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The history of diarrheal disease in man weaves a colorful but morbid tapestry. Long before the United States existed as a nation, cholera and cholera-like disease decimated armies, deposed kings, and in India sparked a cult religious following in hopes of placating the disease's fearsome wrath.[1] In the United States during the 19th century, diarrheal illness grew and expanded alongside the fledgling nation. It was carried by the gold rush to California, and otherwise disseminated across the nation's midsection by way of the expanding canal and railroad systems.[1]

Even in the modern era, diarrheal disease of any cause remains a serious worldwide public health concern, resulting in almost 3 million deaths annually.[2] Children in the developing world are affected particularly hard, accounting for almost 2 million of these deaths, such that diarrhea is the second most common non-neonatal cause of death in children younger than 5 years, after respiratory illness.[3] This article reviews the pathophysiology, clinical presentation and supportive management strategies for rotavirus.

— The Editor

Rotavirus: An Update on Current Diagnosis and Management

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History

Despite its monumental importance, rotavirus was not known to be a human pathogen before 1973. On the heels of the discovery of the Norwalk virus in 1972 by other researchers,[4] Bishop and colleagues[5] identified idiotypical virus particles, mor-

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phologically distinct from the Norwalk virus, by electron microscopy of inflamed mucosa. Bishop later discovered that the agent was identified more easily in fecal material, and was the causative agent for as much as 81% of local cases of sporadic diarrhea.[6] A year after Bishop's discovery, Flewett and colleagues suggested the name 'rotavirus', based upon the virus' wheel-like morphology.[7] (*See Figure 1.*)

Scope of the Disease

Rotavirus is one of the most common causes of diarrheal disease. Worldwide it accounts for more than 125 million cases per year, 25% of all deaths due to diarrheal disease, and 6% of all deaths of children younger than 5 years.[8] Whereas, worldwide rotavirus causes as many as 600,000 pediatric deaths annually,[8] 82% of these deaths occur in developing countries.[9] In the United States, rotavirus-related gastrointestinal illness remains common, but the mortality rate is much lower (fewer than 40 deaths per year).[10] In the United States, the burden of rotavirus gastrointestinal disease in children is better measured in terms of the 500,000 outpatient visits,[11] 160,000 emergency department (ED) visits, and the 50,000 hospitalizations for which it is responsible.[10] In relative terms, this means that each year nearly 1 in 10 children younger than 5 years has an outpatient visit for rotavirus-related symptoms.[12] Based on estimates of $2,303 per hospitalization and $57 per outpatient visit[1] and other associated costs, the annual economic impact of rotavirus disease in children in the United States has been calculated to be in the $200 million range for this age group alone.[12-13]

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Taxonomy and Classification

Since the time of Bishop's discovery there has been much advancement in the taxonomy of this wheel-shaped virus. Currently, the rotavirus genus describes a group of double-stranded RNA viruses belonging to the *Reovirus* family. There are seven major groups of rotavirus, groups A through G. Variations within structural viral protein six (VP6) are responsible for the group designations. Groups A, B, and C, are thought to cause human illness. Group A is the rotavirus group most associated with endemic human gastrointestinal illness,[14,15] whereas groups B and C are currently thought to cause disease more regionally, or sporadically.

Strains of rotavirus can be further broken down by subgroup and serotype. The taxonomy is based upon the structural viral proteins composing the outer viral capsid. Variations in the VP6 protein determine not only group (A,B,C, others) but also subgroup (I or II).[15,16] In contrast, the serotype is determined by the specificities of other structural outer capsid proteins, VP4 (a protease sensitive protein) and VP7 (a glycoprotein).[15,16] The VP4 protein often is referred to as the "P" (for protease sensitive) serotype determinant. The VP7 protein often is referred to as the 'G' (for glycoprotein) serotype determinant.[15] These two determinants are important in the human immunologic response as evidenced by the production of neutralizing antibodies against these proteins.[17]

In theory, many different rotavirus group A strains could exist. In practice, the vast majority that cause human disease fall into one of six strains P[8]G1, P[4]G2, P[8]G3, P[8]G4, P[8]G9, and P[6]G9.[16,18] Which of these strains predominate varies geographically.[16,18]

Group B rotavirus gastrointestinal disease in humans, also called 'adult diarrhea rotavirus', has been most commonly reported in China, India, and Bangladesh.[19] However, occasional detection of antibodies to group B rotavirus in a global sampling of human sera indicates that sporadic disease is probably more widespread geographically.[20]

Group C rotavirus has been reported worldwide including in the United States, but in a sporadic fashion.[21] Limitations of the most commonly used assays may underestimate the disease burden related to this group.[4,21]

This article focuses on group A rotavirus, which is the group most likely to be seen by the practitioner in North America.

Epidemiology

Human infection with rotavirus is considered ubiquitous. Infection occurs at all age ranges, but severity of symptoms varies considerably by age.[22] When infected, neonates tend to be exposed through nosocomial avenues;[16] most other age groups

Figure 1. Human Rotavirus Seen Through Electron Microscopy

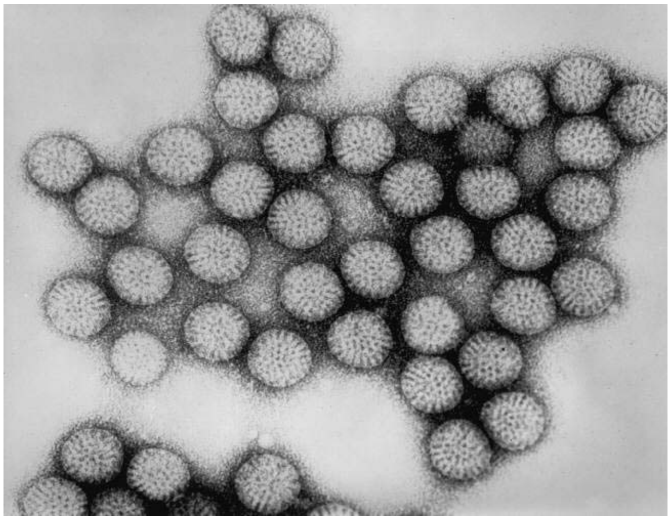

Figure 1. Transmission electron micrograph of intact rotavirus particles, double-shelled. Distinctive rim of radiating capsomeres.

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are infected by passage from infected family members and day-care peers.[22] Humans are first infected early in life, with nearly all children infected by 2 to 5 years of age.[16,22] Serum evidence points to repeated infection, at least with strains novel to the individual.[22]

In temperate climates there is marked seasonal variation in the incidence of rotavirus infection.[23,24] This variation seems to correlate best with temperature, rather than humidity, but is not present in tropical climates.[23] In North America—and the United States in particular—peak incidence of infection follows a repetitive pattern. Peak incidence in Mexico occurs in October to November. In the southwestern United States, November and December are peak months of incidence. As one moves geographically north and east, peak incidence occurs later and later in the year.

The resulting appearance is that of an annual epidemic that spreads across the United States from southwest to northeast during a three-to-four-month period.[25] It is tempting to think of this as an annual spread by contagious transmission, rather than regional recrudescence due to regional climatic changes, but this is not known. In the United States, rotavirus may go entirely undetected for several months during surveillance, suggesting that annual spread is by contagious transmission from region to contiguous region.

Worldwide the likelihood of seasonal variation increases with distance from the equator. There is a marked autumn to spring predilection far north and south of the equator, and there is less variability nearer the equator.[24]

Transmission

Rotavirus has been shown to be transmissible by the fecal oral route.[16,22] Large numbers of viral infectious particles (up to 10^{10} infectious particles per milliliter) are shed in the stool.[4,22] However, alternative mechanisms of transmission, such as aerosol transmission may be possible. While respiratory illness with rotavirus has been described,[26] another possible source of aerosolization is vomiting. Rotavirus has been isolated from emesis material in infected adults;[27,28] in one outbreak nosocomial spread was postulated to have occurred at least in part through aerosols elaborated in the process of vomiting.[27]The probable relative contributions to human transmission are not known, but currently it is believed that the human fecal–oral route is the primary mode of transmission.[16,22]

Nosocomial exposure represents an important source of infection for hospitalized children.[22] Rotavirus particles can survive for prolonged periods (>12 days) on materials commonly found in domestic and institutional settings. The survivability seems greatest at low humidity and on nonporous surfaces.[29] In a study screening environmental surfaces for rotavirus in a pediatric inpatient ward and ward playroom in Riyadh, Saudi Arabia, 7% of all tested surfaces were contaminated. Contaminated surfaces included toys, furniture, vital signs chart, toilet handles, patient hands, and sinks.[30] Rates of infection for children hospitalized with non-diarrheal illnesses range between 0.6% and 24%, depending upon the age of the child and the length of hospital stay.[31,35] Of interest, various disinfectants are effective at inactivating the virus on fomite surfaces, but tap water alone is not adequately effective.[33]

As humans are not the only rotavirus host, the potential of zoonotic spread of rotaviral infections to humans has been understood for many years. The greatest risk of zoonotic transmission is probably to farming communities and those persons in contact with livestock, but family pets including dogs and cats represent potential, if uncommon sources of infection as well.[34]

Clinical Manifestations

After oral exposure, the rotavirus must travel through the stomach to the intestinal villi to infect the host. The inhospitable gastric environment represents a significant barrier to infectivity. Inactivation occurs very quickly under experimental conditions, mimicking the normal fasting stomach (pH =2.0).[35] Inactivation was attenuated at pH=3.0, but was minimal at pH=4.0.[35] Pepsin also is believed to be an important factor in gastric barrier function.[36] The barrier effect of pH and 'pH and concentration-dependent factors' with gastric juice is such that under experimental conditions for the study of infectivity, bicarbonate administration is necessary to prevent rotavirus inactivation.[36,37] However, under these conditions in a porcine model, as little as a single plaque-forming unit of rotavirus is adequate to initiate infection.[37]

Rotavirus particles that are able to evade inactivation in the stomach infect the mature enterocytes in the mid and upper villi of the small intestine.[38] The VP4 protein appears to be important in this process of infectivity.

Once infected, there is a two- to four-day incubation period before clinical symptoms are manifest. Severity of gastrointestinal disease may be related in part to strain and electropherotype within strains, but individual and age-related differences are likely a manifestation of differences in acquired immunity.[26] Neonates are commonly asymptomatic or mildy symptomatic.[39] When symptoms do occur in neonates, watery stools are predominant, but bloody mucoid stools, abdominal distension, dilated bowel loops, and even frank necrotizing enterocolitis may be seen in premature neonates.[40] Adults may be asymptomatic as well. In a study of adult volunteers challenged with an oral rotavirus inoculum, 12 of 18 developed serologic evidence of infection, but only 4 (33%) of those with evidence of infection developed symptoms.[41]

In infants and children, acute gastroenteritis is the most common illness caused by rotavirus. Cross-sectional descriptive studies of rotavirus symptomology use diarrhea as entrance criteria, and stool testing is the most convenient method of determining infection. A recent survey of children seeking care for rotavirus diarrhea in Venezuela showed that 78% also had vomiting, 29% were dehydrated, and 30% had fever.[42] An older study of U.S. children who were hospitalized with rotavirus gastroenteritis showed a higher frequency of these findings: 96% had vomiting, 83% were dehydrated, 77% had fever, perhaps consistent with the more severe nature of their hospitalized status. Vomiting typically lasted 2 to 3 days, fever lasted 3 days, and diarrhea lasted an average of 5 days, but ranged from 1 to 9 days.[14] In addition to these typical gastrointestinal symptoms, the study also revealed extraintestinal symptoms: irritability in 47%, lethargy in 36%, pharyngeal erythema in 49%, rhinitis in 26%, tympanic erythema in 19%, and palpable cervical nodes in 18%.[14]

This original study of rotavirus symptomology has not been the only one to describe extraintestinal manifestations of rotavirus infection. Most concerning are the case reports related to the central nervous system (CNS). Case reports and series describe the recovery from cerebrospinal fluid of rotavirus antigen by enzyme immunoassay (EIA), RNA by polymerase chain reaction (PCR), or actual viral particles by electron microscopy. Clinical illness related to these cases involved aseptic meningitis, encephalitis, and seizures.[43] Rotavirus has been recovered from tracheal aspirates of children with clinical pneumonia[26] and from liver and kidney of children with immune deficiencies and diarrhea.[44] The route of exposure in these cases of extraintestinal disease is not known. Spread to other organ systems from the proximal small bowel is suspected to be by viremia. In some of the cases of CNS disease, rotavirus RNA was recoverable from the blood by PCR. In fact, subsequent investigation has revealed rotaviral antigenemia or viremia to be a common event, occurring in 43-66% of serum samples of children with rotavirus gastroenteritis.[45,46]

While interesting, CNS extraintestinal rotavirus disease is exceedingly rare. Acute gastroenteritis with manifestation of vomiting, diarrhea, fever, abdominal pain, and dehydration is the most common presentation. Although usually an acute disease, the illness can have more lasting adverse effects on growth and nutrition. This was first studied systematically in the 1970s but remains a problem.[47,48] Other complications of acute rotavirus gastroenteritis include hyponatremia, hypernatremia, metabolic acidosis, and diaper rash. Additionally, besides the more common acute gastroenteritis, the entity of 'chronic rotavirus diarrhea' has been reported in children with immunodeficiency.[44]

Necrotizing enterocolitis occurs in the setting of rotavirus in some neonates. While the cause of necrotizing enterocolitis is multifactorial, in small series rotavirus has been found to be present in 30-40% of cases.[50,51] The rotavirus-associated necrotizing enterocolitis tends to be milder and involve bowel more distally than the non-rotavirus associated necrotizing enterocolitis.[50,51] Finally, following the withdrawal of the original rotavirus vaccine, the possibility of the occurrence of intussusception in the setting of naturally occurring rotavirus infection was raised. However, epidemiologic data do not support any association of intussuception with naturally occurring or wild type rotavirus infection.[52,53]

Pathology and Pathophysiology

Little is known about the gross physical changes of the infected bowel in humans that may lead to the intestinal manifestations of disease described above. Ultrasound has revealed increased ileal wall thickness and less consistently mesenteric adenopathy.[54,55] More is understood on the histologic level. In Bishop's initial description of human rotavirus infection, all biopsy specimens revealed histologic abnormalities,[5] whereas a more recent prospective study revealed microscopic abnormalities in only 5% of biopsied specimens.[56] When histologic changes do occur, they are most notable in the mucosal surface. Villi become shortened, blunted, and appear atrophic. On higher power magnification, there is denudation of the microvilli, the lamina propria becomes infiltrated with mononuclear cells, and the enterocytes become more cuboidal.[5,38]

There is no information on gastric tissue derangement related to rotavirus, except for a single case report of gastric rupture occurring in a 3-month-old girl with vomiting and rotavirus identified in her stool.[57] Nevertheless, vomiting is a common symptom of rotavirus gastroenteritis, occurring in about 80% of those seeking care[42] and about 90% or greater in those hospitalized for rotavirus gastroenteritis.[14,58] Vomiting has been shown to be a dependable independent predictor of clinical dehydration in acute diarrhea of unspecified cause,[59,60] but this has not been studied for rotavirus specifically. The pathophysiology of vomiting in rotavirus acute gastroenteritis has not been fully established, but abnormal gastric motor function resulting in a delay of gastric emptying has been in shown in acute versus convalescent children.[61]

The pathophysiologic mechanisms responsible for diarrheal fluid loss are better elucidated. Derangements of several physiologic mechanisms have been found experimentally. Sodium-glucose co-transport and sodium-amino acid co-transport are inhibited during the conditions of rotaviral infection.[38] The activity of mucosal disaccharidases including sucrase, lactase, and maltase are diminished.[38] Net fluid and electrolyte secretion has been observed experimentally. Additionally, there is a reduction of sodium/potassium ATPase activity, although it is not certain if this is a direct effect on the transporter or an effect related to loss of transporter-bearing enterocytes.[38]

The known histologic and physiologic derangements point to several possible explanations for the fluid loss. The loss of absorptive villus surface area with the relative sparing of the fluid and electrolyte-secreting secretory crypts may result in an imbalance with net fluid loss.[38] A second possibility is that one of the nonstructural rotavirus proteins (NSP4) may function as a secretory viral enterotoxin. Alternatively, rotavirus infection may stimulate the enteric nervous system speeding the transit of intraluminal contents beyond the absorptive capacity. The stimulant might be the posited NSP4 enterotoxin, or as yet undetermined mechanisms.[38] Finally, some investigators have proposed a post-ischemic hyperemic recovery phase of intestinal disease.[62] This phase is characterized initially by hypersecretion by the rapidly dividing recovering enterocytes and secondly by a loss of hyperosmolality at the villous tips wrought by a hyperemia-disturbed countercurrent mechanism. The loss of the hyperosmolality then results in impaired water absorption from the villi.[62]

Diagnosis

Clinical Diagnosis. Multiple studies confirm that more than 80% of stool-positive cases of rotavirus diarrhea have vomiting and fever occurs in 30-65%. Additionally, there may be signs of dehydration and not uncommonly symptoms of respiratory infection.[11,42,63,64] The stool produced often is described as watery and free of blood.[64,65] However, the range of diarrhea includes cases whose clinical presentation (e.g., bloody or nonwatery) is more typical of invasive diarrheal syndromes.[66]

Rotavirus illness lasts 4 to 7 days in most cases, which is shorter than some other infectious causes of enteric illness.[66-67]

Age and seasonality can be helpful in trying to decide to include rotavirus in the differential, but definitive diagnosis cannot be made on these factors alone. Most cases are in infants and children age 6 months to 24 months, during peak months of December through March; however, infection can occur at any age, and in tropical climates there is no seasonal variation.[15,66,68,69]

Despite some of these defining features, the symptoms of rotavirus infection in children are not specific to this infectious agent. However, because treatment is supportive, in the right setting a presumptive diagnosis is adequate. Laboratory-aided diagnosis is often available.

Laboratory Diagnosis. Because it is not possible to diagnose rotavirus infection based upon clinical presentation, specific diagnostic tests were developed. Laboratory diagnosis requires using methods to detect rotavirus in stool and rectal swab specimens. Electron microscopy was the first detection method followed by enzyme-linked immunosorbent assay (ELISA) and later latex agglutination (LA) tests.[68] Other testing methods include radioimmunoassay, counterimmunoelectro-osmophoresis, tissue culture, polyacrylamide gel electrophoresis (PAGE), and polymerase chain reaction (PCR). Serum can be tested using acute and convalescent-phase antibody titers and demonstrating a fourfold or greater increase in antibody to rotavirus antigen. In addition, serologic evidence of rotavirus can be accomplished by ELISA immunofluroescence, neutralization, and complement fixation.[70]

Of all of these tests, only ELISA and LA have found their way to common clinical use. During their advent, published studies revealed sensitivity ranges from 70% to 90% and specificity ranges from 80% to 100%.[63,71,72] Modern ELISA-based chromatographic immunoassays are now the most commonly used; Specific commercial test kits often advertise sensitivities and specificities in the high 90-100% range, cost US$12-15 per kit, and can yield results in 10 to 15 minutes.[73-75] Some latex agglutination tests still are used and have similar performance characteristics, but are more labor intensive.[76]

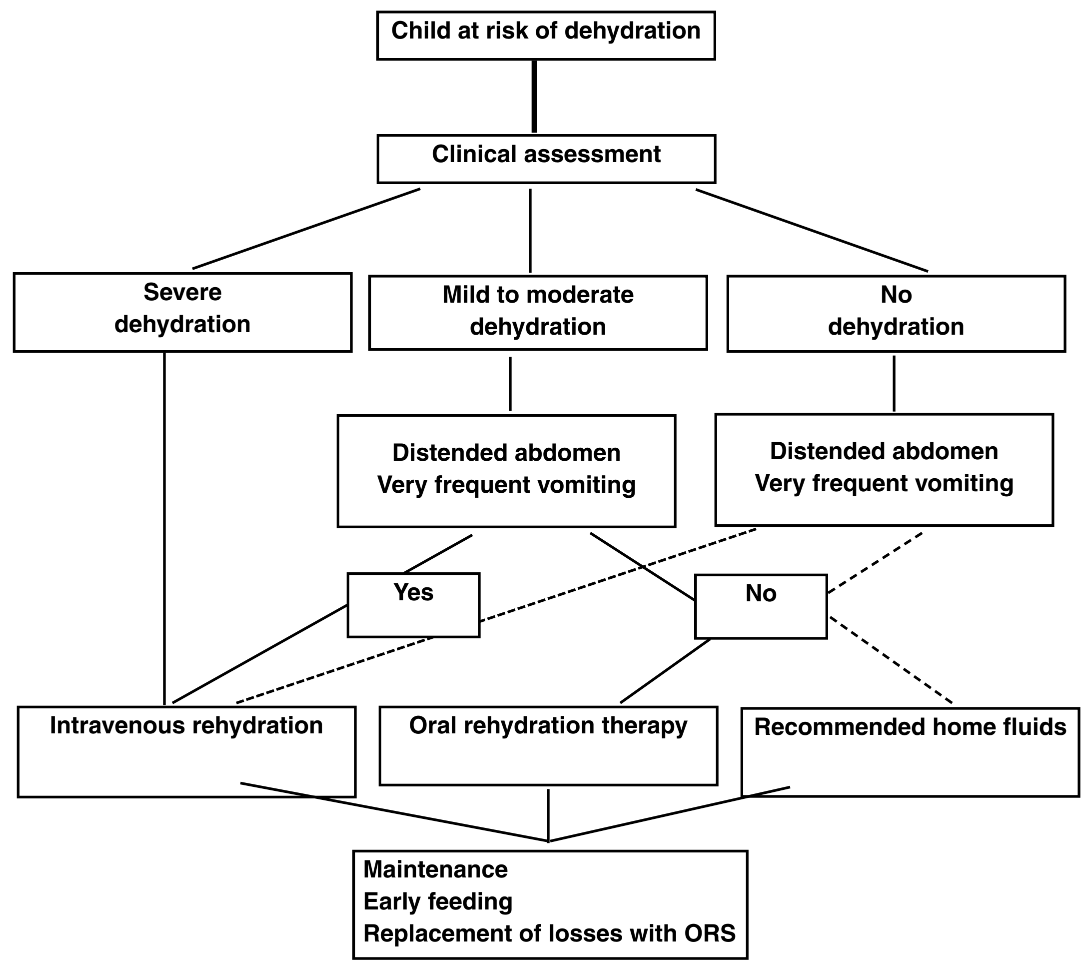
Treatment

Currently, there are no specific antiviral chemotherapeutic agents recommended or routinely employed against rotavirus. For typical rotaviral acute gastroenteritis, therapeutic intervention most often is limited to supportive care. Specifically, ensuring hydration is the most important consideration in children with this illness. However in specific cases, certain additional modalities, discussed below, can be considered. Treatment for specific complications of rotavirus acute gastrointestinal disease other than dehydration, (e.g., hyponatremia, hypernatremia, metabolic acidosis, diaper rash) and others will not be addressed beyond this introductory paragraph. While derangements of sodium accompany less than 5% of cases of rotavirus acute gastroenteritis, caution in management of these children is necessary because central pontine myelinolysis has been reported in the setting of caring for a child with severe hyponatremia.[77] Metabolic acidosis may occur in the setting of rotavirus acute gastroenteritis.[78,79] The anion gap may be useful in helping to determine if the acidosis is due to perfusion–related metabolic disturbances or diarrheal bicarbonate loss.[80,81] Diaper rashes are a common accompaniment to diarrhea and are best managed early with barrier creams and pastes.[82] Extraintestinal infection, including central nervous system involvement, is rare enough that recommended treatment options do not exist at this time.

Hydration and Nutrition

Maintenance of hydration and correction of dehydration are the most important goals of treating rotavirus acute gastroenteritis. The appropriate setting and mode of hydration delivery depends upon the age of the child, the symptoms, and the symp-

Figure 2. Approach to Hydration and Nutrition



tom severity. Severely dehydrated patients will require intravenous rehydration and possibly intensive suppportive care. Moderately and mildly dehydrated children should receive oral rehydration solution (ORS), while children who are not dehydrated may continue their age-appropriate diet once vomiting has ceased,[83] while increasing their fluid intake with usual dietary fluids with ORS or with recommended home fluids.[84,85] (See *Figure 2.*)

Intravenous Fluid Rehydration. Intravenous rehydration is indicated in severely dehydrated children, less severely dehydrated children who continue to vomit after the initiation of oral rehydration solution, and children with abdominal distension, obstruction, or ileus.[83] Some would add to this list of indications the following: excessive stool losses (>15mL/kg/hour)[86] and social considerations, specifically the ability and willingness of parents to perform the labor intensive task of administration of ORS.[83] Access may be difficult in the most severely dehydrated children who are also those who need it the most. In the presence of shock, venous access must be obtained in the most expedient means possible; standard peripheral intravenous access, intraosseus, or central venous access.[87] In less severely dehydrated children without bowel obstruction but in whom intravenous fluid is nevertheless indicated, nasogastric ORS may be used while access is being obtained.[83,88]

Intravenous therapy is often the goal of many families seeking care at an ED for acute rotaviral gastroenteritis. However, many children seeking care in an ED do not require intravenous management, but can be treated with oral rehydration. Some EDs report performing intravenous rehydration in less than 10% of patients presenting with acute gastroenteritis.[89]

Recipes for rapid intravenous rehydration vary. Crystalloids are recommended, and generally isotonic fluids (normal saline or lactated Ringers) are used, at least initially,[90,91] but not uniformly.[61] For severely dehydrated children, an initial bolus of 20 mL/kg and a volume in the range of 60 to 100 mL/kg over several hours is often indicated.[91] However, clinical judgment and frequent reassessment are critical. For less severely dehydrated children, initial rapid intravenous rehydration may be achieved with as little as 30mL/kg, then supplemented with oral rehydration. Many children in this category will cease vomiting once rehydrated.[78,86,92]

Whether continued intravenous or oral rehydration should continue in the inpatient setting after initial rapid ED rehydration is decided on a case-by-case basis. Most patients who were less severely dehydrated will be able to tolerate oral fluids and be discharged.[78,83,92] Patients who are severely dehydrated on intake generally require admission and may require additional evaluation for illnesses mimicking severe dehydration (e.g., congestive heart failure, myocarditis, hemolytic uremic syndrome, intussusception, midgut volvulus, elevated intracranial pressure, sepsis, encephalitis, meningitis).

Most have found that routine laboratory values do not contribute to care.[88,92] Several authors have investigated serum bicarbonate as a predictor of severe dehydration and need for admission, but only one has shown the value of this approach.[78] Additionally, ancillary testing is essential if there is suspicion of other illnesses besides rotaviral acute gastroenteritis, or if there is concern of other complications of gastroenteritis besides dehydration (e.g., hypoglycemia).

Table 1. Constituents of Common Oral Rehydration Solutions

	SODIUM (MEQ/L)	POTASSIUM (MEQ/L)	CARBOHYDRATE (GM/L)	BASE (MEQ/L)	OSMOLALITY (MOSM/L)
WHO ORS*	75	20	75	10	245
Rehydralyte®†	75	20	25	30	305
Pedialyte®†	45	20	25	30	250-270
Enfalyte®‡	50	25	30	34	170
Kaolectrolyte®§	50	21	21	42	not available

* World Health Organization. Information available at http://www.who.int/child-adolescent-health/New_Publications/NEWS/Expert_consultation.htm
† Ross Products. Information available at: http://rpdcon40.ross.com/pn/PediatricProducts.NSF/web_Ross.com_XML_PediatricNutrition/CCF3870065DFF53585256A80007546E8?OpenDocument
‡ Mead Johnson Nutritionals. Information available at http://www.meadjohnson.com/professional/products/enfalyte.html
§ LDS Consumer Products. Information available at http://www.lildrugstore.com/kaolectrolyte/

Oral Rehydration Solution. ORS was introduced in 1979 and has become the mainstay of treatment of children with mild to moderate dehydration.[85] It has been hugely successful, credited with saving 3 million children's lives annually.[85]

Recommended ORSs contain water, sodium, glucose or rice syrup solids, potassium, chloride, and either bicarbonate or citrate. (*See Table 1.*) However, there is some variability in the relative concentrations of these constituents. The original World Health Organization (WHO) ORS contained 90 mEq/L sodium, 111 mmol/L of glucose, and was hyperosmolar at 310 mOsm/L.[93] In 2001 the osmolality was reduced to 245 mOsm/L, the sodium was reduced to 75 mEq/L, and glucose was reduced to 75 mmol/L. This change was based on the meta-analysis of available data, which revealed several benefits with the reduced-osmolarity formulation over the standard WHO ORS in children with non-cholera diarrhea: decreased stool output, decreased vomiting, and decreased need for intravenous therapy.[93] It should be noted, however, that there was an increase in the risk of hyponatremia with the reduced-osmolarity ORS. In the United States, non-WHO commercial products are used for ORS most frequently. Most of these contain less salt (between 45 to 75 mEq/L sodium) and a little more glucose (140 mmol/L). (*See Table 1.*)

ORS should be given frequently in small amounts with a target of 50-100mL/kg during a four-hour period, depending upon the degree of dehydration (mild or moderate).[83] For the average 12-kg 2-year-old child, providing one teaspoon every two minutes if mildly dehydrated, or one teaspoon every one minute if severely dehydrated, satisfies these recommendations. Additionally stool and diarrheal losses must be assessed and replaced under this plan. Following the four-hour rehydration period, normal age-appropriate feeding restarts with increased fluid intake.[83]

Recommended Home Fluids. Recommended home fluids were developed to identify widely available fluids or easily home-prepared fluids that might help prevent dehydration in non-dehydrated children. Acceptable solutions vary by nation.[85] In the United States, fluids that might be considered for this category include breast milk, formula, water, homemade salt and sugar solutions, soups, diluted fruit juices, and sports drinks, but much controversy exists over which fluids are acceptable and recommendations tend to be clinician dependent.[84]

Feeding

In contrast to the marked decrease in mortality advanced by the initiation of oral rehydration therapy, nutritional morbidity remains problematic.[49] Because ORS is calorie poor,[86] early feeding is recommended as soon as rehydration has taken place.[83-85] Early feeding has been shown to promote recovery, and shorten the duration of diarrhea.[84] Current recommendations accent the avoidance of foods high in fat and simple sugar, and promote complex carbohydrates, lean meats, yogurt, fruits, and vegetables.[83] Although uncommon, acquired lactose intolerance may require limitation of lactose-containing solutions.[84]

The classic BRAT (bananas, rice, applesauce, and toast) diet has fallen out of vogue, being described as low energy, low protein, and low fat.[83] However, recent advances in gastroenterologic science may spark renewed interest in this old favorite. Translating theory to practice, green bananas and pectin were shown to reduce diarrheal fluid loss in children with persistent diarrhea in a Bangladesh study.[94] The benefit of rice is not only theoretic: rice-based ORSs have been shown to be superior to standard glucose-based ORSs when considering only volume of stool output.[95]

Breast Milk. Many pediatric patients presenting with rotavirus acute gastroenteritis will have breast milk as a solitary or main food source. While considered the optimal source of food for the healthy young infant, there are additional potential benefits conferred to the infant with diarrheal disease. Besides passive immunization in the form of anti-rotaviral IgA,[49,96] other breast milk constituents (e.g., lactoferrin and lactadherin) seem to have antirotaviral effects.[97,98] However, it is less clear if breastfeeding can be considered an effective treatment per se for rotavirus infection. Some studies have failed to demonstrate an effect, others have shown mild benefit, and still others have demonstrated both marked amelioration of symptoms and also preventative effects.[99] Certainly, most recommend a continuation of breast feeding during acute gastroenteritis,[96,100] but the addition of conventional ORS is recommended in those who are mildly or moderately dehydrated.[83]

Treatment of Vomiting

The use of antiemetic agents for the treatment of gastroenteritis associated with vomiting remains somewhat controversial. In the past, the untoward side effects consisting especially of sedation, akathesia, and dystonia that sometimes accompanied the use of older antiemetics was thought to be disruptive to oral rehydration therapy.[86,101] However, the advent of the better-tolerated 5-HT3 receptor antagonist class of antiemetic agents has opened the door to reconsideration of the use of antiemetic agents for vomiting in childhood gastroenteritis. In recent years, three randomized controlled clinical trials have demonstrated benefit of ondansetron, not only in terms of reduction of vomiting,[102-104] but decreased need for intravenous rehydration and decreased rate of hospital admission.[102-104] Diarrhea was increased, however, and there were higher ED revisit rates in children given ondansetron in one study.[103,104] Based upon this information and the discomfort associated with nausea and vomiting, some physicians have moved toward more frequent use of 5-HT3 receptor antagonist antiemetics.[101] However, the American Academy of Pediatrics (AAP) has not endorsed the use of antiemetic agents.[83]

Treatment of Diarrhea

Conventional Antidiarrheal Agents. There are a number of conventional physiologically active over-the-counter compounds that alter the frequency, volume, or texture of diarrhea. Chief among these are antimotility agents, antisecretory agents, and adsorbents. In general, these pharmacologic agents have not been recommended for use against diarrhea.[83] Opiate and anticholinergic agents are especially to be avoided because of demonstrated toxicity and side effects in children.[83] While certain adsorptive agents—attapulgite in particular— appear safe and reduce diarrhea, there are limited data on the reduction of fluid loss.[83,105] Concern exists that focus on cosmetic improvement of diarrhea texture may distract parents attention from a child's hydration status.

Probiotics. Probiotics are one treatment option whose safety and efficacy may lead to more commonplace use in the future.[106] Probiotics, available as tablets, granules, acidophilus milk, and naturally within live culture yogurts, are whole live bacterial species that are either normally found colonizing the human bowel or are very similar to these species. Probiotics were specifically considered —but not supported—in the AAP's practice parameter on the management of acute gastroenteritis in children.[83] The AAP parameter specifically stated that efficacy rather than safety was the concern resulting in the consensus.[83] However, considerable research has been performed since the 1996 statement supporting the effectiveness of probiotics.[106]

The mechanisms by which various species of *Lactobacillus* and *Bifidobacterium genera* contribute to intestinal health still are being elucidated, but may be multifactorial. Some have suggested that these organisms, which are just some of the normal colonizing bacteria in humans, form a monolayer and thus function literally as a microbial barrier against more pathologic enteric species.[107] While not every study has demonstrated benefit, in a recent meta-analysis of therapeutic trials in humans, the pooled effect of probiotic use resulted in 0.7 fewer days of diarrhea and 1.6 fewer stools by day 2 of illness.[107]

Zinc. Zinc-based enzymes play a critical role in cell function and growth.[90] Tissues with high turnover may demand more zinc to fulfill demands of re-growth. Many studies of acute childhood diarrhea, but not rotavirus specifically, have shown a decrease in duration of diarrhea when zinc was supplemented at 1 to 4 times the recommended daily allowance.[108] This reduction for most studies was in the range of 0.4 to 1.4 days.[108] The WHO now recommends 14 days of zinc supplementation for all children with acute diarrhea: 10 mg daily for infants younger than 6 months, and 20 mg daily for all infants and children older.[90] It should be noted that vomiting may be a side effect of zinc administration.[108] Further, the studies showing efficacy were performed in developing nations where zinc deficiency is common.[108] The AAP's 1996 practice parameters do not address zinc supplementation during diarrheal disease.[83]

Complementary and Alternative Medicine and Food Additives. A number of substances considered as foods (*See also the section on feeding, page 35.*), food additives, or herbal medicines have been studied for their anti-rotaviral effects. Brazilian investigators studied the effects of 12 traditional Brazilian herbal medicines against human rotavirus. Three of these (*Artocarpus integrifolia*, *Myristica fragrans*, and *Spongias lutea*) displayed in vitro antirotaviral activity.[109] Japanese investigators revealed anti-rotaviral effects of both green tea and black tea extracts.[110] These substances have not been investigated in vivo. Barring future evidence to the contrary, caffeinated tea is not recommended because of its diuretic effects.[90]

There have been three randomized controlled clinical trials of homeopathic therapy for acute childhood diarrhea, but not for rotavirus specifically. In a meta-analysis of these three studies the pooled effect was a reduction of 0.8 days of diarrhea.[111] While the trials appear well designed, the application of homeopathy to diarrheal disease has been met with some resistance. Homeopathy was not addressed in the 1996 AAP practice parameters.[83] Because the choice of homeopathic medication is individualized based upon particular features of the diarrheal illness, it is not known if homeopathic medications are effective when prescribed by practitioners who are not trained in homeopathic methods.

Future Therapeutic Modalities

Interesting research continues to explore future therapeutic modalities. The most promising among these is racecadotril, an enkephalinase inhibitor, with antisecretory effects. This drug is well tolerated and has been shown to decrease stool volume, reduce required oral rehydration intake, and decrease ED revisits.[112-114] Other potential future therapies exist. Human interferon alpha, for example, was discovered to be elevated in the serum of children with rotavirus acute gastroenteritis.[115] When human interferon alpha is given orally to rotavirus infected neonatal pigs, the mortality rate decreases substantially.[116] Another potential future modality capitalizes on the replication cycle of the organism. A cysteine protease inhibitor and a serine protease inhibitor have been shown to reduce diarrhea in rotavirus infected mice.[117] More recently, a soybean trypsin inhibitor showed protection against rotavirus-induced derangement of intestinal enzyme and digestive functions.[118,119] Various tissue growth factors have been studied as well; the rationale for these factors is to speed recovery of the damaged intestinal mucosa.[120] *(These drugs have not been approved by the FDA.)*

Prevention

Active Immunization. Natural infection with rotavirus is known to confer clinical immunity against the infecting subgroup.[121,122] Additionally, serum antibody titers against rotavirus are known to be protective against clinical illness.[123] Building upon this knowledge, the first live oral rotavirus vaccine was licensed by the U.S. Food and Drug Administration (FDA) in 1998. However, during the first year of usage there were 15 cases of intussusception reported to the Vaccine Adverse Events Reporting System (VAERS), and the vaccine was withdrawn.[124] Because of the burden to society in terms of morbidity, mortality, and other societal costs, investigation into new rotavirus vaccines continues. A new rotavirus vaccine is in use in Central and South America; two new vaccines are in advanced stages of testing for U.S. licensure, and RotaTeq received FDA approval in February of 2006.[124,125]

Summary

Rotavirus is a wheel-shaped RNA virus that routinely infects the proximal small bowel, resulting in acute gastroenteritis. The virus is spread by the oral-fecal route and results in vomiting, diarrhea, fever, and abdominal pain. In the United States, the most common serious complication is dehydration. Various therapies against vomiting and diarrhea are emerging, but the focus should not be removed from assessment and treatment of dehydration. In these cases, the use of oral rehydration therapy and intravenous rehydration in severe and refractory cases may be lifesaving.

References

1. Bray R. *Armies of Pestilence, The Impact of Disease on History*. New York:Barnes and Noble Books;2000.
2. Murray C, Lopez A. Mortality by cause for eight regions of the world:Global burden of disease study. *Lancet* 1997;349:1269-1276.
3. Bryce J, Baschi-Pinto C, Shibuya K, et al. WHO Child Health Reference Group. *Lancet* 2005 Mar 26-Apr 1;365(9465):1147-52.
4. Glass R, Bresee J, Jiang B, et al. Gastroenteritis viruses: An overview. *Novartis Found Symp* 2001;238:5-19; discussion 19-25.
5. Bishop R, Davidson G, Holmes I, et al. Virus particles in epithelial cells of duodenal mucosa from children with acute non-bacterial gastroenteritis. *Lancet* 1973;Dec 8;2(7841):1281-1283.
6. Bishop R, Davidson G, Holmes I, et al. Detection of a new virus by electron microscopy of faecal extracts from children with acute gastroenteritis. *Lancet* 1974;1(7849):149-51.
7. Flewett T, Bryden A, Davies H, et al. Relation between viruses from acute gastroenteritis of children and newborn calves. *Lancet* 1974;2(7872):61-3.
8. World Health Organization. Rotavirus vaccine-WHO position paper. *Wkly Epidemiol Rec* 1999;74(5):33-40.
9. Parashar U,Hummelman E, Bresee J, et al. Global illness and deaths caused by rotavirus disease in children. *Emerg Infect Dis* 2003;9(5):565-572.
10. Glass R, Kilgore P, Holman R, et al. The epidemiology of rotavirus diarrhea in the United States: Surveillance and estimates of disease burden. *J Infect Dis* 1996;174(suppl):S5-11.
11. Advisory Committee on Immunization Practices. Rotavirus vaccine for the prevention of rotavirus gastroenteritis among children. *MMWR* 1999;48:1-23.
12. Zimmerman C, Bresee J, Parashar U, et al. Costs of diarrhea-associated hospitalizations and outpatient visits in an insured population of young children in the United States. *Pediatr Infec Dis J* 2001;20:14-9.
13. Avendano P, Matson D, Long J, et al. Costs associated with office visits for diarrhea in infants and toddlers. *Pediatr Infect Dis J* 1993;12(11):897-902.
14. Rodriguez W, Kim H, Arrobio J, et al. Clinical features of acute gastroenteritis associated with human reovirus-like agent in infants and young children. *J Pediatr* 1977;91(2):188-193.
15. Hoshino Y, Kapikian A. Rotavirus serotypes: Classification and importance in epidemiology, immunity and vaccine development. *J Health Popul Nutr* 2000;18(1):5-14.
16. Parashar U, Breese J, Gentsch J, et al. Rotavirus. *Emerg Infect Dis* 1998;4(4):561-570.
17. Offit P. Rotaviruses: Immunological determinants of protection against infection and disease. *Adv Virus Re* 1994;44:161-202.
18. Banyai K, Gentsch J, Griffin D, et al. Genetic variability among serotype G6 human rotaviruses: Identifcation of a novel lineage isolated in Hungary. *J Med Virol* 2003;71:124-134.
19. Jiang B, Wang Y, Glass R, et al. The evolution of human group B rotaviruses: Correction and an update. *J Clin Virol* 2005;34(2):158-9.
20. Nakata S, Estes M, Graham D, et al. Detection of antibody to group B adult diarrhea rotavirus in humans. *J Clin Microbiol* 1987;25(5):812-818.
21. Jiang B, Dennehy P, Spangenberger S et al. First detection of group C rotavirus in fecal specimens of children with diarrhea in the United States. *J Infect Dis* 1995;172(1):45-50.
22. Bishop R. natural history of human rotavirus infection. *Arch Virology* 1996; supplement 12:119-128.
23. Konno T, Suzuki H, Katsushima N, Imai A, et al. Influence of temperature and relative humidity on human rotavirus infectio in Japan. *J Infect Dis* 1983;147(1):125-128.
24. Cook S. Glass R, Lebaron C, et al. Global seasonality of rotavirus infections. *Bull World Health Organ* 1990;68 (2):171-177.
25. Lebaron C,Lew J,Glass R, et al. The Rotavirus Study Group. Annual rotavirus epidemic patterns in North America. *JAMA* 1990;264(8):983-988.
26. Zhaori G, Fu L, Xu Y, et al. Detection of rotavirus antigen in tracheal aspirates of infants and children with pneumonia. *Chin Med J* 1991;104(10):830-833.
27. Cotterill H, Curry A, Riordan T. Rotavirus in vomit. *J Infect* 1988;16(2):206-207.
28. Youngs E, Pugh S. Rotavirus in vomit. *J Infect* 1989;18(1):99.
29. Sattar S, Lloyd-Evans N, Springthorpe V, et al. Institutional outbreaks of rotavirus diarrhea:potential role of fomites and environmental surfaces as vehicles for virus transmission . *J Hyg* (Lond) 1986;96(2): 277-89.
30. Akhter J, Al-Hajjar S, Myint S, et al. Viral contamination of environmental surfaces on a general pediatric ward and playroom in a major referral center in Riyadh. *Eur J Epidemiol* 1995;11(5):587-90.
31. Cone R, Mohan K, Thouless M, et al. Nosocomial transmission of rotavirus infection. *Pediatr Infect Dis J* 1988;7(2):103-9.
32. Ford-Jones E, Mindorff C, Gold R, et al. The incidence of viral-associated diarrhea after admission to a pediatric hospital. *Am J Epidemiol* 1990;131(4):711-8.
33. Sattar S, Jacobsen H, Rahman H, et al. Interruption of rotavirus spread through chemical disinfection. *Infect Control Hosp Epidemiol* 1994;15(12):751-6.

34. Cook N, Bridger J, Kendall K, et al. The zoonotic potential of rotavirus. *J Infect* 2004;48(4):289-302.
35. Weiss C, Clark H. Rapid inactivation of rotaviruses by exposure to acid buffer or acidic gastric juice. *J Gen Virol* 1985;66(12):2725-30.
36. Bass D, Baylor M, Broome R, et al. Molecular basis of age-dependent gastric inactivation of rhesus rotavirus in the mouse. *J Clin Invest* 1992;89(6):1741-5.
37. Graham D, Dufour G, Estes M. Minimal infective does of rotavirus. *Arch Virol* 1987;92(3-4):261-271)
38. Lundgren O, Svensson L. Pathogenesis of rotavirus diarrhea. *Microbes Infect* 2001;3:1145-1156.
39. Haffejee I. Neonatal rotavirus infections. *Rev Infect Dis* 1991;13(5):957-962.
40. Sharma R, Hudak M, Premachandra B, et al. Clinical manifestations of rotavirus infection in the neonatal intensive care unit. *Pediatr Infect Dis J* 2002;21(12):1099-1105.
41. Kapikian A, Wyatt R, Levine M, et al. Oral administration of human rotavirus to volunteers:induction of illness and correlates of resistance. *J Infect Dis* 1983;147(1):95-106.
42. Salinas B, Gonzalez G, Gonzalez R, et al. Epidemiologic and clinical characteristics of rotavirus disease during five years of surveillance in Venezuela. *Pediatr Infect Dis* J 23(10 Suppl):S161-167.
43. Lynch M, Lee B, Azimi P, et al. Rotavirus and central nervous system symptoms: cause or contaminant?Case reports and review. *Clin Infect Dis* 2001;33:932-938.
44. Gilger M, Matson O, Conner M et al. Extraintestinal rotavirus infections in children with immunodeficiency. *J Pediatr* 1992;120:912-917.
45. Fischer T, Ashley D, Kerin T, et al. Rotavirus antigenemia in patients with acute gastroenteritis. *J Infect Dis* 2005;192(5):913-919.
46. Blutt S, Kirkwood C,Parreno V, et al. Rotavirus antigenemia and viraemia: a common event? *Lancet* 2003;362:1445-1449.
47. Rowland M, Cole T, Whitehead R.A quantitative study into the role of infection in determining nutritional status in Gambian village children. *Br J Nutr* 1977;37(3):441-50.
48. Martorell R, Habicht J, Yarbrough C et al. Acute morbidity and physical growth failure in rural Guatemalan children. *Am Dis Child* 1975;129(11):1296-1301.
49. Hammarstrom L. Passive Immunity against rotavirus in infants. *Acta Paediatr Suppl* 1999;430:127-132.
50. Keller K, Schmidt H, Wirth S, et al. Differences in the clinical and radiologic patterns of rotavirus and non-rotavirus enterocolitis. *Pediatr Infect Dis J* 1991;10(10):734-738.
51. Sharma R, Garrison R, Tepas J, et al. Rotavirus-associated necrotizing enterocolitis: an insight into a potentially preventable disease? *J Pediatr Surg* 2004;39(3):453-457.
52. Chang E, Zangwill K, Lee H, et al. Lack of association between rotavirus infection and intussusception: implications for use of attenuated rotavirus vaccines. *Pediatr Infect Dis J* 2002;21(2):97-102.
53. Velazquez F, Luna G, Cedillo R, et al. Natural rotavirus infection is not associated to intussusception in Mexican children. *Pediatr Infec Dis J* 2004;23(10 Suppl):S173-178.
54. Bass D, Cordoba E, Dekker C, et al. Intestinal imaging of children with acute rotavirus gastroenteritis. *J Pediatr Gastroenterol Nutr* 2004;39(3):270-274.
55. Robinson C, Hernanz-Schulman M, Griffin M, et al. Evaluation of anatomic changes in young children with natural rotavirus infection:is intussusception biologically plausible? *J Infect Dis* 2004; 189(8):1382-7.
56. Kohler T, Erben U, Wiedersberg H, et al. Histological findings of the small intestinal mucosa in rotavirus infections in infants and young children. *Kinderarztl Prax* 1990;58(6):323-327.
57. Shimizu T, Horiuchi T, Hirooka S, et al.Idiopathic gastric rupture in a 3-month-old girl. *Acta Paediatr* 2003;92(5):628-30.
58. Rivest P, Proulx M, Lonergan G, et al. Hospitalizations for gastroenteritis: the role of roatavirus. *Vaccine* 2004;22(15-16):2013-2017.
59. Zodpey S, Deshpande S, Ughade S, et al. Risk factors for development of dehydration in children aged under five who have acute watery diarrhoea: A case control study. *Public Health* 1998;112(4):233-236.
60. Ahmed F, Karim E. Children at risk of developing dehydration from diarrhea: a case control study. *J Trop Pediatr* 2002;48(5):259-263.
61. Bardhan P, Salam M, Molla A. Gastric emptying of liquid in children suffering from acute rotaviral gastroenteritis. *Gut* 1992;33(1):26-29.
62. Osborne M, Haddon S, Worton K, et al. Rotavirus-induced changes in the microcirculation of intestinal villi of neonatal mice in relation to the induction and persistence of diarrhea. *J Pediatr Gastroenterol Nutr* 1991;12(1):111-120.
63. Brandt C, Arndt C, Evans G. Evaluation of a latex test for rotavirus detection. *J Clin Microbiol* 1987 Sep;25(9):1800-2.
64. Chen H, Chen B, Wang S, et al. Rotavirus gastroenteritis in children: A clinical study of 125 patients in Hsin-Tien area. *Zhonghua Min Guo Xiao Er Ke Yi Xue Hui Za Zhi* 1991 Mar-Apr;32(2):73-8.
65. Clemens J, Ahmed M, Butler T, et al. Rotavirus diarrhoea: An expanding clinical spectrum. *J Trop Med Hyg* 1983 Jun;86(3):117-22.
66. Ford-Jones E, Wang E, et al. Hospitalization for community-acquired, rotavirus-associated diarrhea. *Arch Pediatr Adolesc Med* 2000;154:578-585.
67. Uhnoo I, Olding-Stenkvist E, Kreuger A. Clinical features of acute gastroenteritis associated with rotavirus, enteric adenoviruses, and bacteria. *Arch Dis Child* 1986;61(8):732-8.
68. Bernstein D, Ward R. Rotaviruses. In: Feigin R, Cherry J. *Textbook of Pediatric Infectious Diseases.* 4th ed. Philadelphia: W.B. Saunders Company; 1998:1901-1922.
69. Rodriquez-Baez N, Obrien R, et al. Astrovirus, adenovirus, and rotavirus in hospitalized children: Prevalence and association with gastroenteritis. *J Pediatr Gastroenterol Nutr* 2002;35:64-68.
70. Kapikian A, Shope R. Rotaviruses, reoviruses, coltiviruses, and orbiviruses. In: Baron S, ed. *Medical Microbiology*. 4th ed. Galveston: The University of Texas Medical Branch at Galveston; 1996:Section 2.63.
71. Sanders R, Campbell A, Jenkins M. Routine detection of human rotavirus by latex agglutination: Comparison with enzyme-linked immunosorbent assay, electron microscopy and polyacrylamide gel electrophoresis. *J Virol Methods* 1986 Jul;13(4):285-90.
72. Dennehy P, Gauntlett D, Tente W. Comparison of nine commercial immunoassays for the detection of rotavirus in fecal specimens. *J Clin Microbiol* 1988 Sep;26(9):130-4.
73. Immunocard STAT rotavirus [package insert]. Cincinnati, Ohio: Meridian Bioscience; 2001.
74. Fisher Sure-Vue Rota test Kit [Internet product page]. Accessed 11/15/05 through mainpage: https://www1.fishersci.com/index.jsp
75. VIKIA Rota-Adeno [Internet product page]. Accessed 11/15/05 though mainpage: http://www.biomerieux.com
76. Virogen Rotatest [package insert]. Cranbury, New Jersey:Wampole Laboratories;1994
77. Gregorio L, Sutton C, Lee D. Central pontine myelinolysis in a previously healthy 4-year-old child with acute rotavirus gastroenteritis. *Pediatrics* 1997;99:738-743.
78. Reid S, Bonadio W. Outpatient rapid intravenous rehydration to correct dehydration and resolve vomiting in children with acute gastroenteritis. *Ann Emerg Med* 1996;28(3):318-23.
79. Zvizdic S, Telalbasic S, Cavaljuga S, et al. Clinical characteristics of rotavirus disease. *Bosn J Basic Med Sci* 2004;4(2):22-4.
80. Larsen R, Hansen M, Bindslev N. Duodenal secretion in humans mediated by the EP4 receptor subtype. *Acta Physiol Scand* 2005;185(2):133-40.
81. Quigley R, Baum M. Neonatal acid base balance and disturbances. *Semin Perinatol* 2004;28(2):97-102.
82. Haugen V. Perineal skin care for patients with frequent diarrhea or fecal incontinence. *Gastroenterol Nurs* 1997 May-Jun;20(3):87-90.
83. Practice parameter: The management of acute gastroenteritis in young children. American

Academy of Pediatrics, Provisional Committee on Quality Improvement, Subcommittee on Acute Gastroenteritis. *Pediatrics* 1996 Mar;97(3):424-35.

84. Hick J, Suara R, Nazarian L. Questions on the practice parameter-gastroenteritis. *Pediatrics* 1997;99:143-4.
85. Cesar V, Bryce J, Fontaine O, et al. Reducing deaths from diarrhea through oral rehydration therapy. *Bull World Health Organ* 2000;78(10):1246-1255.
86. Sett A, Mitra U. Answers to questions in relation to oral rehydration therapy. *Indian J Public Health* 1994;38(2):87-8.
87. Guidelines 2000 for Cardiopulmonary resuscitation and emergency cardiovascular care. Part 10: Pediatric advanced life support. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. *Circulation* 2000 Aug 22;102(8 Suppl):I291-342.
88. Nager A, Wang V. Comparison of nasogastric and intravenous methods of rehydration in pediatric patients with acute dehydration. *Pediatrics* 2002;109(4):566-72.
89. Rosenstein B, Baker M. Pediatric outpatient intravenous rehydration. *Am J Emerg Med* 1987;5(3):183-6.
90. World Health Organization. Department of Child and Adolescent Health and Development. *The Treatment of Diarrhoea-A Manual for Physicians and Other Senior Health Workers*. Available at: http://whqlibdoc.who.int/hq/2003/WHO_FCH_CAH_03.7.pdf
91. Assadi F, Copelovitch L. Simplified treatment strategies to fluid therapy in diarrhea. *Pediatr Nephrol* 2003;18(11):1152-6.
92. Moineau G, Newman J. Rapid intravenous rehydration in the pediatric emergency department. *Pediatr Emerg Care* 1990;6(3):186-8.
93. *Reduced Osmolarity Oral Rehydration Salts (ORS) Formulation.* Report from a meeting of experts jointly organized by UNICEF and WHO. UNICEF House, New York, 2001. Available at: http://www.who.int/child-adolescent-health/New_Publications/NEWS/Expert_consultation.htm
94. Rabbani G, Teka T, Saha S, et al. Green banana and pectin improve small intestinal permeability and reduce fluid loss in Bangladeshi children with persistent diarrhea. *Dig Dis Sci* 2004;49(3):475-84.
95. Gore S, Fontaine O, Pierce N. Impact of rice based oral rehydration solution on stool output and duration of diarrhoea: Meta-analysis of 13 clinical trials. *BMJ* 1992;304(6822):287-91.
96. Khin M, Nyunt-Nyunt Wai, Myo-Khin, et al. Effect on clinical outcome of breast feeding during acute diarrhoea. *Br Med J* (CLin Res Ed) 1985;290(6468):587-589.
97. Kvistgaard A, Pallesen L, Arias C, et al. Inhibitory effects of human and bovine milk constituents on rotavirus infections. *J Dairy Sci* 2004;87(12):4088-4096.
98. Van der Strate B, Beljaars L, Molema G, et al. Antiviral activities of lactoferrin. *Antiviral Res* 2001;52(3):225-239.
99. Monson T. Pediatric viral gastroenteritis. *Am Fam Physician* 1986;34(1):95-99.
100. Golding J, Emmet P, Rogers I. Gastroenteritis, diarrhea and breast feeding. *Early Hum Dev* 1997; 49 Suppl:S83-103.
101. Hariharan S, Pohlgeers A, Reeves S. "Doctor, my child needs some medicine!" *Pediatr Emerg Care* 2004;20(8):540-6.
102. Reeves J, Shannon M, Fleisher G. Ondansetron decreases vomiting associated with acute gastroenteritis: A randomized, controlled trial. *Pediatrics* 2002 Apr;109(4):e62.
103. Ramsook C, Sahagun-Carreon I, Kozinetz C, et al. A randomized clinical trial comparing oral ondansetron with placebo in children with vomiting from acute gastroenteritis. *Ann Emerg Med* 2002 Apr;39(4):397-403.
104. Cubeddu L, Trujillo L, Talmaciu I, et al. Antiemetic activity of ondansetron in acute gastroenteritis. *Aliment Pharmacol Ther* 1997 Feb;11(1):185-91.
105. Zaid M, Hasan M, Khan A. Attapulgite in the treatment of acute diarrhea: A double-blind placebo-controlled study. *J Diarrhoeal Dis Res* 1995;13(1):44-6.
106. Van Niel C, Feudtner C, Garrison M, et al. Lactobacillus therapy for acute infectious diarrhea in children: A meta-analysis. *Pediatrics* 2002;109(4):678-684.
107. Servin A. Antagonistic activities of Lactobacilli and bifidobacteria against microbial pathogens. *FEMS MicrobiolRev* 2004;28:405-440.
108. Fontaine O. Effect of zinc supplementation on clinical course of acute diarrhoea. *J Health Popul Nutr* 2001;19(4):339-46.
109. Goncalves J, Lopes R, Oliviera D, et al. In vitro anti-rotavirus activity of some medicinal plants used in Brazil against diarrhea. *J Ethnopharmacol* 2005;99(3):403-407.
110. Mukoyama A, Ushijima H, Nishimura S, et al. Inhibition of rotavirus and enterovirus infections by tea extracts. *Jpn J Med Sci Biol* 1991;44(4):181-6.
111. Jacobs J, Jonas W, Jimenez-Perez M, et al. Homeopathy for childhood diarrhea: Combined results and metanalysis from three randomized, controlled clinical trials. *Pediatr Infect Dis J* 2003;22(3):229-34.
112. Salazar-Lindo E, Santisteban-Ponce J, Chea-Woo E, et al. Racecadotril in the treatment of acute watery diarrhea in children. *N Engl J Med* 2000;343(7):463-7.
113. Cezard J, Duhamel J, Meyer M, et al. Efficacy and tolerability of racecadotril in acute diarrhea in children. *Gastroenterol* 2001;120(4):799-805.
114. Cojocaru B, Bocquet N, Timsit S, et al. Effect of racecadotril in the management of acute diarrhea in infants and children *Arch Pediatr* 2002;9(8):774-9.
115. De Boissieu D, Lebon P, Badoual J, et al. Rotavirus induces alpha-interferon release in children with gastroenteritis. *J Pediatr Gastroenterol Nutr* 1993;16(1):29-32.
116. Lecce J, Cummins J, Richards A. Treatment of rotavirus infection in neonate and weanling pigs using natural human interferon alpha. *Mol Biother* 1990;2(4):211-6
117. Ebina T, Tsukada K. Protease inhibitors prevent the development of human rotavirus-induced diarrhea in suckling mice. *Microbiol Immunol* 1991;35(7):583-588

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To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion. When your evaluation is received, a certificate will be mailed to you.

CME Objectives

The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

a.) Quickly recognize or increase index of suspicion for specific conditions;

b.) Understand the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed;

e.) Provide patients with any necessary discharge instructions.

118. Katyal R, Rana S, Singh V, et al. Protection against rotavirus diarrheoea in mice by trypsin inhibitor. *Indian J Exp Biol* 2001;39(6):546-50.
119. Katyal R, Rana S, Ojha S, et al. Soybean trypsin inhibitor confers protection against rotavirus infection ininfant mice. *Trop Gastroenterol* 2001;22(4):207-10.
120. Farthing M. Treatment of gastrointestinal viruses. *Novartis Found Symp* 2001;238:289-300
121. Bishop R, Barnes G, Cipriani E et al. Clinical immunity after neonatal rotavirus infection. A prospective longitudinal study in young children. *N Engl J Med* 1983;309(2):72-6.
122. Bhan M, Lew J, Sazawal A, et al. Protection conferred by neonatal rotavirus infection against subsequent rotavirus diarrhea. *J Infect Dis* 1993;168(2):282-7.
123. Velazquez F, Matson D, Guerrer M, et al. Serum antibody as a marker of protection against natural rotavirus infection and disease. *J Infect Dis* 2000;182(6):1602-9.
124. Bines J. Intussuception and rotavirus vaccines. Vaccine 2005; Aug 10 [Epub ahead of print].
125. U.S. Food and Drug Administration. FDA approves new vaccine to prevent rotavirus gastroenterisis in infants. *FDA News* P06-16. February 3, 2006.

CME Questions

21. There are seven major groups of rotavirus. Which group is responsible for most of human gastrointestinal illness in the United States?
    A. Group B
    B. Group G
    C. Group C
    D Group F
    E. Group A

22. What is the most common age group affected by rotavirus?
    A. Adolescents
    B. 6 months to 2 years
    C. 6 to 10 years
    D. Adults

23. Worldwide, rotavirus accounts for what percentage of all deaths due to diarrheal illness?
    A. 1%
    B. 50%
    C. 25%
    D. 75%
    E. 90%

24. What is the primary mode of transmission of rotavirus?
    A. fecal-oral route
    B. aerosol
    C. nosocomial
    D. fomite

25. The incidence of rotavirus infection in temperate climates is highest in which season?
    A. spring
    B summer
    C. fall
    D. winter

26. The rotavirus vaccine licensed in 1998 was withdrawn due to concern over association with which of the following conditions?
    A. Anaphylaxis
    B. Guillian-Barre syndrome
    C. Fever
    D. Intussusception
    E. Prolonged crying

27. Possible beneficial therapies for treatment of rotavirus infection include all of the following agents, *except:*
    A. zinc
    B. attapulgite
    C. anticholinergic agents
    D. probiotics

28. The most commonly used detection method(s) for identifying rotavirus is:
    A. tissue culture
    B. electron microscopy
    C. PAGE
    D. LA and ELISA
    E. acute and convalescent serum titers

29. Which of the following clinical manifestations is *not* commonly associated with rotavirus infection in patients out of the neonatal period?
    A. watery diarrhea
    B. vomiting
    C. fever
    D. bloody diarrhea
    E. dehydration

30. Oral rehydration therapy should begin with:
    A. 50-100 mL/kg over 4 hours
    B. 10-15 mL/kg over 4 hours
    C. 100 mL/kg per hour
    D. 300-400 mL/kg over 4 hours

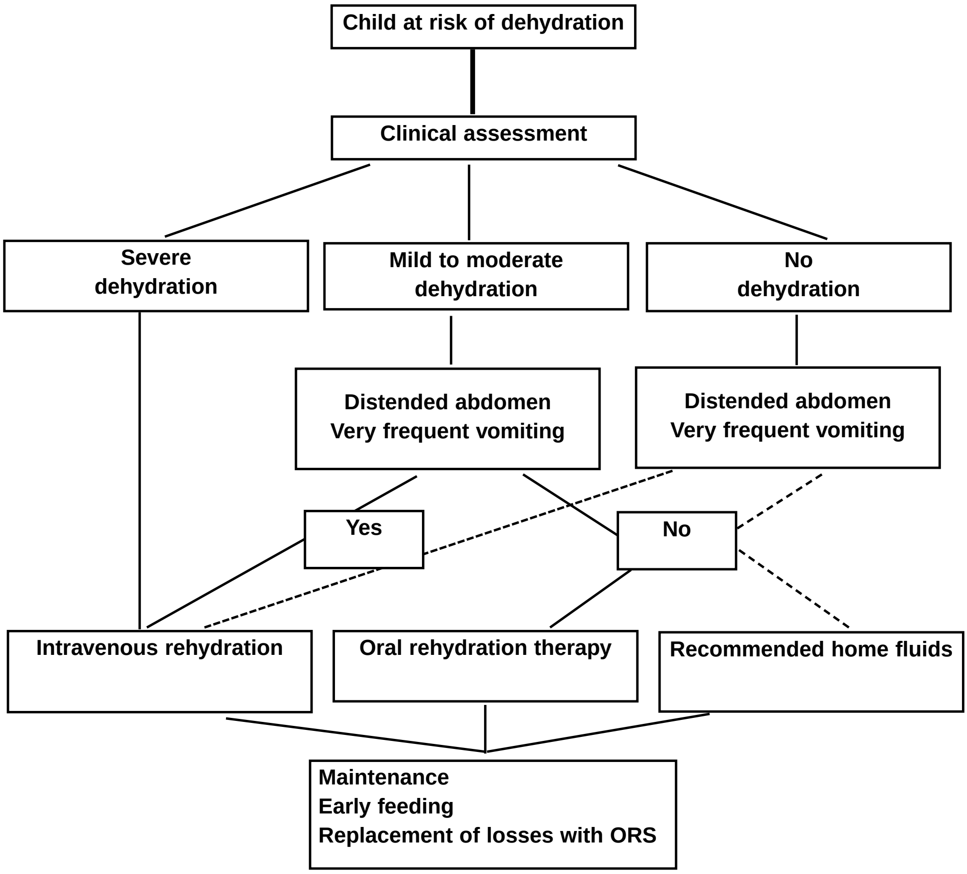
Answers:
21. E
22. B
23. C
24. A
25. D
26. D
27. C
28. D
29. D
30. A

In Future Issues: **Hypertension in children**

PEDIATRIC Emergency Medicine Reports
The Practical Journal of Pediatric Emergency Medicine

Rotavirus

Approach to Hydration and Nutrition



Constituents of Common Oral Rehydration Solutions

	SODIUM (MEQ/L)	POTASSIUM (MEQ/L)	CARBOHYDRATE (GM/L)	BASE (MEQ/L)	OSMOLALITY (MOSM/L)
WHO ORS*	75	20	75	10	245
Rehydralyte®†	75	20	25	30	305
Pedialyte®†	45	20	25	30	250-270
Enfalyte®‡	50	25	30	34	170
Kaolectrolyte®§	50	21	21	42	not available

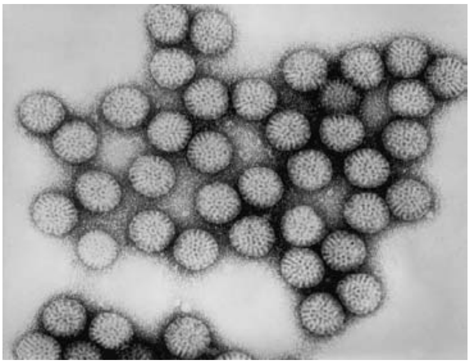
* World Health Organization. Information available at http://www.who.int/child-adolescent-health/New_Publications/NEWS/Expert_consultation.htm

† Ross Products. Information available at: http://rpdcon40.ross.com/pn/PediatricProducts.NSF/web_Ross.com_XML_PediatricNutrition/CCF3870065DFF53585256A80007546E8?OpenDocument

‡ Mead Johnson Nutritionals. Information available at http://www.meadjohnson.com/professional/products/enfalyte.html

§ LDS Consumer Products. Information available at http://www.lildrugstore.com/kaolectrolyte/

Human Rotavirus Seen through Electron Microscopy



Transmission electron micrograph of intact rotavirus particles, double-shelled. Distinctive rim of radiating capsomeres.

Reprinted from: Centers for Disease Control and Prevention. Public Health Image Library ID# 273. Available at: http://phil.cdc.gov/phil/quicksearch.asp

Supplement to *Pediatric Emergency Medicine Reports*, March 2006: "Rotavirus: An Update on Current Diagnosis and Management." *Author:* **Dante Pappano, MD**, Senior Clinical Instructor, Pediatric Emergency Medicine, Department of Emergency Medicine, University of Rochester School of Medicine and Dentistry, Rochester, New York; **Ellen S. Bass, MD, MPH**, Attending Physician, Strong Memorial Hospital; Senior Clinical Instructor, Departments of Emergency Medicine and Pediatrics, University of Rochester School of Medicine and Dentistry, Rochester, New York; **Sharon Humiston, MD, MPH,** Associate Professor, Emergency Medicine & Pediatrics, Department of Emergency Medicine, University of Rochester School of Medicine and Dentistry, Rochester, New York. *Peer Reviewer:* **James A. Wilde, MD, FAAP,** Director, Pediatric Emergency Medicine, Medical College of Georgia, Augusta; Medical Director, Georgia United Against Antibiotic Resistant Disease (GUARD). *Pediatric Emergency Medicine Reports' "Rapid Access Guidelines."* Copyright © 2006 Thomson American Health Consultants, Atlanta, GA. **Vice President and Group Publisher:** Brenda Mooney. **Editor-in-Chief:** Ann Dietrich, MD, FAAP, FACEP. **Editorial Group Head:** Glen Harris. **Managing Editor:** Martha Jo Dendinger. For customer service, call: **1-800-688-2421.** This is an educational publication designed to present scientific information and opinion to health care professionals. It does not provide advice regarding medical diagnosis or treatment for any individual case. Not intended for use by the layman.

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Trauma Reports

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March/April 2006

Managing the Airway of the Pediatric Trauma Patient: Meeting the Challenge

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Accidental injury is the number one cause of death and disability in all children between 1 and 18 years of age. In 2002, trauma accounted for 33-39% of mortality in children aged 1-14 years.[1]

Airway management is paramount in the management of the trauma patient and especially in the case of the pediatric trauma patient. A known pitfall in trauma management—under-resuscitation and subsequent hypoxia— is the most common cause of cardiac arrest in the child.[2-4] Early identification and aggressive management of pediatric airway difficulties may affect the child's outcome.

This article reviews the anatomic and physiologic differences between pediatric and adult airways and the critical aspects of management.

—The Editor

Introduction

Although children do have an increased susceptibility to respiratory compromise (*Table 1*), appropriate and timely airway management can profoundly influence mortality and morbidity and lead to a good outcome. Acute trauma may result in airway obstruction in the case of orofacial trauma or altered level of consciousness. Decreased oxygenation occurs in pulmonary injuries such as pneumothorax, hemothorax, chest wall injuries, pulmonary contusions, and aspiration. Ventilation can be compromised by airway obstruction, diaphragmatic injury, or central nervous system (CNS) depression. One of the main causes of early death in pediatric trauma is airway compromise often secondary to inadequate airway management.[2-4] Many trauma-associated deaths are preventable if appropriate, prompt care is provided at initial presentation.[5] Once respiratory arrest leads to pulseless cardiac arrest, survival as well as neurologic

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outcome is dismal.[2]

The pediatric airway presents a special challenge to many clinicians. Although airway management priorities in trauma are the same for children and adults, the pediatric airway has several unique features that emergency department (ED) physicians must understand to manage a pediatric trauma patient optimally.

This article does not extensively review basic airway management in trauma. Rather, the anatomic and physiologic differences between pediatric and adult airways as well as how and when these differences translate into differences in management are highlighted.

The Pediatric Airway: Anatomy

Overall, the pediatric airway is anatomically different than the adult airway. It has a different shape, is more pliable, and is smaller in diameter and length than the adult airway (*Figure 1*). These anatomic differences are most pronounced in infants and evolve with the child's growth. Generally, by the age of 8 years, the airway is considered to have the characteristics of the adult airway.[6]

Unique features in the child (*Table 2*) include a mouth that is smaller and oropharyngeal soft tissues, tongue, and tonsils that are proportionately larger than in the adult. The tongue is the main cause of airway obstruction in the child and may make visualization of the larynx difficult. In infants and children, the larynx is more cephalad and anterior than in adults.[7] The larynx or glottic opening of the trachea is at the level of C1 during infancy, transitioning to the level of C4-C5 in the adult.[8] Therefore, alignment of the oropharynx and larynx usually requires a straight laryngoscope blade. The thinner tip of the straight laryngoscope blade also helps elevate the floppy epiglottis out of the visual field. In children, the angle between the epiglottis and the larynx is more acute than in the adult, thus making blind nasotracheal intubation contraindicated in the young child; this angle is difficult to maneuver and blind manipulation can cause trauma and bleeding of the adenoids. The pediatric epiglottis is long, narrow, and floppy. A child's vocal cords have a slightly more anterocaudal angle than in an adult, therefore, if a child's head is not in the normal supine anatomical position, the vocal cords are more difficult to visualize during intubation. While an adult airway narrows after the cricoid cartilage, the narrowest portion of a pediatric airway is the cricoid cartilage. Historically, uncuffed endotracheal tubes (ETTs) have been recommended in children younger than 8 years because of concern about tissue necrosis from an improperly inflated balloon. New cuffed ETTs, which are safe in young children, have been developed, are widely available, and are becoming the preferred choice for intubation.[9] The rationale for using these cuffed ETTs is to increase effective ventilation by decreasing air-leak, a critical issue in children who require increased ventilatory pressures (e.g., children with acute respiratory distress syndrome).[2] A child's anatomically funnel-shaped larynx allows secretions to accumulate in the retropharyngeal area and increases the frequency of airway obstruction in children.[10]

In an infant, the relatively large occiput causes passive flexion of the cervical spine, as well as buckling of the pharyngeal airway.[2] To maintain cervical spine protection and an open airway, the patient must be kept in the 'sniffing position'. In this position, the external auditory meatus is aligned with the anterior border of the shoulder (*Figure 2*).

A pediatric trachea is smaller in diameter and length (5 cm long and grows to 7 cm by 18 months of age)[11] and is more pliable and compressible than the adult airway. Failure to appreciate this short length may result in right main stem bronchus intubation, inadequate ventilation, accidental tube dislodgement, and/or mechanical barotrauma.[11] The cricothyroid membrane is very small and difficult to palpate and to incise, therefore, cricothyroidotomy is contraindicated in children younger than 8 years.

Infants are obligate nasal breathers up to 6 months of age.[6] Infant nares are proportionately smaller than in adults and when occluded with mucous or secretions, they may cause significant airflow obstruction. This condition can be exacerbated by compressing the infant's nares with a face mask.[6]

The pediatric chest is more pliable than the adult chest. Even in the setting of significant blunt thoracic trauma, a child may have severe pulmonary contusions with no outward indication of severe trauma.[14] The high incidence of tension pneumothorax may be due to the increased pliability of the pediatric chest. The diaphragm and the abdominal wall musculature are relatively immature and weak despite being the major respiratory muscles in the child. Serious abdominal trauma or abdominal distention from bleeding or perforation can severely compromise a child's ventilatory status.

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Table 1. Reasons for Increased Pediatric Susceptibility to Respiratory Emergencies

PEDIATRIC CHARACTERISTIC	REASON FOR INCREASED SUSCEPTIBILITY TO RESPIRATORY EMERGENCIES
Higher metabolic requirements	Less tolerant of hypoxia and shock
Smaller airway diameter	Increased airflow resistance
Collapsible airways	Easily occluded
Compliant chest wall	May have significant lung injury and decreased air exchange despite relatively few external signs of trauma
Underdeveloped respiratory muscles	Diaphragm (the major muscle of respiration) fatigues relatively easy
Behavioral immaturity	Inability to verbalize respiratory distress or cooperate with intervention

Adapted from Mahadevan S, et al. *Principles of Emergency Medicine*. 1st. ed. Cambridge, UK; Cambridge University Press;2005.

Figure 1. Comparison of Pediatric and Adult Airway

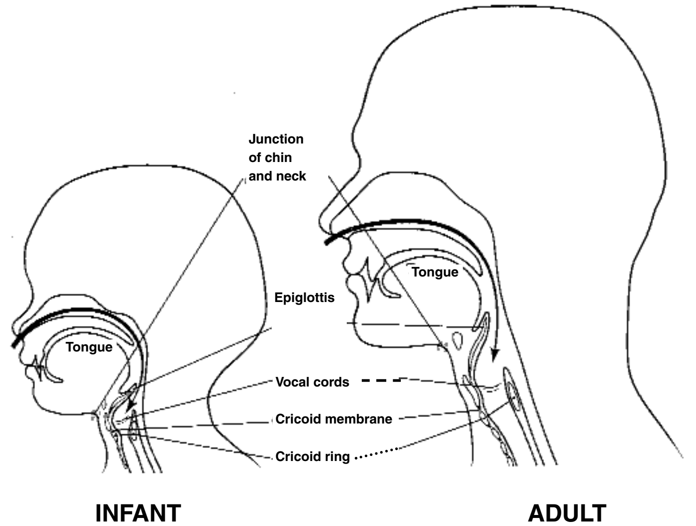

Figure 1. The anatomic differences particular to children are these: 1) Higher, more anterior position for the glottic opening. (Note the relationship of the vocal cords to the chin/neck junction.) 2) Relatively larger tongue in the infant, which lies between the mouth and glottic opening. 3) Relatively larger and more floppy epiglottis in the child. 4) Cricoid ring is the narrowest portion of the pediatric airway versus the vocal cords in the adult. 5) Position and size of the cricothyroid membrane in the infant. 6) Sharper, more difficult angle for blind nasotracheal intubation. 7) Larger relative size of the occiput in the infant.

Reprinted with permission from: Wall RM. *Manual of Emergency Airway Management.* Philadelphia:Lippincott Williams & Wilkins;2000:145.

The Pediatric Airway: Physiology

Children have great compensatory mechanisms, and their vital signs may remain normal even with a severe physiologic compromise. Likewise, changes in vital signs may be subtle and misleading and may result in inadequate assessment of respiratory status, leading to inadequate resuscitation and precipitous deterioration of the pediatric trauma patient.

The major cause of traumatic death in children is respiratory failure.[2-4] Given the increased metabolic rate of children, hypoxia is poorly tolerated. Normal cerebral blood flow increases proportionately to nearly twice that of adult levels by the age of 5 years, and then decreases,[2] a partial reason for children's severe susceptibility to cerebral hypoxia. Children and infants in particular have smaller lung volumes and higher oxygen demands than adults. Therefore, they consume their respiratory reserves more quickly than in adults.

Children also have increased oral secretions and vagal tone compared with adults. Historically, this has led to the dictum of pretreatment with atropine to avoid life-threatening bradycardia with laryngeal manipulation. Although a recent study documented that pretreatment with atropine may not prevent bradycardia in all cases, and thus may not be required for all pediatric patients,[8] it is still the standard of care.

Pediatric Airway Management Goals

The overall goals of airway management in the trauma patient are to: 1) provide a secure patent airway, 2) supply supplementary oxygen, 3) support ventilation, and 4) prevent aspiration. These goals are the same for the pediatric and the adult patient.

Assessment of the Pediatric Respiratory Status

Accurate assessment of the pediatric respiratory status is of the utmost importance. Failure to recognize and respond to pediatric respiratory distress can precipitously lead to cardiopulmonary arrest. Pediatric vital signs must be compared with age-related normal values. Pulse oximetry is a useful adjunct for monitoring a child's respiratory status.

Tachypnea is the initial compensatory mechanism for preserving minute volume.[2] Observation of a child's respiratory effort is the most accurate method to identify respiratory distress. Tidal volume assessed by chest rise or abdominal excursion can reveal the adequacy of the trauma patient's respirations. Respiratory noises (e.g., snoring or stridor indicating upper airway obstruction; wheezes indicating lower airway obstruction; and grunting, ominous for severe respiratory distress) are indications of work of breathing and airway obstruction.

The pediatric brain has high oxygen consumption, and hypoxia may result in an altered mental status. Thus, evaluation of overall general appearance (e.g., posture, level of alertness, or responsiveness) and other organ systems such as CNS (e.g., lethargy, agitation, or other altered mental status) and skin (e.g.,

Table 2. Unique Characteristics of Pediatric Airway Anatomy and Their Consequences on Airway Management

PEDIATRIC ANATOMIC FEATURE	AIRWAY MANAGEMENT SIGNIFICANCE
Large head, short neck	1. Neutral C-spine position/sniffing position difficult to achieve 2. Increased incidence of head trauma
Small mouth	1. Small mouth in conjunction with large soft tissue structures leaves less space for laryngoscope blade and subsequent airway visualization 2. Smaller straight laryngoscope blade can fit better into the child's mouth and lift soft tissues out of the way
Large tongue	1. Most common cause of airway obstruction
Large adenoids	1. Blind nasotracheal intubation contraindicated in children < 9 years secondary to large adenoids and potential to bleed 2. Oropharyngeal airway should not be inserted upside down and turned
Anterior and cephalad airway	1. Acute angle between epiglottis and anterior glottic opening makes nasotracheal intubation difficult 2. Alignment of different airway axes is often better accomplished with a straight blade than a curved blade
Long floppy epiglottis	1. Epiglottis can block visualization of the airway. A straight blade, with a narrower tip can be used to lift it out of the way.
Cricoid ring is narrowest part of the pediatric airway	1.Historically, uncuffed ETTs were recommended secondary to possible tissue necrosis from inflated balloon. 2. New data support the use of new cuffed ETTs, which decrease air leak and improve ventilation
Pediatric airway smaller, shorter, and delicate	1. Pediatric airway more prone to obstruction, compression 2. Right main stem intubation, and tube dislodgement are more common
Nares smaller; Infants up to 6 months of age are obligate nasal breathers	1. Small nares cause increased resistance to airflow especially when narrowed by secretions. 2. Compression of nares, can cause significant respiratory obstruction
Chest wall pliability	1. Children can have increased lung parenchymal damage with few outward signs.
Diaphragm is the main muscle of breathing	1. Abdominal trauma and distention can cause or contribute to severe respiratory compromise
Small cricothyroid membrane	1. Cricothyroidotomy contraindicated in children younger than 8 years

pallor, cyanosis, or delayed capillary refill) also will aid the recognition of respiratory distress and impending respiratory failure in a child.[2]

Management of the Pediatric Airway

Positioning. In a spontaneously breathing child, the airway should be optimized by placing the child's head in the sniffing position while maintaining neutral alignment of the cervical spine (*Figure 2*). Often a shoulder roll in an infant is recommended to help in obtaining a sniffing position. Due to the prominence of the tongue, the chin lift or jaw thrust maneuver combined with bimanual inline spinal immobilization may be necessary to maintain an unobstructed airway. The mouth and oropharynx should be cleared of secretions or debris, and supplemental oxygen should be administered. If the child's airway or ventilation is compromised, then mechanical methods may be necessary. Before attempts to mechanically establish an airway, the child should be pre-oxygenated.

Airway Adjuncts. Positioning alone may not be enough to keep the tongue from obstructing the airway. The nasal and oral airways are common airway adjuncts used in children.

Oral Airway. The oral airway is contraindicated for use in children who are conscious because it can trigger the gag reflex; but in an unconscious patient it may relieve upper airway obstruction. The appropriate oral airway will extend from the level of the incisors in the child's mouth to just anterior to the angle of the mandible. Placement of the oral airway by depressing the tongue with a tongue blade is recommended. The practice of inserting the oral airway backward and rotating it into position is not recommended for pediatric patients. Trauma with resultant hemorrhage into soft tissues may occur. Note that if the oral airway is too small, the tongue may be pushed into the pharynx causing increased airway obstruction. Proper positioning should result in improved and easier ventilation.

Ventilation and Breathing. If spontaneous ventilation and breathing are not adequate, children with trauma must have ventilatory assistance. Bag valve mask (BVM) ventilation can be successful; however, BVM ventilation may contribute to gastric distention with air and potential regurgitation and aspiration.[10]

Bag Valve Mask. Usual spontaneous tidal volumes vary from 5 to 8 mL/kg in infants and children.[2] Although most BVM devices are designed to limit the amount of pressure that can be exerted manually on the child's airway, the physician must minimize the potential for iatrogenic bronchoalveolar injury or development of a pneumothorax. Remember that self-inflating BVM devices will not deliver oxygen unless squeezed.[2] Putting this type of BVM device over the face of a spontaneously breathing child without squeezing the bag will result in a lack of oxygen delivery to the child.

If airway obstruction occurs and BVM ventilation cannot be achieved, the airway should be re-positioned. Jaw thrust or chin lift may be used, but chin tilt should be avoided in the setting of a suspected cervical spine injury. If repositioning does not succeed, an airway adjunct may be needed. Due to the relative size of an

Figure 2. Positioning of the Child for Ventilation

A

B

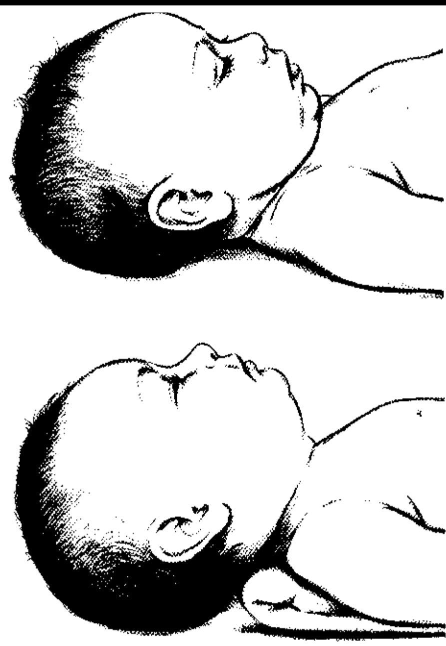

Figure 2. Positioning fo the Child for Ventilation.
A: Incorrect position with neck flexion. **B**: Correct position for infant. Note that the external ear canal is anterior to the shoulder.

Adapted with permission: Cote CJ, et al. *A Practice of Anesthesia for Infants and Children*. Philadelphia: WB Saunders;1993.

adult's hand to a child's neck, it is easy for a practitioner to accidentally apply additional pressure to the child's neck and cause further obstruction of the airway. The optimal placement of the hand is to apply the index finger to the mask, the middle finger to the mentum, and the ring and little finger to the bony angle of the mandible.[7]

Equipment. Equipment size changes with age. The ED physician caring for a child with trauma must have immediate access and knowledge of the appropriate sized equipment to use, as well as a systematic organization of this equipment. Length-based resuscitation methods provide accurately sized equipment and medication doses, and the most common form used is the Broselow tape. The Broselow tape should be measured with the red arrow at the patient's head and the color-coded section at the patient's heel. Color-coded carts and resuscitation supplies that correspond to the Broselow tape color-coded system have been shown to be more efficient than previous standard organization systems.[15]

A recent small study looked at common problems with the Broselow tape. The most common problem stemmed from holding the tape in the wrong direction: Specifically, the wrong end was put at the top of the patient's head. In several cases, clinicians had difficulty using the Broselow tape to determine the correct medication and equipment size to use. The older version of the Broselow tape had two white-colored sections, one of which was at the top and was not an actual dosing zone. The newest version has corrected this by making the zone closest to the head gray in color.[16]

ETT size can be estimated by comparing the diameter of the ETT to the size of the nail on the child's fifth finger, or calculated by the formulas:

Uncuffed ETT size: (Age in years)/4 +4

Cuffed ETT size: (Age in years)/4 +3

Most ETTs have a mark at the end of the tube that indicates the point of the tube that should be just past the vocal cords. The appropriate ETT depth of insertion in centimeters can be calculated by the formula: ETT depth cm = 3 × ETT size.

Laryngoscope Blades. Straight blades are easier to insert in the child's mouth, but the thinness of the straight blade can make moving and retracting a large tongue difficult. The curved laryngoscope blade is larger and bulkier than the straight blade, but it retracts the tongue more easily and may be useful in certain pediatric populations when the tongue is larger or bulkier than usual.

Endotracheal Intubation

Endotracheal intubation is performed to achieve definitive airway management. It is indicated in acute respiratory failure, as well as for airway protection, to decrease the work of breathing and for some therapeutic interventions (e.g., closed head injury with increased intracranial pressure). (*See Table 3*.) Almost half of endotracheal intubations in children are for trauma, with head injury being the single most common indication for intubation in children (25% of cases).[17-18]

Rapid sequence intubation (RSI) is the use of a systematic protocol involving sedatives and neuromuscular blocking agents to increase chances of a successful intubation, decrease risks of aspiration, and to avoid increases in intracranial pressure for head injured patients. Numerous studies have demonstrated that intubation without paralysis and with or without a sedative has a lower success rate in children and adults, as well as a higher complication rate than RSI.[17,19,20]

The ten steps of RSI include 1) a brief history, 2) preparation, 3) monitoring, 4) preoxygenation, 5) premedication, 6) assisted ventilation and cricoid pressure, 7) sedation, 8) paralysis, 9) intubation, and 10) confirmation of tube placement.[21] (*See Table 4.*)

Major differences between pediatric and adult RSI include the steps of preoxygenation and RSI pharmacy. Preoxygenation with the patient breathing 100% oxygen spontaneously for 5 minutes will 'washout' all the nitrogen in the lungs, providing the patient with an oxygen reserve. An adult patient can tolerate up to 5 minutes of apnea without developing hypoxia. A child has smaller lungs and higher metabolic needs than an adult; often times children can tolerate only 2-3 minutes of apnea before developing hypoxia.[18]

RSI pharmacy involves a choice of different pre-medications, sedative agents, and paralytic agents depending upon the clinical scenario. Important considerations are the presence of increased intracranial pressure and hypotension. Areas of controversy in pediatric RSI include the use of pre-medications, the use of etomidate as an RSI sedative, and the use of succinylcholine as a paralytic agent.

Premedications

RSI premedications include pharmacologic agents that are intended to blunt the cardiovascular stimulation of intubation; decrease intracranial pressure; decrease bradycardia, which can be caused by succinylcholine; decrease airway secretions; and prevent muscular fasciculations caused by depolarizing neuromuscular blocking agents. Succinylcholine has been demonstrated in studies to cause bradycardia, and atropine has been shown to effectively block it. However, it is unclear if the etiology of the bradycardia stems from the medication itself or from the vagal response to intubation.[21,22]

Lidocaine is used widely to mitigate increased intracranial pressure in head injured patients. However, its use is controversial with little or conflicting evidence as to its effects on intracranial pressure.[18,20,23] Although lidocaine also can cause complications of hypotension and dysrhythmia, it is customarily given as pretreatment for RSI in the setting of head injury.

Opiate analgesic agents (e.g., fentanyl) have been advocated to reduce adverse hemodynamic and intracranial pressure effects of intubation,[24] although the data for this use are scant. Opiates can cause hypotension in trauma patients. In addition, fentanyl, in particular, may be associated with chest wall rigidity, especially when given as a bolus.

Anticholinergics. Atropine (0.01-0.02 mg/kg IV min 0.1 mg; max 1 mg) has been recommended to prevent bradycardia resulting from vagal stimulation. Currently, it is recommended that atropine be used universally in children younger than 1 year to minimize the chance of bradycardia in response to laryngoscopy.[2] In children older than 1 year, atropine should be given only if succinylcholine also is being used. In adolescents and adults who receive a second dose of succinylcholine and in any patient with bradycardia, atropine should be given 1-2 minutes prior to intubation.[2] There is currently more discussion about the validity of the use of atropine.[25-27] However, there have been no definitive studies to support discontinuation of atropine with succinylcholine in children.

Defasciculating Agents. The use of one-tenth the dose of a nondepolarizing muscle relaxant 1-3 minutes before succinylcholine administration will decrease muscular fasciculations. This is not necessary in children 5 years or younger.[20] However, even in older patients administration of a defasciculation agent adds time and complexity to the RSI, and may not be clinically indicated in the management of an acute traumatic airway.[25]

Sedative Agents. Etomidate (0.2-0.3 mg/kg IV) is an imidazole hypnotic agent. It does not cause hypotension or increased intracranial pressure, making it the ideal hypnotic agent for the multitrauma patient with or without potential head trauma. Previously, the use of etomidate has been tempered because it causes adrenal suppression, even after a single dose. However, the clinical significance of this adrenal suppression is unknown.[28] In a recent study, it was found to be safe for pediatric patients with no clinically important adrenocorticoid suppression and a low incidence of clinically important hypotension.[29] Side effects include vomiting and myoclonic jerking, which are not usually clinically significant. The National Emergency Airway Registry (NEAR), a multicenter, prospective emergency medicine led registry, documented etomidate use in 42% of RSI procedures.[18] However, despite the growing amount of data to support the utility and safety of etomidate in children, it currently is not FDA approved in the pediatric population.

Paralytic Agents. Paralytic agents are a critical component of RSI. In one prospective study, intubation occurred successfully in 99% of adult patients with paralysis and in 82% without paralysis.[19] In children, intubation was successful 78% of the time with paralysis and only 44% of the time when only a sedative was used.[17] Although paralytic agents are essential to creating the optimal conditions for intubation, the potential complications of neuromuscular paralysis and inability to secure the airway must be seriously considered. Clinicians seem to use paralytic agents less frequently in children. In one study, paralytic agents were used for only 40% for pediatric intubations in children younger than 1 year.[18]

Paralytic agents are categorized into depolarizing and nondepolarizing agents. Depolarizing agents initially depolarize the motor endplate by acting at the acetylcholine receptor and induce contraction, manifested by fasciculations. Succinylcholine is the only depolarizing agent clinically available. The acetylcholine receptor is blocked, and binding is prevented until the succinylcholine is degraded by acetylcholinesterase. Nondepolarizing agents competitively inhibit the acetylcholine receptor. These agents are then degraded in the liver.

Succinylcholine (1.0-2 mg/kg IV) is the classic depolarizing agent. Its onset is rapid (30-60 seconds), and its duration is short (5-10 minutes). Succinylcholine may cause a rise in intracranial pressure or intraocular pressure, as well as an increase in airway secretions. These increased pressures caused by succinylcholine

Table 3. Indications for Endotracheal Intubation

ACUTE RESPIRATORY FAILURE
PO_2 <60 or Low SaO_2
PCO_2 > 50
Apnea
Hypoventilation
AIRWAY PROTECTION
Neurologic dysfunction (seizure, coma)
Loss of protective airway reflexes
Copious secretions, blood
Upper airway obstruction, airway edema or trauma
DECREASED WORK OF BREATHING
Hemodynamic instability
Metabolic acidosis
SPECIAL SITUATIONS/THERAPEUTIC INTERVENTION
Management of ICP
Conduction of necessary diagnostic tests (e.g. CT head)

Table 4. Rapid Sequence Intubation Modifications for Children

PHASE OF RSI	PEDIATRIC MODIFICATION
History and anatomic assessment (AMPLE)	
Preparation	Use length-based system for equipment organization, size determination, and medication dosages
Monitoring	Pediatric size EZ cap
Preoxygenation	1. 8 vital capacity breaths with high flow oxygen can be used if 5 minutes of preoxygenation not available. 2. Although adults may tolerate 4-5 minutes of apnea before hypoxia develops, children may tolerate only 2-3 minutes of apnea.
Premedication	1. Avoid bradycardia: - Atropine (0.01-0.02 mg/kg IV; min 0.1 mg; max 1 mg) 2. Avoid increased ICP: - Lidocaine
Assisted ventilation and cricoid pressure	
Sedation	1. Etomidate 0.3 mg/kg - Least cardiovascular effects 2. Ketamine 1-4 mg/kg - Contraindicated with increased ICP, but good to maintain blood pressure 3. Midazolam 0.05-0.1mg/kg* 4. Thiopental 0.5-4 mg/kg* * Can cause hypotension
Paralysis	1. Depolarizing neuromuscular blockage - Succinylcholine 1-2 mg/kg 2. Non-depolarizing neuromuscular blockade - Rocuronium 0.6 -1.2 mg/kg - Vecuronium 0.1-0.2 mg/kg
Intubation	
Confirmation of endotracheal tube placement	1. End-tidal CO_2, 2. O_2 saturation 3. EASY_CAP: - Purple: Unsuccessful - Tan: Questionable intubation - Yellow: Successful

are probably modest, and the clinical significance is controversial.[25,27] Complications of succinylcholine use include case reports of asystole, malignant hyperthermia, and hyperkalemia.[21,30]

Succinylcholine may induce an increase in serum potassium levels of approximately 0.5-1.0mEq/l.[30] This increase can be caused by two mechanisms. The first results from depolarization of the muscle and potassium leak. Although the significance of the increase in potassium level secondary to this mechanism may be less clinically important than previously documented, the effect is real.[31,33] The use of succinylcholine is contraindicated in patients who could have hyperkalemia (including patients with renal failure) or a significant burn or crush injury greater than 48 hours old. Succinylcholine also may cause hyperkalemia by receptor stimulation in patients with up-regulation of acetylcholine receptors and subsequent exaggerated release of potassium. This mechanism of hyperkalemia could occur in patients with spinal cord injury and neuromuscular diseases with wasting (e.g., muscular dystrophy).[31-39]

Rocuronium (0.6 to 1.2 mg/kg IV) is a nondepolarizing neuromuscular blocker. It has the shortest onset of all the nondepolarizing agents. The onset of rocuronium is almost comparable to succinylcholine (60-90 seconds) with a duration of action of 30-45 minutes. Meta-analysis showed that although rocuronium was inferior to succinylcholine in providing excellent intubating conditions, it was comparable to succinylcholine in inducing clinically acceptable intubating conditions.[34] A recent report looked at the use of rocuronium and found it to be safe compared with vecuronium and an effective agent for RSI when succinylcholine was contraindicated.[35,36]

Currently, there is controversy over whether to use succinylcholine, a depolarizing neuromuscular blocker, as the main paralytic agent in children.[25] However, despite the complications described in the literature, there is also a consensus that succinylcholine is the most reliable and rapid paralytic agent available for RSI. In patients in whom the use of succinylcholine may be unsafe (e.g., personal or familial history of malignant hypertension, skeletal muscle myopathies, extensive denervation of skeletal muscle or upper motor neuron injury), rocuronium is a documented safe alternative.

Cricoid Pressure

Cricoid pressure improves visualization of the vocal cords because the larynx will be displaced posteriorly; it also decreases the risk of aspiration. Cricoid pressure should be initiated as soon as sedation is started. Cricoid pressure prevents passive aspiration.[2, 37] If active vomiting occurs, cricoid pressure is to be lifted and the patient rolled to the side because esophageal perforation can occur.[38]

Intubation

Because the radius of the airway is inversely proportional to the resistance of flow, even a small amount of airway edema will result in a significantly increased resistance to flow causing increased work of breathing and possibly signs of respiratory distress in the pediatric patient. A child with any kind of upper res-

Table 5. Appropriate-Sized LMA Based Upon Patient Weight

WEIGHT (KG)	LMA SIZE
<5	1.0
5-10	1.5
10-20	2.0
20-35	2.5
35-55	3.0*
>55	4.0

* An ETT can be inserted into all LMAs, but removal of the LMA and extubation is tricky, often requiring destruction of the LMA. Separate intubating LMAs, designed for intubation through the LMA and subsequent removal of the LMA, exist for size 3.0 and above.

Adapted from Infosino A. Pediatric upper airway and congenital anomalies. *Anesthesiol Clin North Am* 2002;20(4):747-766.

piratory infection or repeated attempts at intubation may have an additional degree of restriction on the airway.

Any single intubation attempt should last no longer than 30 seconds to prevent profound hypoxemia.[2] Children, and especially infants, have smaller lung volumes and higher oxygen demands than adults, therefore, they consume their respiratory reserves more quickly than in adults.

Confirmation of Intubation

Disposable capnographic devices, (e.g., Easy-Cap) and end-tidal carbon dioxide monitors are mandatory adjuncts to evaluate endotracheal intubation. Visualization of the ETT being passed through the vocal cords, confirmation of proper placement by auscultation over the lung fields and the epigastric area, and vapor in the ETT are the initial nondefinitive confirmations of successful endotracheal intubation.; secondary confirmation is necessary.

Although Pediatric Advanced Life Support (PALS) guidelines do not recommend other confirmation devices in infants and children, devices such as the self-inflating rubber bulb have been found to be highly sensitive and specific for detection of esophageal intubation in adults. Two recent studies showed that the bulbs have high sensitivity and specificity in the pediatric patient population.[39,40]

Rescue Procedures

If RSI is started and the patient cannot be endotracheally intubated, the airway must be secured by other means. Most patients can be ventilated with BVM devices until the neuromuscular blockade has worn off. However, care must be taken not to inflate the stomach; a nasogastric tube should be placed to decompress the stomach.

Blind nasotracheal intubation should not be performed in children younger than 9 years.[20] It requires blind passage around a relatively acute angle in the naropharynx toward the anterosuperiorly located glottis, making intubation by this route difficult. The added potential for penetrating the cranial vault or damaging the more prominent nasopharyngeal adenoidal soft tissues, which results in hemorrhage, makes this procedure ill advised in a child. Additionally, it is a rare child who can cooperate with a nasotracheal intubation.

Awake intubations are performed on patients who potentially may be difficult to intubate and ventilate.[24] This procedure may be difficult in the pediatric patient due to behavioral and developmental inability to cooperate.

The laryngeal mask airway (LMA) can be an alternative means of securing the airway until a definitive airway can be obtained.[20,24,41-44] The LMA is a tube with a silicon cuffed mask-like portion on the distal end. It is inserted into the pharynx until resistance is encountered due to the hypopharynx. The mask is then inflated, sealing off the hypopharynx from the airway. The distal portion of the LMA is ideally situated above the airway, allowing ventilation and oxygenation to be performed. While the LMA does not optimally control the airway, it may be more protective than the BVM.[2,45] There is increasing literature, mostly in the form of case reports, documenting airway management success with LMA in children with abnormal anatomy in whom endotracheal intubation was difficult or impossible.[21] Intubation is possible through LMAs. Table 5 lists the LMA size based upon the child's weight.

Needle cricothyroidotomy with jet insufflation is an appropriate temporizing technique when airway access and control cannot be accomplished by BVM or orotracheal intubation in children younger than 8 years. The procedure consists of inserting a 12-14 gauge catheter through the cricothyroid membrane. The catheter is connected to a high pressure oxygen source (50 psi) and a one second burst of high pressure oxygen is given, followed by a 3-4 second period to allow for exhalation. Care must be taken to hold onto the catheter so that it stays in position. The catheter also may kink secondary to the high pressures; nonkinking catheters with metal spirals are availble. If the patient cannot spontaneously exhale, then jet insufflation is contraindicated. Chest movement should be used as a visible indicator of adequate ventilation. This procedure provides oxygenation but does not provide adequate ventilation, and progressive hypercarbia may occur. Complications include tension pneumothorax.

Emergency cricothyroidotomy is the airway of last resort in an adult with difficult airway. It is used often in patients with extensive facial and upper-airway injury. Cricothyroidotomy is difficult in children and contraindicated for an infant or small child. It only can be performed in an older child in whom the cricothyroid membrane is easily palpable, usually by the age of 8-10 years.[21] There are no data specifically on the complication rates in the pediatric population. But the overall complication rate of cricothyrotomy depends upon technique. In one recent small study, there were no reports of complications associated with the rapid four-step technique when performed in the ED; nonrapid four-step cricothyrotomies in the ED had an associated complication rate of 25%.[46]

Other alternative airways in children have similar indications and procedures as in adults. Retrograde intubation has been described in adults and is a rapid and safe technique; the only true contraindication is a patient who cannot be adequately oxygenated or ventilated.[24,47] The cricothyroid membrane is identified and pierced with an 18-gauge needle aimed cephalad. When air is aspirated, a guidewire is threaded through the needle until the proximal end emerges in the mouth.[21,24] The needle is removed, and a plastic sheath is passed over the wire until resistance is met. Then, the ETT is advanced over the wire, and the wire and sheath are removed from the mouth.[24] In children, a 20-gauge IV catheter with a 0.0021-inch J wire is adequate.[21] Retrograde intubation sets are available, however, an 18-gauge or larger needle and 80 cm J tipped guidewire can be used.[24] If using a central line kit, care must be taken that the J wire is of adequate length.

Lighted stylet, bullard larygoscope, ETT introducer, and fiber optic laryngoscope are all alternative airway techniques that may be used in a child with a difficult airway.[24]

Special Circumstances

Head Trauma. Closed head injury is the most common type of injury seen in pediatric trauma, and consummate airway management is necessary to optimize functional outcome.[5] The outcome of head trauma in children is better than in adults, but the outcome in children younger than 3 years is worse than that of a similar injury in an older child.[14] This difference is probably because young children are particularly susceptible to the effects of secondary brain injury that may be produced by hypovolemia and hypoxia. Maintaining oxygenation and preventing hypercarbia are critical in managing the pediatric head-injured trauma patient.

Cervical Spine Injury. Although the overall incidence of cervical spine injury is low, weak neck muscles, a higher fulcrum (C2-C3), and poor protective reflexes in children contribute to higher level cervical spine injuries than in adults.[20] In addition, cervical spine injuries may not appear on radiographs, therefore, there must be a high suspicion for cervical spine injuries in children with trauma.[5] Cervical spine immobilization may make airway management more difficult. The chin lift and jaw thrust maneuvers, as well as airway adjuncts, must be used in lieu of the head tilt. The larger occiput also may make immobilization of the child's head, neck, and spine in neutral position more difficult (*Figure 2*).

Laryngeal Injury. The difference in laryngeal anatomy in "position, consistency, size and shape" explains the types of injury that occur in children.[48] Laryngeal injuries are less common in children than in adults because of the relatively high location of the larynx in the neck, which allows the airway to be protected by the mandibular arch. A child also has a relatively short neck compared with that of an adult.[49] Up to 80-95% of serious laryngeal injuries in children occur in adolescents, secondary to blunt mechanisms typically resulting from accidents involving motorcycles, snowmobiles, water craft; and clothesline and direct blows during karate, snowboarding, and skating.[48,50] Because of the overall shape and smaller size, the pediatric patient will tolerate traumatic disruption less well than an adult.[48]

A high degree of suspicion is required to diagnose a laryngotracheal separation. Laryngotracheal separation commonly occurs within 2-3 cm of the carina.[49] In the ED, diagnostic assessment should include assessment of airway sounds (e.g., stridor, hoarseness, aphonia, cough, hemoptisis, respiratory distress), cyanosis, subcutaneous air, neck hematoma or tenderness, tracheal deviation, and cartilagenous stepoffs. If feasible, flexible fiberoptic endoscopy is preferred before oral tracheal intubation to avoid complete disruption of the larynx. However, if airway management is emergent—given that cricothyroidotomy is contraindicated in children younger than 10 years—and jet insufflation is fraught with complications, orotracheal intubation, if possible, is the preferred and relatively safe airway management procedure prior to definitive exploration and repair in the operating room.[51]

Tracheobronchial Injuries. Tracheobronchial injuries are also rare.[51] The low incidence is due to the elasticity of the mediastinum and tracheobronchial tree, which allows it to be compressed without causing injury.[51] In adults, bronchial injuries tend to occur in the right-sided main stem bronchus but this predominance is not seen in the pediatric population.[52,53] Therefore, these types of injuries are seen only with major accidents (e.g., falls from high levels or being thrown out of motor vehicles).[53] If tracheobronchial injuries are suspected, endoscopy is mandatory.[49,54]

Blunt Thoracic Injuries. Blunt thoracic injuries are the second leading cause of death from pediatric trauma.[12] Injuries to the vital thoracic structures—not including lung contusions—have a mortality rate of greater than 50%. Multiple studies have shown the most common injury with pediatric blunt chest trauma is a pulmonary contusion. Other common injuries included rib fractures, pneumothorax, and hemopneumothorax. In one study, 25% of the pneumothorax cases were tension pneumothorax.[13] The high incidence of tension pneumothorax may be due to the increased pliability of the pediatric chest.

Children with Special Health Care Needs. Children with apparent Down syndrome facies who undergo trauma should be managed carefully. Children with Down syndrome have a relatively complicated airway due to a large tongue and relative hypotonia. A curved blade may facilitate moving the tongue out of the way. The trachea is usually smaller than in other children of the same age, necessitating an ETT 1-2 sizes smaller than calculated.[55] Atlanto-axial instability also may be present, making them more prone to cervical spine injury.[15] Cervical spine immobilization is particularly important in these cases to prevent further injury.[20] In some cases, an LMA may be the best airway management technique. Children with congenital problems (e.g., Pierre Robin syndrome, tracheomalacia, or cleft palate) who are involved in trauma may have a predictably difficult airway. An LMA may be the best option in these cases because surgical airways are contraindicated in the young child.[21] As stated in the section on RSI, children with neuromuscular diseases should be intubated with a nondepolarizing neuromuscular blockade.

Nonaccidental Trauma. When managing the traumatic airway, the practitioner should always consider the mechanism of the trauma and the possibility of nonaccidental trauma.

Summary

Understanding the differences between the adult and pediatric airway and how these affect airway management in trauma is of critical importance in the timely and appropriate resuscitation of the pediatric trauma patient. This report highlights the specific anatomical and physiologic features unique in children and the implications for pediatric trauma management.

References

1. U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics. Vital Statistics of the United States, 2002 In: *National Vital Statistics Report.* Hyattsville, MD;2005;13.
2. Hazinski R. Mary Fran Pediatric Advanced Life Support. Dallas, Texas: American Heart Association;2002:49-50.
3. Luterman A, et al. Evaluation of pre-hospital emergency medical service: Defining areas for improvement. *J Trauma* 1983;23:702-707.
4. Ramenofsky ML, et al. Maximum survival in pediatric trauma: The ideal system. *J Trauma* 1984;24:818-823.
5. Stallion A. Initial assessment and management of pediatric trauma patient. *Respir Care Clin N Am* 2001;7:1-11.
6. Infosino A. Pediatric upper airway and congenital anomalies. *Anesthesiol Clin North America* 2002;20:747-766.
7. Norton M. *Atlas of the Difficult Airway*. Second ed. St Louis: Mosby Year Book Inc;1996.
8. Fastle RK, Roback MG. Pediatric rapid sequence intubation: Incidence of reflex bradycardia and effects of pretreatment with atropine. *Pediatr Emerg Care* 2004;20:651-655.
9. Newth CJ, et al. The use of cuffed versus uncuffed endotracheal tubes in pediatric intensive care. *J Pediatr* 2004;144:333-337.
10. Luten R. The pediatric patient. In: Walls RM, ed. *Manual of Emergency Airway Management*. First ed. Philadelphia: Lippincott Williams And Wilkins;2000;143-163.
11. Myer MC, Cotton MR, Shott MS. *The Pediatric Airway: An Interdisciplinary Approach*. Philadelphia: JB Lippincott Company;1995.
12. Cooper A. Thoracic injuries. *Semin Pediatr Surg* 1995;4:109-115.
13. Nakayama DK, Ramenofsky ML, Rowe MI. Chest injuries in childhood. *Ann Surg* 1989;210:770-775.
14. Surgeons AC. *Advanced Trauma Life Support: Faculty Manual.* Seventh ed. Chicago: American College of Surgeons;2004:153-158.
15. Agarwal S et al. Comparing the utility of a standard tediatric resuscitation cart with a pediatric resuscitation cart based on the Broselow tape: A randomized, controlled, crossover trial involving simulated resuscitation scenarios. *Pediatrics* 2005;116 (3):e326-33.
16. Hohenhaus SM, Frush KS. Pediatric patient safety: Common problems in the use of resuscitative aids for simplifying pediatric emergency care. *J Emerg Nurs* 2004;30:49-51.
17. Sagarin MJ, et al. Rapid sequence intubation for pediatric emergency airway management. *Pediatr Emerg Care* 2002;18:417-423.
18. Chng Y, et al. Pediatric emergency airway management. *Acad Emerg Med* 2004;11:438-439.
19. Li J, et al. Complications of emergency intubation with and without paralysis. *Am J Emerg Med* 1999;17:141-143.
20. Yamamoto L. Emergency airway management-rapid sequence intubation. *Textbook of Pediatric Emergency Medicine.* Fourth ed. Fleisher G, Ludwig S, eds. Philadelphia: Lippincott Williams and Wilkins;2000;71-82.
21. Gerardi MJ, et al. Rapid-sequence intubation of the pediatric patient. Pediatric Emergency Medicine Committee of the American College of Emergency Physicians. *Ann Emerg Med*, 1996;28:55-74.
22. Cook D. Can succinylcholine be abandoned? *Anesth Analg* 2000;90:S24-28.
23. Robinson N, Clancy N. In patients with head injury undergoing rapid sequence intubation, does pretreatment with intravenous lignocaine/lidocaine lead to an improved neurological outcome? A review of the literature. *Emerg Med J*, 2001;18:453-457.
24. Kaide C, Hollingsworth J. Current strategies for airway managment in the trauma patient Part II. *Trauma Rep* 2003;4(1):1-9.
25. Zelicof-Paul, A, et al. Controversies in rapid sequence intubation in children. *Curr Opin Pediatr* 2005;17(3):355-362.
26. Fleming B, McCullough M, Henderson SO. Myth: Atropine should be administered before succinylcholine for neonatal and pediatric intubation. *Canadian J Emerg Med* 2005;7(2):114-117.
27. Wadbrook PS. Advances in airway pharmacology. Emerging trends and evolving controversy. *Emerg Med Clin North Am*, 2000;18(4):767-788.
28. Schenarts CL, Burton JH, Riker RR. Adrenocortical dysfunction following etomidate induction in emergency department patients. *Acad Emerg Med* 2001;8(1):1-7.
29. Sokolove, PE, Price DD, Okada P. The safety of etomidate for emergency rapid sequence intubation of pediatric patients. *Pediatr Emerg Care* 2000;16(1):18-21.
30. Thapa S, Brull SJ. Succinylcholine-induced hyperkalemia in patients with renal failure: An old question revisited. *Anesth Analg* 2000;91(1):237-241.
31. Larach M, Rosenberg H, Gronert G. Hyperkalemic cardiac arrest during anesthesia in infants and children with occult myopathies. *Clin Pediatr* 1997;36:9-16.
32. Gronert B, Brandom B. Neuromuscular blocking drugs in infants and children. *Pediatr Clin North Am*;1994;41:73-91.
33. Kerr T, Durward A, Hodgson S. Hyperkalemic arrest in a manifeting carrier of Duchenne muscular dystrophy following general anesthesia. *Eur J Ped* 2001;169:579-580.
34. Perry J, Lee J, Wells G. Rocuronium versus succinylcholine for rapid sequence induction intubation. *Cochrane Database Syst Rev*, 2003(1):CD002788.
35. Mendez DR, et al. Safety and efficacy of rocuronium for controlled intubation with paralytics in the pediatric emergency department. *Pediatr Emerg Care* 2001;17(4):233-236.
36. Perry JJ, Lee J, Wells G. Are intubation conditions using rocuronium equivalent to those using succinylcholine? *Acad Emerg Med*, 2002;9(8):813-823.
37. Moynihan RJ, et al. The effect of cricoid pressure on preventing gastric insufflation in infants and children. *Anesthesiology* 1993; 78(4):652-656.

38. Mahadevan S, Sovndal S. *Principles of Emergency Medicine*. First ed. Cambridge, UK: Cambridge University Press. 2005:19-45, 96.
39. Sharieff GQ, et al. The self-inflating bulb as an esophageal detector device in children weighing more than twenty kilograms: A comparison of two techniques. *Ann Emerg Med* 2003;41(5):623-629.
40. Sharieff GQ, et al. The self-inflating bulb as an airway adjunct: Is it reliable in children weighing less than 20 kilograms? *Acad Emerg Med* 2003;10(4):303-308.
41. Parmet JL, et al. The laryngeal mask airway reliably provides rescue ventilation in cases of unanticipated difficult tracheal intubation along with difficult mask ventilation. *Anesth Analg* 1998;87(3):661-665.
42. Levy RJ, Helfaer MA. Pediatric airway issues. *Crit Care Clin* 2000; 16(3):489-504.
43. Grein AJ, Weiner GM. Laryngeal mask airway versus bag-mask ventilation or endotracheal intubation for neonatal resuscitation. *Cochrane Database Syst Rev* 2005(2):CD003314.
44. Lopez-Gil M, Brimacombe J. The ProSeal laryngeal mask airway in children. *Paediatr Anaesth* 2005;15(3):229-234.
45. Stone BJ, Chantler PJ, Baskett PJ. The incidence of regurgitation during cardiopulmonary resuscitation: A comparison between the bag valve mask and laryngeal mask airway. *Resuscitation* 1998; 38(1):3-6.
46. Bair AE, et al. Cricothyrotomy: A 5-year experience at one institution. *J Emerg Med* 2003;24(2):151-156.
47. Hung O, Murphy M. Lightwands, lighted styles and blind techniques of intubation. *ACNA* 1995;13:477-489.
48. Myer CM 3rd. Trauma of the larynx and craniofacial structures: Airway implications. *Paediatr Anaesth* 2004;14(1):103-106.
49. Granholm T, Farmer DL. The surgical airway. *Respir Care Clin* N *Am* 2001;7(1):13-23.
50. Mandell D. Traumatic emergencies involving the pediatric airway. *Clin Pediatr Emerg Med* 2005;6:41-48.
51. Gussack GS, Jurkovich GJ, Luterman A. Laryngotracheal trauma: A protocol approach to a rare injury. *Laryngoscope* 1986;96(6): 660-665.
52. Ein SH, et al. Traumatic bronchial injuries in children. *Pediatr Pulmonol* 1986;2(1):60-64.
53. Slimane MA, et al. Tracheobronchial ruptures from blunt thoracic trauma in children. *J Pediatr Surg* 1999;34(12):1847-1850.
54. Hancock BJ,Wiseman NE. Tracheobronchial injuries in children. *J Pediatr Surg* 1991;26(11):1316-1319.
55. Shott S. Down syndrome: Analysis of airway size and a guide for appropriate intubation. *Laryngoscope* 2000;110(4):585-592.

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you <u>must</u> complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

CE/CME Objectives

Upon completing this program, the participants will be able to:

a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

Physician CME Questions

1. At what age is a child's airway considered to be equivalent to an adult's airway?
   A. 5 years
   B. 8 years
   C. 12 years
   D. 16 years

2. Which one of the following paralytic agents is the first choice for pediatric rapid sequence intubation?
   A. Rocuronium
   B. Vecuronium
   C. Succinylcholine
   D. Isradipine

3. An 8-year-old male lost his grip while climbing over a fence and fell chest forward into the fence. He was brought to the emergency department by his mother. He has a puncture wound to his left chest made by the top of the fence. Initially, the patient was breathing comfortably with equal breath sounds. While awaiting chest x-ray, his breathing pattern changed. What would be the first mechanism of respiratory compensation in a pediatric patient in respiratory distress?
   A. Increased work of breathing
   B. Wheezing
   C. Tachypnea
   D. Head bobbing

4. You choose succinylcholine as your paralytic agent to intubate a 6-year-old child involved in a serious motor vehicle accident. Which one of the following medications is recommended before giving succinylcholine to this patient?
   A. Lidocaine
   B. Atropine
   C. Robinul
   D. Midazolam

5. An 8-year-old male with Down syndrome fell from the top of a play structure and hit his head on one of the platforms as he fell. Which

one of the following statements is true when considering intubation of a Down syndrome patient?

A. A straight blade may facilitate moving the tongue out of the way.

B. An ETT 1-2 sizes larger than calculated may be necessary for intubation.

C. These children are more prone to cervical spine injury.

D. An LMA is contraindicated in the Down syndrome population.

6. A 10-year-old female was brought in after falling in gymnastics class from the balance beam. The coach reported that she hit her head on the edge of the balance beam when she attempted to dismount. She arrived by ambulance wearing a cervical spine collar. When you initially examined her, she was responsive and cooperative to your questions with only complaints of pain on the neck. As you were finishing your primary survey, she became increasingly uncooperative and agitated. Which of the following is the *most* important initial step?

A. Re-assess her pupillary response

B. Re-assess her respiratory status

C. Prepare for intubation

D. Rush the patient for a CT scan

7. Which one of the following statements is true about the use of atropine in children?

A. Atropine is used in children younger than 1 year only if succinylcholine is the paralytic agent.

B. Atropine is not indicated for use in adolescents, except for bradycardia.

C. Atropine is given to prevent bradycardia resulting from vagal stimulation.

D. The correct dosing of atropine for a child is 0.1-0.2 mg/kg IV.

E. The maximum dose of atropine in the pediatric population is 2 mg.

8. A completely unresponsive 4-month-old child is brought into the emergency department by his parents. The parents reported he just suddenly "stopped breathing." You can see several bruises on his chest and head. You decide to intubate. Which one of the following procedures is appropriate for positioning this child for intubation?

A. Perform head tilt

B. Place a shoulder roll

C. Place the arms to the side

D. Extend the head back

9. When placing an oral airway in a child, which of the following statements is true?

A. For correct placement, insert the oral airway backward and rotate it into position.

B. Oral airways have no contraindications in the pediatric population.

C. Emesis may occur with oral airways in patients who are conscious.

D. For patients with relatively large tongues, use a smaller oral airway than recommended by age.

10. Which of the following statements regarding the preoxygenation phase of rapid sequence intubation of a child is true?

A. A child can tolerate up to 5 minutes of apnea before developing hypoxia.

B. Preoxygenation is the first step of rapid sequence intubation.

C. Five vital capacity breaths with high flow oxygen can be used if 5 minutes of preoxygenation is not available.

D. Any single intubation attempt should last no longer than 30 seconds.

Answers:

1. B
2. C
3. C
4. B
5. C
6. B
7. C
8. B
9. C
10. D

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Dear *Trauma Reports* Subscriber:

This issue of your newsletter marks the start of a new continuing medical education (CME) or continuing education (CE) semester and provides us with an opportunity to review the procedures.

***Trauma Reports*, sponsored by Thomson American Health Consultants,** provides you with evidence-based information and best practices that help you make informed decisions concerning treatment options and medical practices. Our intent is the same as yours — the best possible patient care.

The objectives of *Trauma Reports* are to:

1. Discuss conditions that should increase suspicion for traumatic injuries;
2. Describe the various modalities used to identify different traumatic conditions;
3. Cite methods of quickly stabilizing and managing patients; and
4. Identify possible complications that may occur with traumatic injuries.

Each issue of your newsletter contains questions relating to the information provided in that issue. After reading the issue, answer the questions at the end of the issue to the best of your ability. You can then compare your answers against the correct answers provided in an answer key in the newsletter. If any of your answers were incorrect, please refer back to the source material to clarify any misunderstanding.

Enclosed in this issue is an evaluation form to complete and return in an envelope we will provide. Please make sure you sign the attestation verifying that you have completed the activity as designed. Once we have received your completed evaluation form we will mail you a letter of credit. This activity is valid 24 months from the date of publication. The target audience for this activity is emergency, trauma, and surgical physicians and nurses.

If you have any questions about the process, please call us at (800) 688-2421, or outside the U.S. at (404) 262-5476. You can also fax us at (800) 284-3291, or outside the U.S. at (404) 262-5525. You can also email us at: ahc.customerservice@thomson.com.

On behalf of Thomson American Health Consultants, we thank you for your trust and look forward to a continuing education partnership.

Sincerely,

Brenda L. Mooney

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Vice-President/Group Publisher
Thomson American Health Consultants