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Apparent life-threatening events (ALTE) are terrifying episodes for both the family and the emergency medicine physician. The authors provide a practical approach to recognition, differentiation, and management of these events. We are also reminded that approximately 50% of the cases will have an identifiable medical or surgical cause for the event. The emphasis on a systematic, diagnostic evaluation and comprehensive treatment program will allow the emergency physician a greater degree of comfort when confronted with a child with an ALTE.

—The Editor

Apparent Life-Threatening Events: Recognition, Differentiation, and Management

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Introduction

Infants with an apparent life-threatening event (ALTE) present for medical attention because an acute and unexpected change in behavior alarmed the caregivers. These frightening episodes of apnea and color change in infants have generated considerable concern in both professional and lay groups. Questions have focused on the significance of these episodes. The hypothesis that these events might be premortem markers of the infant at high risk of sudden infant death syndrome (SIDS) explains the large amount of attention, research, and clinical resources that have been focused on the problem of ALTEs in infants.[1]

Definition

The current definition of ALTE was established by the 1986 National Institutes of Health Consensus Development Conference on Infantile Apnea and Home Monitoring:[2]

An episode that is frightening to the observer and is characterized by some combination of apnea (central or occasionally obstructive), color change (usually cyanotic or pallid but occasionally erythematous or plethoric), marked change in muscle tone (usually marked limpness), choking, or gagging. In some cases, the observer fears that the infant has died. Previously used terminology such as "aborted crib death"or "near-miss SIDS" should be abandoned because it implies a possibly misleading, close association between this type of spell and SIDS.

Given the subjective nature of caregiver observations and interpretation, the initial medical problem is to determine which descriptive variations of the reported episodes place the infant at increased risk for sudden death and future life-threatening episodes and which are a reflection of parental anxiety or of an acute, nonrecurring problem. Consistent with the recommendations of the NIH Consensus Development Conference, this differentiation is often made by assuming that reports of

"frightening" episodes of apnea or respiratory difficulty are clinically of greater severity, and, thus, more apt to be of medical significance if the parents also observe a change in skin color or muscle tone or provide vigorous stimulation, mouth-to-mouth resuscitation, or CPR.[3] Conversely, it is often concluded that the reported episode is not medically important if it is not associated with these latter observations or does not require some type of vigorous resuscitative intervention. This assumption is made, although studies have demonstrated that parental reports of an acute event are a poor reflection of an infant's physiologic status.[1,3-5] There is a need for research studies to critically examine the clinical implications of the ALTE characteristics by employing a variety of objectively measured outcomes. Until such a time as there are objective means of adequate sensitivity and specificity to assist in making medical decisions (e.g., hospital admission, specialized clinical follow-up, home apnea monitoring), it may be necessary to primarily rely on an expression of parental concern that they had observed an episode sufficiently frightening to them to bring the infant to medical attention.[3]

Table 1. ALTE—Known Origin

INFECTION
Sepsis/meningitis
Respiratory syncytial virus
Pertussis
Other respiratory infections
GASTROINTESTINAL
Gastroesophageal reflux
Pharyngeal incoordination stimulating laryngeal chemoreceptors
Malformations
SEIZURE
BREATH-HOLDING SPELLS
Cyanotic
Pallid (vasovagal syncope)
OTHER NEUROLOGIC DISORDERS
CNS tumor
Subdural hemorrhage
Apnea associated with Arnold-Chiari malformations
CARDIAC ARRYTHMIAS
ABNORMALITIES OF RESPIRATORY DRIVE
Immature respiratory center
Apnea of prematurity
Respiratory center dysfunction
Central hypoventilation syndrome (Ondine's curse)
Drug exposed infants
OBSTRUCTIVE SLEEP APNEA
METABOLIC AND ENDOCRINE
Hypoglycemia
Hypocalcemia
Abnormal metabolism of fatty acids
CHILD ABUSE
MUNCHAUSEN SYNDROME BY PROXY

Incidence and Relationship to SIDS

Various estimates place the incidence of ALTEs in the general population at between 0.5% and 6.0% of all infants.[1] The higher ranges of reported incidence (4-6%) come from studies in which parents were questioned as to whether their infant had ever turned blue or stopped breathing. However, this mode of ascertainment tends to overestimate incidence.[1,6] For a multitude of reasons, the true frequency and the prevalence of ALTEs are unknown.[6]

The potential relation of ALTE and SIDS is debated. Most studies report an incidence of prior ALTE in SIDS victims of about 5-10%.[1,6-8] Therefore, most SIDS victims do not come from the ALTE population. Compared with SIDS victims, infants with ALTE present earlier; they are 1-3 weeks younger than SIDS patients.[6] Also, contrary to SIDS victims, 82% of the

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Table 2. ALTE Evaluation

Careful history from the person witnessing the event

Color
- red
- pale
- cyanotic

Respiratory Efforts
- apnea
- obstructed
- irregular

Sleep state
- awake
- asleep

Position
- prone
- supine
- upright

Noises
- stridor
- choking

Eye Movements
- closed
- startled
- rolled
- fluttering

Relationship to feeding

Fluid in mouth?

Duration

Need for intervention

Obtain medical history and family history *(see text)*

Table 3. Suggested Evaluation of Infants with ALTE

ALL INFANTS
- Inhospital observation with cardiorespiratory monitoring
- Careful physical and neurologic examination
- Complete blood count
- Blood glucose, electrolytes, calcium, phosphate, bicarbonate, magnesium, and ammonia
- Chest roentgenogram
- Electrocardiogram
- Evaluation of cardiorespiratory function. Multichannel recording with pulse oximetry is essential in such instances

SELECTED INFANTS UNDER CERTAIN CLINICAL CIRCUMSTANCES
- Septic workup (blood, urine, CSF cultures)
- Barium swallow
- Laryngoscopy, bronchoscopy
- Radionuclide milk scan of swallowing
- Esophageal pH study with multichannel recording
- Ultrasound or computerized tomography scan of the brain
- Arterial blood gases
- Echocardiogram
- Electroencephalogram
- Polysomnogram
- Evaluation for inborn errors of metabolism
- Holter monitoring

ALTE events occurred between 800 and 2000 hours, when the caretakers are awake.[6]

Considered from a different, and perhaps more important, perspective, the incidence of sudden and unexpected death in ALTE infants appears to vary depending on the initial presentation. Those infants whose frightening episode occurred during sleep and who were perceived to require cardiopulmonary resuscitation had a 10-13% risk of subsequent sudden unexpected death, even with the prescription of home monitoring devices.[2,8] Infants who responded only to resuscitation and have a subsequent similar episode are siblings of victims of SIDS or develop a seizure disorder during monitoring and have a greater than 25% risk of dying.[4] The precise risk for infants who experience less severe ALTE events has not been determined, but in many cases it may be no greater than the general population. Identification of a specific cause of ALTE does not necessarily eliminate the risk of sudden and unexpected death.[2]

In spite of the fact that the relationship of ALTEs and SIDS will long be debated, it is well accepted that following such an episode, infants are at risk of further episodes and also of SIDS.[9]

Etiology

There are multiple identifiable causes of ALTEs; after evaluation, approximately 50% of these infants will have a specific diagnosis.[1,2,6,8] Table 1 outlines the major clinical diagnostic groups. Symptoms reported by parents greatly contribute to establishing most diagnoses.[6] As can be seen after reviewing Table 1, ALTE can be a symptom of many specific disorders including gastroesophageal reflux, infection, seizures, airway abnormalities, hypoglycemia, or other metabolic problems, as well as impaired regulation of breathing during sleep and feeding. These episodes can occur during sleep, wakefulness, or feeding and occur in infants who are generally of greater than 37 week's gestational age at the time of onset.[2]

Diagnosis

Infants usually appear entirely normal by the time they reach medical attention following the ALTE. The most important initial diagnostic step is to obtain a careful history from the person witnessing the event. *(See Table 2.)* One should specifically ask about the infant's color, tone, apnea, and the need for intervention. Was the infant awake or asleep? How long did the event last?

The patient's medical history should also be probed for evidence of previous frightening episodes, abnormal breathing patterns, feeding problems, seizures, perinatal insults, and other medical problems. A family history of apnea, infant deaths, seizures, or other cardiopulmonary problems should be explored.

After a careful history and physical examination are performed, the physician is in a position to make an initial judgment about whether the event represents an ALTE or not. If not, no further diagnostic evaluation is usually required. If a physician believes that the infant did have an ALTE, then an aggres-

Table 4. Indications for Home Cardiorespiratory Monitoring
• History of severe, life-threatening event of apneic episode
• Multichannel recording documentation of osbtructive apnea
• Sibling of a SIDS victim
• Twin of a SIDS victim
• Severe feeding difficulties with apnea and bradycardia
• Gastroesophageal reflux-associated apnea
• Technology-dependent patient
• Patient with certain pulmonary, cardiac, or neurologic problems
• Some infants exposed to cocaine or opiates in utero

sive approach to identifying the etiology of the event and instituting appropriate therapy is necessary. Hospital admission for protective monitoring, to facilitate the diagnostic evaluation, and for parent training is recommended.[6,8,10] The diagnostic evaluation should include, but is not limited to, the items listed in Table 3. Although not every infant will require all of these tests, many of them are often performed before the episodes are termed "unexplained."

Management

Infants in whom a treatable cause of ALTE can be identified are best managed by treating the specific etiology of the event. *(See specific disease section.)* Occasionally, some may require the addition of home apnea-bradycardia monitoring if ALTE cannot be controlled despite specific treatment.[2,8,10] *(See Table 4.)*

The diagnosis of apnea of infancy (AOI) is used when an identifiable cause for the ALTE cannot be found.[2] There are presently no specific treatments for AOI, thus, home apnea-bradycardia monitoring for these infants is recommended. Although scientific studies have not been performed to prove the efficacy or lack of efficacy of home apnea-bradycardia monitoring in saving the lives of these infants, they have a high risk for subsequent apneas, and home monitoring is used to detect these episodes. Thus, home apnea-bradycardia monitoring is instituted in infants, with AOI as the primary mode of management.[2,8,10]

Parents or caregivers are instructed to used home monitors whenever the infant is not being otherwise observed. They are most commonly used during sleep, in automobiles, and at other times when the infant is unobserved. Alarms are set to sound for central apneas greater than 20-seconds and/or bradycardia less than 80 beats/min in the first month of life, less than 60-70 beats/min from 1-3 months, less than 60 beats/min from 3-12 months, and less than 50 beats/min thereafter. The high heart rate alarm can be set as high as possible on the individual monitor, unless the child is being monitored for a tachyarrhythmia.[10] Home monitors do not sound an alarm for obstructive apnea events, unless they are accompanied by bradycardia.

Home monitors only alert the caregiver that a potential episode is occurring. The caregiver must then respond to evaluate and/or terminate the episode. Parents and caregivers must be trained in the proper operation of the monitor, a graded response to monitor alarms, and infant cardiopulmonary resuscitation. Thorough education of parents and psychosocial support of families using monitors are important for successful home apnea-bradycardia monitoring. Monitoring can be both a source of stress and a source of support and reassurance for parents.

Documented Monitoring (Event Recordings). It may be difficult to distinguish true apnea or bradycardia alarms from loose lead alarms or alarms for nonsignificant events. Documented monitoring, with event recorders built into the monitors, provide objective recordings of apnea and bradycardia alarms, and may be helpful in making these distinctions.[11] Documented monitoring allows the clinician to know whether or not the infant was actually having an apnea or bradycardia during a given alarm. In addition, it provides information regarding compliance with monitor use, because the length of time the monitor was turned on each day is recorded. In a large, consecutive series of referred monitored patients, it was found through event recordings that only 8% of 14,131 events were caused by true apnea or bradycardia.[11] Of these true events, 70% were triggered by apnea and 30% by bradycardia. These true events occurred in 48% of the patients. Loose lead alarms accounted for 69% of all events and false alarms for 23%. The most common reason for a false alarm was a low amplitude signal.[11] Therefore, in many instances, alarms viewed as real by the caregivers can be shown to be false, and the time of monitoring may be decreased.[12-14] However, documented monitoring may also help confirm the caregiver's observations regarding the clinical significance of episodes. Compliance with monitoring may be enhanced with documented monitoring because the physician has access to data on monitor use.

Outpatient Management. After discharge, AOI infants should be followed on an outpatient basis every 1-2 months. Parents are requested to keep a log or diary of all apnea and bradycardia alarms which were thought to be real, especially those requiring intervention. During outpatient visits, the clinician reviews these logs with the family and compares them with event recordings, if available. It is important to note the infant's pattern of alarms during physical stresses, such as fatigue or respiratory infections. Parents should bring the home monitor to clinic with the infant. This allows monitor settings to be changed easily, or minor troubleshooting to be performed. The usual clinical pattern is that true alarms will decrease in both frequency and severity with time. Infants whose alarms become more frequent or severe, those infants with multiple alarms requiring intervention, and those infants who continue to have true alarms after 6-8 months of monitoring require further diagnostic evaluation, including home event recordings with oximetry or overnight polysomnography.[12,15,16] With severe episodes, these infants may require hospitalization for observation and further diagnostic evaluation. Sometimes the character of the events may change, suggesting the presence or development of other clinical problems, such as a seizure disorder, which also require specific evaluation.

Outpatient visits are used to reinforce parent education and compliance. Parent response to monitor, alarms including infant cardiopulmonary resuscitation, should be reviewed at each outpatient visit. It is important to emphasize compulsive use of the home monitor at all times when the infant is not being otherwise observed.[4] Because of this, parents often find it difficult to stop

home monitoring once their infant no longer requires it. Parents are taught the criteria for monitor discontinuation. Therefore, parents can begin psychological preparation for the time when home monitoring can be safely discontinued.

The criteria for monitor discontinuation should be based on the infant's clinical condition. Clinical experience and the literature support monitor discontinuation when infants with ALTEs have had 2-3 months without significant alarms or apnea.[2] Additionally, assessing the infant's ability to tolerate stress (e.g., immunizations, illnesses) during this time is advisable.[8]

Specific Causes of ALTE

Infections. Apnea is the initial presenting symptom in some infants with respiratory syncytial virus (RSV) infections.[17-19] Although the apnea can be life-threatening, episodes usually are of short duration and occur during the first week of illness. Apnea in infants with RSV bronchiolitis occurs most commonly in young infants (less than 3 months) with a history of premature birth and apnea of prematurity.[17,19] The apnea occurs primarily in quiet sleep and is not obstructive. Infants with bronchiolitis also may develop apnea from increased work of breathing, respiratory muscle fatigue, and hypoxemia.[18] Apnea may also complicate other infections, including meningitis, encephalitis, bacterial sepsis, botulism, pertussis, and other respiratory infections.[20]

Seizure-Induced ALTE. Seizures can be associated with apnea and hypoxemia. Seizures have been shown to occur in 4-7% of infants with ALTE, and such infants have a particularly high risk of sudden death.[21] However, it is often difficult to prove whether seizures resulted from or caused an ALTE. Usually, the possibility of seizure-induced events are considered on clinical grounds (e.g., clustering of attacks, facial/eye movements, or an increase in muscle tone), however, apnea may occur as the only clinical manifestation of seizures in neonates, infants, and young children.[21-23]

ALTE that results from a seizure disorder frequently follows a characteristic pattern when carefully studied. First there is a change in the electroencephalogram (EEG), followed by one or more pauses in breathing movements, and, ultimately, a decrease in oxygen saturation in the presence of sinus tachycardia.[21]

Airway Obstruction. Investigators using overnight polysomnography have found that following an ALTE, infants have increased obstructive and mixed apneas compared to non-risk groups and that some infants who have had ALTEs later die of SIDS.[9,24-26] The obstructed breaths occurred mainly in rapid eye movement sleep and were accompanied by drops in heart rate and saturation.[24] The future SIDS victims were also noted to have more frequent episodes of regurgitation after feeding and to move less during sleep.[24] Such findings are in agreement with reports suggesting that although obstructive apneas are rare in normal infants,[27] they may play a role in ALTE and in SIDS.[6,9,24-26] Obstructive apneas may be related to craniofacial malformations, gastroesophageal reflux, bulky arytenoid apparatus, central nervous system disorder, or vascular malformations.[1,6,9,28] Recent evidence suggests that obstructive sleep apnea (OSA) in adults and sudden unexpected infant death/ALTE in their biologic relatives appear to be related.[29] Familial factors influencing this association may include the degree of predilection for OSA, liability for respiratory illness or allergy, dimensions of the oral-pharyngeal airway, and ventilatory response to hypoxia.

Polysomnography is required to establish the diagnosis of obstructive apnea, as history is often misleading.[28]

Breath-holding Spells. Cyanotic breath-holding episodes have been appreciated as a medical problem in pediatrics for many years. They are a common phenomena, with approximately 3% of all children manifesting these episodes sometime during their early childhood. More than 25% begin before 6 months of age.[30]

There are two main clinical features of cyanotic breath-holding episodes: a) The first is a prolonged expiratory apnea, and b) the second is the rapid development of central cyanosis, reflecting severe hypoxemia.[31]

Episodes usually begin in response to a sudden painful or unpleasant stimulus, with the infant or child becoming rapidly cyanotic (usually within 5 seconds of the onset of the event) and holding his/her breath. The characteristics of the breath-holding are that of prolonged expiratory efforts without inspiratory efforts. However, on some occasions, the cyanosis may occur despite continued breathing.[32]

Although the episodes are often precipitated by a cry or an attempt to cry, during the episode the patient usually remains silent. As the apnea and cyanosis progress, the patient may develop an opisthotonic posture, usually at around 10-20 seconds into the attack. Loss of consciousness usually occurs within 30 seconds and may be followed by a generalized seizure.[33] Recovery is heralded by a gasp, which often appears to be stimulated by a sudden shock to the patient, such as the application of cold water, blowing in the face, or an attempt at mouth-to-mouth resuscitation. On recovery from an episode, the patient may remain drowsy, pale, and may sleep for several hours. Sometimes one attack will follow another within minutes, with the succeeding episodes being more severe. From the parental point of view, these are frightening situations, and many parents believe their child to be dying during such episodes. The application of mouth-to-mouth resuscitation by parents in this situation is not uncommon. In many patients with such cyanotic/apneic episodes, crying without apnea is also accompanied by central cyanosis.

In the majority of instances, the cyanotic breath-holding episodes begin within the first year of life, with some starting within the first few days of life.[33] In one report the symptoms began at a median age of 7 weeks, and 77% began at less than 4 months.[32]

Central nervous system sympathetic activity in response to a environmental stimuli (stress) is felt to produce the prolonged expiratory apnea through effects on the brainstem respiratory centers and right-to-left intrapulmonary shunting through effects on the pulmonary vasculature. These episodes are involuntary and reflexive and occur during active or full expiration.[33]

Other indications of an autonomic dysfunction can be derived from a review of literature.[32-35] Infants with breath-holding spells (BHS) have breathing disorders during sleep. Compared to a control group, infants with BHS have obstructed breathing, snoring, and sweating during sleep.[34]

BHS may be associated with several structural neurologic problems including medullary tumor, bilateral abductor vocal cord paralysis, hydrocephalus, and Arnold Chiari malformation. An association with complex partial seizures has been described.[36]

Pallid BHS are associated with severe bradycardia or asystole, whereas cyanotic BHS are not.[35] Pallid BHS is a misnomer, for they are not breath-holding spells at all, but rather vasovagal syncope. One can often simulate these spells by applying pressure on the eyeballs, while listening to the pulse or with an EKG monitor. This will confirm the excess vagal discharge; slowing of the heart rate is documented with an occasional period of brief asystole.[35]

The evaluation of BHS consists of a careful history. Laboratory testing is usually not indicated unless accompanied by other findings on neurologic examination. Treatment consists of parenteral reassurance. The spells are usually outgrown without residua.

Gastrointestinal Causes of ALTE

A high percentage of identifiable etiologies of ALTE are the result of a gastrointestinal problem.[6] Investigation of a potential gastrointestinal cause for an ALTE begins with the history that is often the most important part of the entire GI evaluation. Specific questions pertinent to GI-related causes of the event(s) include:

- **Was the episode related to feedings?** If there seems to be a temporal association between feeding and the events, it is important to differentiate distress during swallowing from distress after the infant finishes a meal. This may differentiate structural problems, such as H-type tracheoesophageal fistula or laryngeal cleft from gastroesophageal reflux (GER) induced events.
- **If the events occur after a feeding, how long after?** Often, infants with GER have an associated delay in gastric emptying that may manifest as regurgitation of milk many hours after it was ingested.[37]
- **Does vomiting occur, or was there evidence of regurgitation during the event?** While half of all healthy infants between 2 and 8 months of age regurgitate two or more times each day, and 15% vomit three or more times daily, regurgitation leading to an ALTE is considered a "red flag" for pathological GER and requires further evaluation.[38]
- **Is there a chronic history of vomiting or spitting up behavior?** A long-standing history of vomiting in an infant with ALTE suggests a potential GI-related cause, but cannot differentiate metabolic or central nervous system disorders from GI disorders by itself.
- **Does the infant seem to experience pain when he eats or immediately after eating?** Odynophagia, or pain with swallowing, indicates a mucosal lesion such as reflux-induced esophagitis or peptic stricture, which would require further evaluation for GER-induced events.
- **Has there ever been evidence of aspiration?** GER-induced laryngospasm or bronchospasm may lead to actual aspiration events, but primary aspiration due to oral or pharyngeal motor dysfunction can also lead to lobar infiltrates on chest radiography.
- **Is there a history of hematemesis?** Again, this would suggest a disruption of mucosal integrity, such as is seen in moderate to severe reflux esophagitis.
- **Have there ever been any problems with weight gain?** Under-nutrition due to excessive vomiting or poor intake due to pain with eating can lead to growth problems, and is also considered a "red flag" for pathological GER, requiring further evaluation. However, certain central nervous system disorders (e.g., diencephalic syndrome) or metabolic diseases may manifest with poor weight gain and ALTE.
- **Is there a history of multiple formula changes, and, if so, what are the reasons?** Often, the primary care practitioner or parent will presume that symptoms such as vomiting, excessive irritability, or gassiness are caused by maldigestion of disaccharides in formula or by protein allergy. When an infant presents with an ALTE, part of the history should help to differentiate other symptoms that prompted formula changes, as these symptoms may suggest specific causes of the ALTE.
- **When symptoms such as vomiting are present, is there a distinct difference in the symptoms with a change in infant position?** Infants with GER often improve with a prone and slightly elevated posture.[39] Infants with other causes of obstructive apnea may also change their symptoms with positional changes.

After the thorough history and examination, diagnostic testing may be performed judiciously. When the event occurs during swallowing but without a history of vomiting or regurgitation, then oropharyngeal problems such as motor dysfunction of the tongue, palate, or glottis need to be considered. Similarly, more occult mechanical problems such as H-type tracheoesophageal fistulae or laryngeal cleft need to be more prominent in the differential diagnosis.

Contrast radiography using barium is generally the best study to define the anatomy of the upper digestive tract in infants with ALTE. Anatomic abnormalities of the esophagus, cardioesophageal junction, gastric outlet, and duodenal sweep are easily defined in this manner. More unusual anatomic causes of ALTE, such as gastric volvulus, can be identified with contrast radiography.[40] The test is widely available, and generally not invasive or cost-prohibitive. However, because it is a static rather than dynamic test, it is neither sensitive nor specific in diagnosing GER, which is one potential GI-related cause of ALTE. Similarly, contrast radiography may be suggestive but not diagnostic of esophageal motility disorders. These are commonly found in infants with birth defects, such as esophageal atresia post-repair, or in children with chromosomal abnormalities, such as Trisomy 21 or Cornelia de Lange Syndrome. They may also be found as primary lesions, as in achalasia. All of these motility disorders can present in infancy with ALTE, especially when regurgitation is noted, along with choking and obstructive apnea with cyanosis.

When esophageal motility disorders are suspected by history or by contrast radiography, esophageal manometry using a multi-channel water-perfused or solid state catheter is performed. This study is useful in the evaluation of esophageal peristalsis, as well as in the evaluation of the upper and lower esophageal spincter tone and relaxation. In achalasia, incomplete or absent reflex relaxation of the lower esophageal sphincter is seen, along with low amplitude or absent peristalsis in the esophageal body. The approach to management is often surgical in infancy, including many varieties of myotomy. In older children, pneumatic dilation of the LES may be useful in promoting esophageal emptying. Minimal success has been found using medications such as calcium channel blockers or prokinetics.

Gastroesophageal Reflux

GER is characterized by the effortless passage of gastric contents into the lower esophagus. Reflux can be classified as physiologic, in which the infant remains free of clinical sequelae, and as pathologic reflux or gastroesophageal reflux disease (GERD), in which gastrointestinal, pulmonary, or neuropsychiatric complications are associated with hypenation of intraesophageal acidification.[38] A classification that is particularly useful to the clinician categorizes reflux by its expected natural history. Thus, infantile reflux, which results from a delay in the acquisition of normal upper gastrointestinal motility, is likely to resolve by the first birthday. In contrast, childhood GER, although it may begin during infancy, appears to be a chronic disorder similar to reflux encountered in the adult population.

Symptoms due to reflux are summarized in Table 5.[38,41,42] Infantile apnea remains the most controversial of the possible relationships between GER and pulmonary disease. Intraesophageal acidification results in two clinically distinct patterns of apnea. Awake apnea is characterized by a sudden staring or startled appearance within the hour after a feeding and is often preceded by a change in position.[43] A history of choking, coughing, or vomiting is obtained for a minority of these infants. Spitzer and colleagues have confirmed the temporal relationship between intraesophageal acidification and awake apnea and the response of these infants to anti-reflux therapy.[43] The majority of these children had some degree of obstructive apnea during the periods of GER.

Although there is general agreement that awake apnea is reflux related, there is less consensus regarding the relationship between sleep apnea or ALTE and GER. Attempts at confirming a temporal relationship between reflux and sleep apnea have produced variable results.[38] Episodes of reflux occur more frequently in infants when awake than when asleep, although when episodes do occur during sleep they tend to be of longer duration.[44] It has been suggested that the duration and not the frequency of these episodes during sleep may be an important determinate of both reflux associated respiratory disease and ALTE.[44-46] Nocturnal reflux is uncommon, but when it occurs in infants it is more apt to occur during active sleep than during quiet sleep. In infants, active (rapid eye movement) sleep is accompanied by reduced pulmonary oxygen reserves, depressed respiratory muscle responsiveness, and reduced upper esophageal sphincter pressure; therefore, reflux during sleep may occur at a time of increased vulnerability of the respiratory system.[47] Children with prominent nocturnal cough and wheezing are especially likely to have nocturnal reflux. Additionally, nocturnal reflux predisposes to esophagitis because of the prolonged esophageal acid exposure which occurs during sleep.[47]

Another mechanism by which GER or pharyngeal incoordination cause apnea is by stimulation of chemoreceptors around the larynx that respond to a variety of stimuli such as gastric acid and water; the reflex response is comprised of central apnea, bradycardia, and pallor due to central pooling of blood.[1] In the older infant, the more mature response includes swallowing.

The infant presenting with an ALTE and symptoms suggestive of GER is best evaluated with a 24-hour intraesophageal pH study. This test has been the gold standard in identifying pathological GER for more than 20 years.

In using the 24-hour pH monitor to evaluate infants with ALTE, clinicians have focused on correlating reflux events with changes in cardiorespiratory status. Events that mimic the described ALTE and that are immediately preceded by a reflux episode on the pH recording are said to be caused by GER. However, the changes that are noted in cardiorespiratory status may not replicate the symptoms that brought the infant to medical attention, and in fact, most often the ALTE is not repeated during the monitored period. This can make it very difficult to prove a cause-and-effect relationship between GER and the ALTE.

In an effort to identify infants with respiratory symptoms from GER, Jolley et al evaluated the mean duration of reflux during sleep, or ZMD score.[48] They found that the ZMD score provided the best separation between two groups of patients; those with GER and respiratory symptoms caused by reflux, and those with reflux and respiratory symptoms due to other factors. In children with reflux-related respiratory symptoms, 94% had a ZMD score greater than four minutes. Jolley et al then went on to examine their 10-year experience with 499 infants who had undergone extended esophageal pH monitoring.[49] Of the 19 deaths found in the series, three were classified as SIDS, and two were in-hospital deaths caused by reflux-induced aspiration. All five of these infants had an elevated ZMD score when tested for GER, and the authors suggested that a cause-and-effect relationship exists between GER and sudden death in nearly 10% of infants with documented GER and elevated ZMD score. While they concluded that surgical management was the most effective and appropriate therapy in this specific pattern of GER, medical management with prokinetic agents such as cisapride or metoclopramide[50,51] as well as thickened feedings[52] and appropriate

Table 5. Symptoms of GERD

SYMPTOMS DUE TO REGURGITATION
Emesis with weight loss
Failure to thrive
Dental enamel erosion
Recurent otitis media
SYMPTOMS DUE TO ESOPHAGITIS
Chest pain, irritability, feeding problems
Anemia
Hematemesis
Esophageal obstruction due to stricture
RESPIRATORY SYMPTOMS
Pneumonia (especially recurrent or chronic)
Wheezing, bronchospasm (especially intractable asthma)
Apnea, cyanotic episodes (especially obstructive apnea)
Complex respiratory disease-reflux interactions
Esophageal atresia and tracheoesphageal fistula
Cystic fibrosis
Bronchopulmonary dysplasia
Miscellaneous: stridor, cough, hiccups, hoarseness
NEUROBEHAVIORAL SYMPTOMS
Infant “spells” (including seizure-like events)
Sandifer syndrome (atypical torticollis)

positioning[53] have generally been helpful in most infants. This allows surgery to be saved for only the most refractory cases. When medical therapy is chosen, and a cause-and-effect relationship has truly been established between GER and ALTE, a home apnea/bradycardia monitor is prescribed to alert the caretaker to further episodes when the infant is asleep or unattended.

When aspiration of foodstuffs is suspected, bronchioalveolar lavage (BAL) is a helpful tool in finding direct evidence of such an event.[54] Via the bronchoscope, saline washing and retrieval of bronchial fluid is performed. When the aspirate is stained with oil red-O stain, lipid-laden macrophages can be identified when aspiration of fat-containing formula or food has occurred. While this finding effectively proves that aspiration occurred, it does not differentiate GER-induced aspiration from primary aspiration due to swallowing dysfunction or esophageal dysmotility. Therefore, BAL is another procedure which can help support (but not prove) an exact GI-related etiology for the ALTE.

Inborn Errors of Beta-Oxidation of Fatty Acids

The rare association between inborn errors of fat oxidation and ALTE or with SIDS-like deaths has been described.[55,56] An inborn error of metabolism is more likely to be associated with an ALTE if the initial ALTE is severe and if there is a family history of ALTE, consanguinity, seizure disorders, or SIDS.[56] Inborn errors of fat oxidation may only be apparent during times of metabolic stress, such as fasting associated with an infectious illness, when the infant is forced to utilize fatty acids as substrates for energy production, rather than carbohydrates. The clinical presentation may include nonketotic-hypoglycemic attacks in a previously healthy infant, which are triggered by a physical stress or a period of fasting. Progression of symptoms may mimic an ALTE. Medium chain acyl-CoA dehydrogenase deficiency (MCADD) is the most common of these disorders, and the most likely to present as an ALTE. Inborn errors of beta-oxidation of fatty acids can cause up to 4% of severe ALTE and up to 5% of cases of SIDS.[55,56]

Each infant with an ALTE should have blood NH4 measured as a screening test. A more thorough diagnostic evaluation is recommended for ALTE infants with: 1) an elevated serum NH4; 2) a positive family history for AOI, seizure disorders, SIDS, or other sudden infant deaths; 3) laboratory evidence of hypoglycemia, hyperammonemia, metabolic acidosis, elevated liver enzymes, or abnormal hemostasis; and/or 4) a positive patient history of unexplained failure to thrive, developmental delay, or seizures. This includes blood and urine carnitine, urine non-volatile organic acids, urine acylglycine, and blood and urine acylcarnitines. Treatment of MCADD involves the avoidance of fasting, L-carnitine supplementation, a low-fat/high carbohydrate diet, and home apnea-bradycardia monitoring for infants with ALTE.

Child Abuse/Muchausen Syndrome by Proxy

Child abuse as a cause of ALTE is the most difficult to diagnose. The circumstances associated with child mistreatment may range from a sudden isolated loss of control by a parent to circumstances in which there is a long-standing catalog of premeditated and intentional acts of harm to the child. The latter form of abuse may involve injuries such as fractures of different ages, deliberate burns or scalds, pinch or human bite marks, and the induction of illness in the child.[57] One particular symptom, namely recurrent apneic or cyanotic episodes, may have occurred as a result of intentional suffocation.[57-62]

Munchausen syndrome is a bizarre illness in which a patient either fabricates or actually causes symptoms that require medical intervention. First described by Asher in 1951, the name is derived from Baron von Munchausen, an 18th century mercenary who became known for fanciful and wildly embellished tales of his travels abroad.[63] Munchausen Syndrome by Proxy (MSBP), first described in 1977 by Meadow, is a form of child abuse in which a parent, usually the mother, systematically fabricates information about their child's health or intentionally makes the child ill.[64-66] The term "Polle syndrome" has been used to refer to the same problem.[67,68] Polle was the only son of Baron von Munchausen; he died at 1 year of age, and his death may have been caused by the Baron.

When a parent induces a life-threatening illness or fabricates an illness, resulting in invasive diagnostic, anesthetic, or surgical procedures for the child, the case becomes one of child abuse.[68] The psychodynamics involved in these situations are quite different from those operative in the typical child-abusing parent.[68] The parent who is causing the illness (most often the mother) displays model behavior and, therefore, the diagnosis of induced or factitious illness is often unsuspected and hence delayed. In one review, the mean time for onset of signs and symptoms of illness to the diagnosis of MSBP was 14.9 months.[69] Given the 9% mortality associated with the syndrome, early awareness of the associated signs and symptoms by physicians is important.[66,69]

The common presentations of MSBP identified are: bleeding, seizures, central nervous system depression, apnea, diarrhea, vomiting, fever, failure to thrive, rash, infections, and allergies.[69]

MSBP is a condition in which a mother (90% of cases) pretends her infant or child is ill or causes the infant or child to be ill in order to engage in an intensely ambivalent but often destructive relationship with a physician(s).[66] MSBP therefore evolves as a product of the relationship between the mother, who has both the capacity for abuse and the potential to be gratified by the medical system, and a medical system that is specialized, investigation-oriented, fascinated by rare conditions, often ignorant of abusive behaviors, and too accepting of repeated histories.[70] Thorough discussion of MSBP must address the uncanny ability of the mother to fool doctors and the susceptibility of physicians to her manipulations. Factors that should raise suspicion of MSBP have been suggested in numerous publications and are presented in Table 6.[62,64,66,69]

Apnea has been frequently described as a manifestation of MSBP, and conclusive proof of the parent's role has been provided by the use of covert video surveillance (CVS) to film the mother during the act of asphyxiating the child.[57-62] Covert surveillance reveals that though abusing parents appear caring and kind in the presence of professionals, they can become cruel and sadistic within seconds of being alone with their child.

Techniques used by these mothers to asphyxiate their infants include covering the mouth or nose with one or both hands, a piece of cotton fabric, an article of the patient's clothing, domestic plastic film, a pillow, holding the child tightly to their chest and also inserting the fingers into the back of the mouth.[57,59,62] Infants struggle violently, but there are often no cutaneous markings. If present, bleeding from the nose or mouth in association with an ALTE distinguishes intentional suffocation from ALTE attributable to natural causes.[57] The presence of erythema over the nose

Table 6. Guidelines for Suspecting MSBP

- A child who has an unexplained, prolonged, and extraordinary illness. The medical problems do not respond to treatment.
- Physical and laboratory findings made in relation to the illness cannot be explained, are very unusual, or are considered implausible.
- A parent (usually the mother) who appears to be medically knowledgeable and/or fascinated with medical details, appears to enjoy the hospital environment, and often expresses interest in the details of other patient's medical problems. The suspected parent may work in the health care field herself or profess interest in a health-related job.
- A highly attentive parent who is reluctant to leave her child's side and who herself seems to require constant attention.
- A parent who appears to be unusually calm in the face of serious difficulties in her child's medical course while being highly supportive and encouraging of the physician, or one who is angry, devalues staff, and demands further intervention, more procedures, and the like.
- The signs and symptoms of a child's illness do not occur in the parent's absence (hospitalization and careful monitoring may be necessary to establish this causal relationship).
- A family history of similar sibling illness or unexplained sibling illness or death. The family history may also disclose numerous medical problems that are difficult to substantiate.
- A suspected parent with an emotionally distant relationship with her spouse; the spouse often fails to visit the patient and has little contact with physicians, even when the child is hospitalized with serious illness.

or face should also raise the possibility of intentional suffocation.

It has been suggested that the presence of bleeding from the nose, mouth, or both and a family history of sudden death in childhood should dictate a full and forensic analysis of the family history, including information from social services and other child protection agencies, the police, emergency departments, and the family physician.[57] There should be a low threshold for performing a skeletal survey, retinal examination, and brain imaging. Siblings should also be examined and their records reviewed.

Deliberate human actions are likely to explain a small part of SIDS and ALTE events. The American Academy of Pediatrics estimates that less than 5% of apparent SIDS deaths are due to abuse.[71] They occur, however, and must be considered particularly in light of the implications for subsequent mortality and morbidity in siblings and other infants harmed by a repeat perpetrator.

Conclusion

Infants with an ALTE form an heterogeneous entity. A large array of diagnoses can be found. In approximately 50% of the cases a specific medical or surgical cause can be found for the event. A systematic diagnostic evaluation of infants with an ALTE, together with a comprehensive treatment program is necessary to increase the possibility of quality survival for these infants.

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Physician CME Questions

29. A 2-month-old boy suddenly became limp, cyanotic, and apneic. He was revived with mouth-to-mouth resuscitation. When he was seen in the emergency room, findings on physical examination were normal. Of the following, the *most* appropriate next step in the management of this patient is to:
    A. admit to the hospital for evaluation.
    B. discharge to home with an apnea monitor.
    C. reassure the parent that this is unlikely to happen again.
    D. schedule a follow-up visit in one week.
    E. schedule polysomnography on an ambulatory basis.

30. Each of the following symptoms and finds are associated with gastroesophageal reflux, *except*:
    A. failure to thrive.
    B. iron-deficiency anemia.
    C. recurrent pneumonia.
    D. supraventricular tachycardia.
    E. atypical torticollis.

31. Each of the following is a true statement regarding gastroesophageal reflux (GER) in infants and children, *except*:
    A. apnea may be a complication without obvious emesis.
    B. nocturnal reflux may cause cough and wheezing.
    C. the "gold standard" for diagnosing pathologic GER is sampling

of tracheobronchial secretions for detection of lipid-laden macrophages.
D. reflux episodes occur in healthy pediatric patients.

32. The NIH definition of ALTE includes all the following factors, *except*:
A. Apnea
B. CPR is required to interrupt to event
C. Color change
D. Change in muscle tone

33. Which on of the following statements about home apnea monitoring is *incorrect*?
A. Monitoring can be both a source of stress and a source of support and reassurance for parents.
B. Event recordings provide objective recordings of apnea and bradycardia alarms.
C. Studies utilizing event recordings have shown that a very small percentage of events are caused by true apnea or bradycardia.
D. Scientific studies have proven the efficacy of home apnea monitoring in saving the lives of infants.

34. All of the following statements about ALTE in infants are true *except:*
A. there are multiple identifiable causes of ALTEs, after evaluation approximately 90% of these infants will have a specific diagnosis.
B. obstructive apnea may play a role in ALTEs.
C. gastroesophageal reflux (GER) may result in an ALTE in awake and asleep infants.
D. the infant with an ALTE and symptoms suggestive of GER is best evaluated with a 24-hour intraesophageal pH study.

35. Each of the following is a known cause of ALTE in infants *except*:
A. seizure disorder.
B. GER.
C. otitis media.
D. inborn error of fat oxidation.

36. Which one of the following statements about Munchausen Syndrome by Proxy (MSBP) is *incorrect*?
A. MSBP is a form of child abuse in which a care-taker fabricates information about their child's health or intentionally makes the child ill.
B. The person causing the illness in the child is most often the father.
C. Common presentations of MSBP are bleeding, seizures, apnea, and vomiting.
D. One identifiable cause of ALTE is intentional suffocation of the infant.

In Future Issues: Abdominal Trauma

Annual Statement of Ownership, Management, and Circulation