway microbiota in humans has evolved with the use of targeted polymerase-chain-reaction studies, sequencing of bacterial 16S ribosomal RNA genes, and metagenomics. A recent study of oral microbiota in patients with acute stroke identified 103 different bacterial phylotypes, 29 of which had not been reported previously. Whether these new microbes are pathogens is uncertain.

The Human Microbiome Project has helped define the role that intestinal microorganisms play in the development of mucosal immunity and in the interplay between health and disease. Studies of the lung microbiome have challenged our
assumptions of lung sterility and of bacterial access to the lungs through aspiration (microaspiration or macroaspiration) and inhalation. Specifically, genomic methods have defined a complex taxonomic landscape of bacteria in the lung and revealed the presence of diverse communities of microbiota. We are still learning about the role of the microbiome in health and disease, as well as in the pathogenesis of pneumonia. In the healthy state, the immune tone of the airways and alveoli appears to be calibrated by the bacteria constituting the lung microbiota, an observation recently reported in both healthy humans and experimental animal models. Concepts of virulence have also changed; virulence is defined as "the relative capacity of a microorganism to cause damage to a host." Infection is not simply the result of bacterial replication or of bacterial gene products; it is also a consequence of the host response, resultant inflammation, and tissue damage.

Models proposed to explain the possible role of the lung microbiome in pneumonia include the adapted island model of lung biogeography, effects of environmental gradients on lung microbiota (e.g., regional differences in oxygen tension and nutrient availability in the lungs), and finally, pneumonia as an emerging phenomenon in the complex adaptive system of the lung microbial ecosystem. The stability of the lung microbiome is probably maintained by a balance of immigration and elimination of bacteria and by feedback loops. Immigration involves bacterial movement from the oropharynx to the lung primarily by means of microaspiration, and elimination mainly occurs through ciliary clearance and coughing. Negative and positive feedback loops can suppress or magnify signals, respectively, such as those for bacterial growth. An inflammatory event may lead to epithelial and endothelial injury, creating a positive feedback loop that can promote inflammation, disrupt bacterial homeostasis, and increase susceptibility to infection. The complex adaptive system model suggests that acute bacterial pneumonia results from enhancement of a growth-promoting signal by a positive feedback loop. This may result in a rapid shift from a diverse microbial mixture to dominance by a single species (e.g., Streptococcus pneumoniae or Pseudomonas aeruginosa). Various signaling molecules in humans, including neurotransmitters, cytokines, and hormones such as glucocorticoids, have been shown in vitro to promote the growth of S. pneumoniae and certain gram-negative rods.

One hypothesis linking the airway microbiome with aspiration pneumonia is that illness may result in a change in the lung microbiota (dysbiosis), which may, in turn, interfere with or impair pulmonary defenses. A macroaspiration event, particularly in a patient with risk factors for impaired bacterial elimination, such as reduced consciousness or an impaired cough reflex, could then overwhelm the elimination side of the immigration–elimination balance, further disrupting bacterial homeostasis and triggering an increase in a positive feedback loop leading to acute infection.

Bacteria may colonize various sites in the human oral cavity, such as the gingiva, dental plaque, and tongue. Pathogenic bacteria, including gram-negative species that are not seen in the normal host, may emerge in the elderly, as well as in patients in nursing homes or hospitals and those with nasogastric tubes. Cleavage of cellsurface fibronectin exposes receptors for gram-negative rods on underlying airway epithelial cells and is more closely correlated with host factors such as acute illness than with the site of care.

In the 1970s, anaerobes with or without aerobes were the predominant pathogens in aspiration pneumonia. More recently, there has been a shift to bacteria usually associated with community- and hospital-acquired pneumonias, and anaerobes are recovered less frequently. One study of aspiration pneumonia in patients in the intensive care unit showed that in community-acquired cases, the main isolates were S. pneumoniae, Staphylococcus aureus, Haemophilus influenzae, and Enterobacteriaceae, whereas gram-negative bacilli, including P. aeruginosa, were found without anaerobes in hospital-acquired cases. Another study assessed the incidence of anaerobic bacteria in patients with ventilator-associated pneumonia and in those with aspiration pneumonia. Bacterial pneumonia was diagnosed in 63 patients with ventilator-associated pneumonia and in 12 patients with aspiration pneumonia. Among patients with aspiration pneumonia, enteric gram-negative organisms were isolated in the patients with gastrointestinal disorders, whereas S. pneumoniae and H. influenzae predominated in those with community-acquired aspiration events. Only one anaerobic organism was found, and...
the authors questioned the need for anaerobic coverage in both ventilator-associated pneumonia and aspiration pneumonia.28

Studies of the elderly continue to show the trend away from anaerobes. A study involving 95 institutionalized elderly patients with severe aspiration pneumonia reported 67 pathogens.29 Gram-negative enteric bacteria accounted for 49% of the pathogens, anaerobes for 16%, and S. aureus for 12%. Aerobic gram-negative bacteria were found in conjunction with 55% of anaerobic isolates. Another study, involving 62 elderly hospitalized patients with aspiration pneumonia, showed that of 111 bacteria identified, gram-negative bacilli and anaerobes each accounted for 19.8% of the bacteria, and anaerobes and aerobes together were found in 66.7% of patients who died.30 It is unclear why the pathogens have changed, but it may be due to a shift in the demographic characteristics of patients and earlier sampling today than in the past. Prior studies often collected cultures later in the illness, often after the development of empyema or lung abscess.1 This discussion does not apply to chemical pneumonitis, which unlike aspiration pneumonia, is a noninfectious, inflammatory response of the airways and pulmonary parenchyma to acidic gastric contents or bile acids.31

Aspiration Pneumonia

Aspiration Pneumonia

Aspirations of food, liquid, or saliva into the respiratory tract is a common event that occurs in 1% to 3% of the population.32

Aspiration pneumonia is a noninfectious, inflammatory response of the airways and pulmonary parenchyma to acidic gastric contents or bile acids.31

Risk Factors

Aspiration is often the result of impaired swallowing, which allows oral or gastric contents, or both, to enter the lung, especially in patients who also have an ineffective cough reflex (Fig. 1). Large-volume aspiration occurs with dysphagia; head, neck, and esophageal cancer; esophageal strictures and motility disorders; chronic obstructive pulmonary disease; and seizures.1,3,32-41 In a case–control study involving elderly patients with pneumonia and healthy elderly controls, oropharyngeal dysphagia increased the risk of pneumonia (odds ratio, 11.9) and was present in nearly 92% of the patients who had pneumonia. Results of videofluoroscopic evaluation showed that 16.7% of the patients with pneumonia were able to swallow safely, as compared with 80% of the controls.32 In extubated survivors of respiratory failure, dysphagia and aspiration are identified in at least 20% of patients. The frequency of swallowing dysfunction declines over time, but up to 35% of patients with swallowing dysfunction at the time of extubation continue to have this problem at the time of discharge.33

Additional risks include degenerative neurologic diseases (multiple sclerosis, parkinsonism, and dementia) and impaired consciousness, particularly as a result of stroke and intracerebral hemorrhage, which can also impair cough clearance. The frequency of stroke-associated pneumonia is related to the severity of neurologic illness and its associated immune impairment, with higher rates among patients requiring intensive care than among those admitted to a stroke unit.34 Impaired consciousness can also result from drug overdose and medications, including narcotic agents, general anesthetic agents, certain antidepressant agents, and alcohol (Fig. 2A). After adjustment for other risk factors, antipsychotic medications increased the risk of aspiration pneumonia by a factor of 1.5 in a study involving 146,552 hospitalized patients.35 Enteral feeding can lead to high-volume aspiration, especially when associated with gastric dysmotility, poor cough, and altered mental status. In three studies of enteral feeding after a stroke in a total of more than 5000 patients, early tube feeding improved survival, as compared with no feeding, and in the first 2 to 3 weeks after the stroke, nasogastric tube feeding was associated with improved survival and functional outcomes, as compared with percutaneous enteral tube feeding.36 Enteral feeding tubes are not currently recommended for patients with dementia.37

Patients with multiple risks have increased rates of aspiration pneumonia, death, and other adverse outcomes. A meta-analysis of studies involving frail elderly patients showed that dysphagia increased the odds ratio for aspiration pneumonia by a factor of 9.4, but when cerebrovascular disease was also present, the odds ratio rose to 12.9.38 In a study involving 1348 patients with community-acquired pneumonia, 13.8% of the patients were considered to be at risk for aspiration, and this subgroup of patients had a higher 1-year mortality (hazard ratio, 1.73) and increased risks of recurrent pneumonia (hazard ratio, 3.13) and rehospitalization (hazard ratio, 1.52) as compared with the rest of the study population.39 Similarly, a study involving 322 patients with community-acquired pneumonia identified the important risk factors for aspiration pneumonia as dementia (odds ratio, 5.20), poor performance status (odds ratio, 3.31), and

February 14, 2019

The New England Journal of Medicine

Downloaded from nejm.org by MAX SOLANO on February 22, 2019. For personal use only. No other uses without permission.
Copyright © 2019 Massachusetts Medical Society. All rights reserved.
The New England Journal of Medicine

Pathogenesis and risk factors for the development of pneumonia after macroaspiration

**Risk Factors**

**Impaired swallowing**
- Esophageal disease: dysphagia, cancer, stricture
- Chronic obstructive pulmonary disease
- Neurologic diseases: seizures, multiple sclerosis, parkinsonism, stroke, dementia
- Mechanical ventilation extubation

**Impaired consciousness**
- Neurologic disease: stroke
- Cardiac arrest
- Medications
- General anesthesia
- Alcohol consumption

**Increased chance of gastric contents reaching the lung**
- Reflux
- Tube feeding

**Impaired cough reflex**
- Medications
- Alcohol
- Stroke
- Dementia
- Degenerative neurologic disease
- Impaired consciousness

**Figure 1. Pathogenesis of and Risk Factors for Pneumonia after Macroaspiration.**

Macroaspiration can occur as a result of abnormalities in the swallowing mechanism or altered swallowing due to dysfunction of the central nervous system. In patients with these disorders, oropharyngeal or gastric contents can enter the lung. An impaired cough reflex increases the likelihood that aspirated material will reach the lung. Shown are the disease processes that serve as risk factors for macroaspiration by impairing consciousness, swallowing, and cough and by increasing the chance that gastric contents will reach the lung.

The use of sleeping pills (odds ratio, 2.08). Those with two or more risk factors had an increased incidence of recurrent pneumonia and increased 30-day and 6-month mortality, with rates rising in parallel with the number of risk factors. The relationship between distinct risk factors for macroaspiration and the frequency and outcome of aspiration pneumonia underscores the difference between aspiration pneumonia and traditional community-acquired pneumonia: patients with traditional community-acquired pneumonia have no associated increase in the risk of aspiration.

An important clinical context for aspiration pneumonia is cardiac arrest. In a study involving 641 patients with cardiac arrest, pneumonia developed within 3 days after the event in 65% of the patients. The presumed mechanism is aspiration of gastric contents during resuscitation (promoted by stomach ventilation and the resuscitation procedure) and inhalation of oral secretions during bag–valve–mask ventilation and intubation. When therapeutic hypothermia to 33°C was used after cardiac arrest, the odds ratio for early-onset pneumonia rose by a factor of 1.9. However, a target temperature of 36°C may be associated with a lower risk of pneumonia. Some studies showed that the incidence of early—
Aspiration pneumonia decreased among patients receiving systemic antibiotics at the time of cardiac arrest.43 Two intervention studies involving comatose patients showed a benefit of administering prophylactic antibiotics for up to 24 hours after emergency intubation.44,45

**Clinical Features**

Although macroaspiration is an essential feature of aspiration pneumonia and chemical pneumonitis, many episodes are unwitnessed; therefore, the magnitude of the exposure is often unknown. Clinical features range from no symptoms to severe distress with respiratory failure, and the clinical consequences may develop acutely, subacutely, or slowly and progressively. Aspiration into the lung can affect either the airway (causing bronchospasm, asthma, and chronic cough) or the lung parenchyma. This discussion focuses on aspiration into the lung parenchyma, which can take the form of aspiration resulting in chemical pneumonitis, aspiration of bland material (blood or the contents of tube feeding), or aspiration resulting in bacterial pneumonia.

Aspiration pneumonia is usually acute, with
symptoms developing within hours to a few days after a sentinel event, although anaerobic aspiration may be subacute because of the less virulent bacteria, and clinical features are difficult to distinguish from those of other bacterial pneumonias. In a study involving patients with pneumonia who were more than 80 years of age, those with aspiration had a higher mortality, higher serum sodium levels, and worse renal function than patients without aspiration.46 In a group of 53 patients with pneumonia and fluoroscopically documented dysphagia, more patients had bronchopneumonia than lobar pneumonia (68% vs. 15%), and 92% had posterior infiltrates.47 Most patients with poor performance status had diffuse and not focal infiltrates. Aspiration pneumonia is associated with higher mortality than other forms of pneumonia acquired in the community (29.4% vs. 11.6%), a finding that may have implications for hospitals that do not properly code its presence.48 In a survey of more than 1 million patients in more than 4200 hospitals, aspiration was documented in 4 to 26% of episodes of pneumonia. The expected mortality among patients with aspiration pneumonia is higher than that for other forms of pneumonia, and the risk-adjusted mortality (now used as a quality metric) is lower for hospitals reporting a high frequency of aspiration than for hospitals reporting a low frequency of aspiration.48

Macroaspiration of gastric contents can lead to chemical pneumonitis but only with large-volume, low-pH (usually <2.5) aspiration. In animal models, chemical pneumonitis develops only after exposure to at least 120 ml of gastric contents with a pH of 1. Described by Mendelson in 1946 as a consequence of obstetrical anesthesia, chemical pneumonitis is uncommon with modern anesthesia methods (1 case per 3216 procedures), with a higher risk during emergency surgery and a lower risk with elective procedures.49 In up to 64% of patients with aspiration during anesthesia, clinical or radiographic abnormalities do not develop. Lung injury from acid aspiration is due to the release of inflammatory mediators, including chemokines (e.g., interleukin-8), proinflammatory cytokines (e.g., tumor necrosis factor), and neutrophil recruitment.50

Chemical pneumonitis is characterized by a sudden onset of dyspnea, hypoxemia, tachycardia, and diffuse wheezes or crackles on examination. A chest radiograph is usually abnormal, and a pattern that is characteristic of acute respiratory distress syndrome develops in up to 16.5% of patients with witnessed aspiration, although the frequency rises if other risk factors (shock, trauma, or pancreatitis) are also present51 (Fig. 2B). Low-pH aspirates are usually sterile, and bacterial infection is unusual initially, although superinfection may develop subsequently.

In most cases, neither chemical pneumonitis nor aspiration pneumonia occurs with tube feedings or aspirated blood, since the aspirate pH is usually high and uncontaminated by bacteria. Although acid-suppressing therapy is associated with an increased risk of community- or hospital-acquired pneumonia, which is related to gastric overgrowth by gram-negative bacteria, neutralization of gastric pH may reduce the risk of chemical pneumonitis.52 In a prospective cohort study involving 255 patients undergoing gastrointestinal endoscopy, the use of proton-pump inhibitors or histamine H2 blockers was associated with a significant reduction in the risk of gastric contents with a pH of <2.5 (odds ratio, 0.24).53 Asphyxia may result if the aspirated volume of the bland material is large, but there may be few clinical findings if the volume is small. The chest radiograph may initially be abnormal, until the aspirated material is cleared by suction or coughing. In cases of unwitnessed aspiration, it may be difficult to distinguish among chemical pneumonitis, aspiration pneumonia, and aspiration of bland material. An aspirated solid foreign body can obstruct the airway and lead to postobstructive pneumonia, further complicating the distinction from bacterial pneumonia. In a series of patients with foreign-body aspiration who were more than 65 years of age, the event was recognized in only 29% of the patients, leading to a diagnostic delay of 1 to 3 months.54 Chest radiographic findings were in the right lung in 65% of the patients, and food material accounted for more than 80% of the episodes.

**DIAGNOSIS**

The diagnosis of aspiration pneumonia depends on a characteristic clinical history (witnessed macroaspiration), risk factors, and compatible findings on chest radiography. These radiographic findings include infiltrates in gravity-dependent lung segments (superior lower-lobe or posterior upper-lobe segments, if the patient is in a supine
Aspiration pneumonia was confirmed on computed tomography.55 One negative in 28% of patients in whom pneumonia was isolated in 29.6% of patients with only lung abscess. In those with only aspiration pneumonia, anaerobes were not found, and the most frequent aerobes were Escherichia coli, Klebsiella pneumoniae, and P. aeruginosa (Fig. 2C).

Antibiotic selection depends on the site of acquisition (the community, a hospital, or a long-term care facility) and risk factors for infection with multidrug-resistant pathogens. Risk factors include treatment with broad-spectrum antibiotics in the past 90 days and hospitalization for at least 5 days. For most patients with community-acquired cases, treatment with ampicillin–sulbactam, a carbapenem (ertapenem), or a fluoroquinolone (levofloxacin or moxifloxacin) is effective.3 In such patients, we suggest adding clindamycin to another drug only when the risk of predominantly anaerobic infection is high, as it is for patients with severe periodontal disease and necrotizing pneumonia or lung abscess1 (Fig. 3 and Table 1). With mixed infection, elimination of aerobic pathogens usually alters the local redox potential, eliminating anaerobes. For hospital-acquired cases with a low risk of multidrug-resistant pathogens, a similar regimen may be used. If resistance is a concern, broader-spectrum treatment with piperacillin–tazobactam, cefepime, levofloxacin, imipenem, or meropenem, either singly or in combination, is required67 (Fig. 3 and Table 1). In cases of multidrug-resistant infection, an aminoglycoside or colistin may be used as part of a combination regimen, with the addition of vancomycin or linezolid if the patient has documented nasal or respiratory colonization with methicillin-resistant S. aureus.

In a recent study involving comatose, mechanically ventilated patients with aspiration, 43 of the 92 patients (46.7%) had bacterial aspiration pneumonia on the basis of bronchoscopic brush...
Figure 3. An Algorithmic Approach to Antibiotic Therapy for Aspiration Pneumonia.

For patients with suspected aspiration pneumonia, the decision about antibiotic therapy is dictated by the site of acquisition: community, hospital, or long-term care facility. Antibiotics are given to patients who have an aspiration event and an abnormal chest radiograph, although even with an initially normal radiograph, antibiotics are given to those with severe illness (i.e., illness characterized by shock or requiring intubation). If chemical pneumonitis is suspected, antibiotics are not initially recommended, even with an abnormal radiograph, unless the patient is severely ill; in patients with mild-to-moderate illness and an abnormal radiograph, the recommendation is to withhold antibiotics and reassess the patient in 48 hours. Therapy is directed at the pathogens likely to be present in the patient at the time of aspiration, determined on the basis of the site of acquisition, with risk factors for resistant pathogens taken into consideration. Patients with severe illness are treated on the basis of risks associated with their dental health and multidrug resistance (MDR). Routine treatment for anaerobic pathogens is not needed in patients with normal dental health but should be considered in those with poor dental health (e.g., clindamycin in patients with poor dental health and necrotizing pneumonia or lung abscess). If the site of acquisition is the community, outpatient treatment with amoxicillin–clavulanate, moxifloxacin, or clindamycin can be administered orally. In the hospital, treatment is usually administered intravenously, but oral options are available for select patients; if there is nasal or respiratory colonization with methicillin-resistant S. aureus, the addition of vancomycin or linezolid can be considered. BAL denotes bronchoalveolar lavage.
samples. The investigators suggested that routine antibiotic treatment should be started only if bacterial infection is suspected but may be discontinued if bronchoscopic cultures are negative.

On the basis of data from studies of community-acquired pneumonia and hospital-acquired or ventilator-associated pneumonia, we suggest 5 to 7 days of treatment for patients with a good clinical response and no evidence of extrapulmonary infection, and longer treatment for those with necrotizing pneumonia, lung abscess, or empyema. In the case of lung abscess or empyema, drainage for diagnostic and treatment purposes may be needed. The choice of therapy should take into account potential drug-related adverse events, including Clostridium difficile colitis and selection of antibiotic resistance. More data are needed to identify the best antibiotic regimens for aspiration pneumonia and to determine the duration of treatment. No randomized, controlled trials have shown a role for glucocorticoids in the routine treatment of aspiration pneumonia, and we do not recommend their use.

**CHEMICAL PNEUMONITIS**

Initial treatment of gastric aspiration requires airway maintenance, management of airway edema or bronchospasm, and minimization of tissue damage. Depending on the severity of the pneumonitis and the extent of care required, treatment may include suctioning, bronchoscopy, intubation, mechanical ventilation, and intensive care. Routine adjunctive treatment with glucocorticoids is not recommended, and antibiotics are not needed routinely unless the patient is taking acid-suppressing medication or has small-bowel obstruction. In mild-to-moderate cases, we recommend withholding antibiotics even if there is radiographic evidence of an infiltrate, monitoring clinical and radiographic findings, and reassessing after 48 hours. In more serious cases, however, antibiotics should be started empirically, and the decision to continue antibiotic therapy for more than 2 to 3 days should be guided by the clinical course.

**PREVENTION**

Postoperative chemical pneumonitis can be minimized by ensuring that the patient has fasted for at least 8 hours and has abstained from clear liquids for at least 2 hours before surgery is performed (Table 2). Medications known to promote aspiration and interfere with swallowing should be avoided, including sedatives, antipsychotic agents, and for some at-risk patients, antihistamines. Aspiration-prevention efforts focusing on ventilator-associated pneumonia are not discussed here.

For patients with swallowing disorders, particularly after stroke, a full speech and swallowing evaluation is necessary. Efforts should be made to promote oral rather than enteral tube feeding, with the use of a mechanical soft diet with thickened liquids rather than pureed food and thin liquids. In addition, “nutritional rehabilitation” with swallowing exercises and early mobilization may help patients with dysphagia and may prevent recurrence of aspiration pneumonia. Patients should receive enteral feeding in a semirecumbent rather than supine position to minimize the risk of gastric aspiration. For patients with oropharyngeal dysphagia, an effort

### Table 1. Antibiotic Treatment of Aspiration Pneumonia

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose, Schedule, and Route of Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin–sulbactam</td>
<td>1.5–3 g every 6 hr, intravenous</td>
</tr>
<tr>
<td>Amoxicillin–clavulanate</td>
<td>875 mg twice daily, oral</td>
</tr>
<tr>
<td>Piperacillin–tazobactam</td>
<td>4.5 g every 8 hr or 3.375 g every 6 hr, intravenous</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>1–2 g once daily, intravenous</td>
</tr>
<tr>
<td>Cefepime</td>
<td>2 g every 8–12 hr, intravenous</td>
</tr>
<tr>
<td>Ertapenem</td>
<td>1 g once daily, intravenous</td>
</tr>
<tr>
<td>Imipenem</td>
<td>500 mg every 6 hr or 1 g every 8 hr, intravenous</td>
</tr>
<tr>
<td>Meropenem</td>
<td>1 g every 8 hr, intravenous</td>
</tr>
<tr>
<td>Levofoxacin</td>
<td>750 mg once daily, intravenous or oral</td>
</tr>
<tr>
<td>Moxifloxacin</td>
<td>400 mg once daily, intravenous or oral</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>450 mg three or four times daily, oral; or 600 mg every 8 hr, intravenous</td>
</tr>
<tr>
<td>Gentamicin or tobramycin†</td>
<td>5–7 mg/kg once daily, intravenous</td>
</tr>
<tr>
<td>Amikacin†</td>
<td>15 mg/kg once daily, intravenous</td>
</tr>
<tr>
<td>Colistin‡</td>
<td>9 million IU per day in two or three divided doses, intravenous</td>
</tr>
<tr>
<td>Vancomycin†</td>
<td>15 mg/kg every 12 hr, intravenous</td>
</tr>
<tr>
<td>Linezolid</td>
<td>600 mg every 12 hr, intravenous or oral</td>
</tr>
</tbody>
</table>

* Doses are for patients with normal renal function.
† The dose should be adjusted to a trough level of less than 1 mg per liter for gentamicin and tobramycin, a trough level of less than 4 mg per liter for amikacin, and a trough level of 10 to 15 μg per milliliter for vancomycin, with renal function taken into consideration in all cases.
‡ A loading dose of 6 million to 9 million IU can be administered.
Table 2. Prevention of Aspiration Pneumonia.

<table>
<thead>
<tr>
<th>Recommended in the appropriate clinical setting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antibiotic therapy for 24 hr in comatose patients after emergency intubation</td>
</tr>
<tr>
<td>No food for at least 8 hr and no clear liquids for at least 2 hr before elective surgery with general anesthesia</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>To be considered in the appropriate clinical setting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swallowing evaluation after stroke and after extubation from mechanical ventilation</td>
</tr>
<tr>
<td>Preference for angiotensin-converting–enzyme inhibitors for blood-pressure control after stroke</td>
</tr>
<tr>
<td>Oral care with brushing and removal of poorly maintained teeth</td>
</tr>
<tr>
<td>Feeding in a semirecumbent position for patients with stroke</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Not yet recommended; more data needed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swallowing exercises for patients with dysphagia after stroke</td>
</tr>
<tr>
<td>Oral chlorhexidine in patients at risk for aspiration</td>
</tr>
</tbody>
</table>

A meta-analysis of five randomized, controlled trials involving nonventilated patients at risk for aspiration pneumonia showed that oral care with chlorhexidine or mechanical oral cleaning was effective in preventing pneumonia (odds ratio, 0.4 to 0.6). However, chlorhexidine use is controversial and may be associated with increased mortality among ventilated patients, possibly as a result of toxic effects if chlorhexidine is aspirated into the lung. In a randomized study involving 252 patients, supplemental nutrition plus daily oral cleaning reduced the frequency of pneumonia (7.8%, vs. 17.7% with usual care; P=0.06). In a case–control study involving 599 patients undergoing surgery for esophageal cancer, postoperative pneumonia developed in 19.1% of the patients. Lack of preoperative oral care, including tooth scaling, mechanical cleaning, and tooth extraction if necessary, was an important risk factor. Despite these promising findings, a cluster-randomized study involving 834 nursing home patients, with a mean observation time of slightly more than 1 year, showed no benefit of a comprehensive oral care program, which included manual tooth and gum brushing, chlorhexidine mouth washes, and upright positioning during feeding, with radiographic evidence of the development of pneumonia in 25% of the patients.

Antibiotic administration for up to 24 hours in comatose patients who have been intubated on an emergency basis has shown a benefit in two trials. An open, randomized, controlled study involving 100 intubated, comatose patients with stroke or head injury showed that 1.5 g of cefuroxime given every 12 hours for two doses reduced the occurrence of pneumonia, particularly early-onset pneumonia. Control patients receiving antibiotics at the time of intubation had lower pneumonia rates than those not receiving antibiotics. A subsequent cohort study showed that a single dose of antibiotic (ceftriaxone or ertapenem) administered within 4 hours after intubation was effective in preventing early-onset but not late-onset pneumonia in comatose patients, including the 25% of patients who were intubated on an emergency basis after cardiac arrest.

Conclusions

Aspiration pneumonia is an important illness that is difficult to accurately diagnose and to
Aspiration pneumonia is treated with antibiotics as required but not with glucocorticoids. Preventive measures should be used for patients at risk for aspiration.

REFERENCES

37. American Geriatrics Society Ethics Committee and Clinical Practice and Mod-


Copyright © 2019 Massachusetts Medical Society.