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In this edition

Child and Adolescent Psychology Service 1
Dental Considerations for Children with Trisomy 21 2
Pumping Insulin 2001 4
Escherichia coli Enteric Infections 6
The Pediatric Patient Presenting With Chest Pain
Should Not Cause You To Have Chest Pain .. 9

PEDIATRIC NEWS

The Newsletter of the
San Antonio
Military Pediatric Center
Vol 8, No.3
June 2001

CHIEF'S CORNER

Child and Adolescent Psychology Service

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BAMC is fortunate enough to have a large, well-staffed child and adolescent psychology service. Army wide, there are few, if any as extensive or as diverse in their role within the hospital. At present CAPS consists of six licensed psychologists, a part-time child psychiatrist, and two enlisted behavioral medicine specialists delivering a wide variety of services to include individual, group, and family therapy for the gamut of childhood psychological disorders. Our providers are placed strategically in the departments of behavioral medicine, pediatrics, and part-time in family medicine. CAPS staff also conduct numerous psychological evaluations and work closely with other BAMC departments such as developmental pediatrics and pediatric endocrinology to provide multidisciplinary assessment and intervention. CAPS outreach efforts include providing services through the Ft. Sam Houston school system. Numerous groups are offered to children, adolescents and parents. CAPS also maintains an active training role. The pediatric psychology fellowship trains two postdoctoral fellows who round out the CAPS professional staff. All CAPS personnel are active in fellowship training and have been involved in the instruction of medical residents in addition to the four psychology residents of the department of behavioral medicine. The bulk of our referrals are made through the primary care clinics of the hospital. Our staff is available to consult and welcomes opportunities for joint endeavors in both clinical intervention and research.

Routine consultations can be obtained through the electronic consult options of CHCS. CAPS also provides ER walk in coverage on weekdays between 0730 and 1600 so that children and adolescents at risk for harm to self or others or who require other emergency intervention can be evaluated. For non-routine consultation please speak to a CAPS staff member directly. Questions about current group offerings may be addressed to me or to any CAPS staff member.

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Dental Considerations for Children with Trisomy 21

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Trisomy 21 is a congenital, autosomal anomaly characterized by generalized physical and mental deficiencies. It affects 1 in 600-1000 live births. In 1866, John L. Down, for whom the syndrome is named, published a paper describing characteristics of the syndrome. The following is a review of dental considerations for children with Trisomy 21.

Dental soft tissue features

Down Syndrome patients have deficient development of the midface. The length, height, and depth, but rarely the width of the palate is affected. This results in a high, V-shaped palate. Characteristic facial features include a descending angle of the mouth, elevation of the upper lip, and an everted lower lip with tongue protrusion. These features manifest due to hypotonia of the perioral muscles. The oral cavity is usually small with a large tongue, which often leads to mouth breathing with resultant xerostomia and potentially chronic periodontitis. The lips may become fissured and dry leading to splitting, bleeding and inflammation. A hypotonic tongue shows characteristic imprints of teeth along the lateral border. The tongue is more furrowed creating areas for plaque and food to accumulate. A degree of airway obstruction and sleep apnea are common due to the relatively large tongue, crowding of the midfacial structures, a high arched narrow palate, micrognathia, short broad neck and a high incidence of tonsillar and adenoidal hypertrophy.

Skeletal features

Down Syndrome patients have a deficient midface resulting in a class III profile where the mandible is ahead of the maxilla. Retardation in the growth of the maxilla and mandible is evident. The mandible does not rotate down and back as in a normal growth pattern. This can result in a dental underbite (Class III malocclusion), or anterior crossbite and/or posterior crossbite. Mild microcephaly is present with up-slanting palpebral fissures.

Aberrations in dental development

1) Missing teeth:

Agenesis of permanent teeth is 10 times greater in patients with Down Syndrome than in the general population with a higher frequency in males than in females. Studies have shown the incidence to be as high as 63 percent of subjects. The teeth most affected by agenesis are mandibular central incisors, followed by maxillary lateral incisors, maxillary second premolars, and then mandibular second premolars. Canines and first molars are rarely affected. The main components in the pattern of agenesis may be related to the peripheral nervous system and abnormal cartilagenous tissue. Nerve, bone, and their mesodermal developmental precursor, such as cartilagenous tissue, are important factors in tooth formation.

2) Tooth size and shape:

Between 35-55% of Down Syndrome children present with microdontia in both the primary and permanent dentition. Clinical crowns are frequently conical, shorter, and smaller than normal and the roots are shorter as well. Lateral incisors may appear peg shaped. Taurodont teeth, which are molars with abnormally long (apical-occlusal height) pulpal chambers, have also been commonly reported.

3) Delayed eruption:

The first primary teeth may not appear until 2 years of age and the dentition may not be complete until 5 years of age. The eruption sometimes follows an abnormal sequence, and some of the primary teeth may be retained until 15 years of age.

Dental caries

Most investigators report a low prevalence of caries in Down Syndrome patients relative to other developmentally delayed or normal children in both the primary and permanent dentition. *Streptococcus mutans* is the primary causative agent of dental caries in man, but the relationship between *S. mutans* and dental caries in children with Down Syndrome has not been well characterized. Living environment, dietary and hygiene habits, fewer erupted teeth, delayed eruption and different proportions of salivary components compared to normal children have been proposed as the causes for the low prevalence rate of dental caries.

Periodontal disease

There is a high prevalence and rapid progression of chronic inflammatory periodontal disease (bone loss) and gingival inflammation in children with Down Syndrome. Reports have shown that up to 90% of Trisomy 21 children exhibit some periodontitis. Other studies have shown the prevalence of periodontal disease to be 60-100% in those under 30 years of age. In contrast, periodontitis is relatively rare in normal young

individuals. The amount of plaque and calculus present does not correlate with the severity of the periodontal condition, therefore, poor oral hygiene alone is not a determinant. The early, rapid gingival inflammation may be due to a more virulent plaque composition and/or to an impaired host response. The host response is associated with reduced neutrophil and monocyte chemotaxis, impaired neutrophil phagocytosis, reduced T-lymphocyte counts and immature T lymphocytes. Research has shown that periodontal herpesvirus-bacteria coinfections may play important roles in the pathogenesis. Herpesviruses may reduce the periodontal defense and promote growth of subgingival bacteria capable of causing periodontal breakdown. Studies have shown that colonization of certain pathogenic organisms for periodontal disease has occurred before 5 years of age and additional colonization increased with age. Tooth morphology, bruxism (grinding of teeth), and malaligned teeth have also been implicated as factors in periodontal destruction.

Patient Management

Most Down Syndrome children have a wonderful disposition and are pleasant, cheerful, and affectionate. They can often be managed in the dental office in a conventional manner with tell-show-do behavior management techniques. Light sedation and immobilization may be indicated in those children who are moderately apprehensive. Special concern and caution must be taken during immobilization due to the presence of possible atlantoaxial instability. If sedation or a technique of anesthesia involving spontaneous ventilation is used, the patient should be carefully monitored as hypoventilation frequently occurs. General anesthesia is appropriate for those children who are unable to cooperate or who need extensive dental treatment. Anesthesia and surgery carry a higher risk than in normal patients and skillful perioperative management is essential for optimal outcome.

Antibiotic prophylaxis before invasive dental procedures is often indicated for medical conditions that predispose patients to bacteremia and subsequent infections. Down Syndrome patients who have cardiac anomalies, or compromised immunity may need antibiotic prophylaxis prior to dental treatment.

Parents often do not realize the need for early intervention in seeking dental treatment for their child with special needs. This may be due to the overwhelming medical, social, and emotional issues they are coping with. The American Academy of Pediatric Dentistry recommends that children should receive their first dental exam within six months of the eruption of the first primary tooth and no later than twelve months of age. This is especially important for children with special needs such as children with Down Syndrome. Anticipatory guidance regarding dental and oral development, fluoride status, oral hygiene, and effects of diet on the dentition are extremely critical. Appropriate and early oral hygiene and care are essential for these children.

The role of the pediatrician is crucial in making appropriate referrals to a pediatric dentist and/or general dentists trained in treating special needs patients. It is also important to establish a relationship with a dental healthcare provider early on so that a child can become more comfortable in the dental setting at an early age. Patients should be seen for regular check-ups every 6 months. If indicated, patients can then be referred to orthodontists and/or periodontists for additional specialized dental care.

Dental management for patients with Down Syndrome can become more complex, and therefore, early referral and regular dental care is crucial for optimal care.

Written in Loving Memory of: Noah Lee Smith 11-12-99 to 3-23-00

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Pumping Insulin 2001

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Introduction

There have been many recent advances in the treatment of Type 1 insulin dependent diabetes, but none so exciting as the recent explosion in the number of children and adolescents being treated with Continuous Subcutaneous Insulin Infusion (CSII) – the insulin pump. The Pediatric Endocrine Service at Brooke Army Medical Center and Wilford Hall Medical Center has been fortunate to be able to offer this form of diabetic management to our diabetic population. As we have been able to extend our services widely in the Tricare Regions 6 and 7, more and more patients are being offered and accepting insulin pumps as part of their daily lives. In addition, patients are arriving from other Tricare military and civilian facilities already on insulin pumps. Therefore, it is important that all of us be familiar with insulin pumps and the risks and benefits that this treatment entails.

Advantages of Insulin Pump Therapy

As insulin pumps become more sophisticated in microprocessor power, infusion motor reliability and durability, insulin pump therapy becomes feasible for the majority of children and adolescents with diabetes. The Diabetes Control and Complication Trial (DCCT) investigated the power of improving diabetes control, as demonstrated by lowering Hemoglobin A1C values, in greatly lessening the occurrence and progression of diabetic complications. Maintaining normal, or near normal, blood glucose levels for the greatest length of time, positively influences outcome. By closely adhering to recommended insulin pump regimens, the patient is provided with the most powerful tool to date for achieving this end.

By matching insulin needs as closely as possible, insulin pumps allow the patient to maintain a normal schedule of eating and lifestyle without sacrificing diabetes control. Patients are no longer tied to eating meals and snacks according to a rigid schedule, in order to avoid hypoglycemic reaction. Patients are able to match insulin needs with three types of pump settings. The first, a "basal rate" is a programmed infusion of fast acting insulin, usually Humalog, that matches insulin needs in the fasting state. Depending on the sophistication of the specific pump, the basal rate can be preprogrammed to deliver a varying basal rate every half-hour over the course of 24 hours. Some pumps allow for 2 different basal rate profiles, which can be used for school days versus sleeping in on weekends, or physical education or sport days vs. couch potato days. Additionally, a "temporary basal rate" can be programmed as needed for sick days or for a dramatic change in activity level, such as an unscheduled sports game.

The second setting is a "carbohydrate meal bolus". The patient has determined a ratio of insulin needs to cover amount of carbohydrate eaten. This ratio is used to program the insulin pump at mealtime for a bolus of fast acting insulin to cover the amount of carbohydrate in the meal. This ratio is calculated from experience by dividing 500 grams by the total daily insulin (TDI) given by injection prior to starting the pump. Each patient should be familiar with their "carb ratios" and can provide this information to new health care providers. Not infrequently, the "carb ratio" may vary among breakfast, lunch and dinner. Most "carb ratios" range from 1 unit insulin to every 8 grams of carbohydrate for an older adolescent, down to 1 unit for every 30 grams of carbohydrate for a newly diagnosed young child. Each patient's "carb ratio" will change with factors such as total duration of diabetes, age and pubertal stage.

The third setting is a "correction bolus" (also referred to as "correction factor", "insulin sensitivity factor", or "glycemic index"). This insulin bolus is designed to provide the necessary insulin to bring a patient's blood glucose back to a desired level. Again, pump patients should be able provide this information to health care providers. The number is derived from dividing 1500 by the TDI usage. For example, a patient using 50 units of insulin each day would lower their blood glucose by 30 mg/dl with each unit of injected insulin, as 1500 divided by 50 equals 30. The adequacy of this "correction bolus" can be determined by a review of the patient's pump logbook, looking to see if high blood glucoses are corrected to a target range by 2 hours after the correction.

Insulin pump users generally find that their total daily insulin amount is less than it was while taking injections. The TDI usually decreases by 35-33% once on a pump. While it is unclear exactly why this occurs, it is felt that by more closely matching insulin needs and keeping blood glucose's in greater control, there is less insulin resistance induced by high blood glucose's. There are indications in the literature that insulin, itself, may be toxic to the smallest blood

vessels and, thus, may contribute to the end organ damage.

Having the ability to rapidly "correct" blood glucoses and match carbohydrates with "carbohydrate meal boluses" gives the patient a previously unattainable level of control. This control empowers patients to increase their level of compliance with all aspects of their diabetes management. Patients see a clearly defined reason to check and record blood glucose's, as now they can act immediately on these numbers. The feelings of helplessness that used to occur when a patient "did everything right" and still had poorly controlled blood glucose's are gone. Patients truly now have control.

Finally, lifestyle issues have to be considered. An insulin pump allows children and adolescents to live the same lifestyle as their family and friends. While a pump is not a license to eat whatever you want whenever you and all that you want, as some children feel, a pump does allow for normal eating patterns. Diabetics can eat three meals without snacks. The fear of the dreaded missed snack hypoglycemia is largely gone. Adolescents can sleep in without fear of extreme hyperglycemia. Most importantly, patients do not have to leave an enjoyable activity just because it is mealtime.

Disadvantages of Insulin Pump Therapy

As with everything in life, there is a con for every pro. Insulin pump therapy has several drawbacks that have to be considered for each patient. The absolute best of therapies hold little value for a noncompliant patient. The adolescent patient who clearly wants nothing to do with an insulin pump, who is forced onto insulin pump therapy by a concerned zealous parent, will find a variety of ways to make insulin pump therapy unsuccessful. Part of our job is to assess each patient's motivation to become an insulin pump user. It is just as easy to be noncompliant with a pump as it is with injections. A great deal of time, expense and effort can be saved by properly assessing motivation.

Technology, for those who embrace it, is a wonderful thing. For those who fear it, technology can be a curse. Many patients have concerns that being hooked up to a pump can be dangerous. Their fears usually are concerns that the pump will malfunction, either delivering too little or too much insulin. In fact, as catheter site infusion occlusions are the most common pump failure, the risk of mild diabetic ketoacidosis rapidly occurring is real. However, for the vigilant patient, this risk can be minimized. Patients are instructed to retest within one hour of attempting to correct a high blood glucose. A second high reading should prompt a catheter site change and a repeat correction bolus. Testing for ketones in the urine is also recommended under these circumstances. In our practice, we have seen a dramatic correction of mild DKA in both the home and hospital setting in our pump patients. While DKA may come about more quickly, correcting DKA is much easier as less metabolic derangement has had time to occur.

Pump therapy is costlier in the short run. Pumps cost about $5000.00. Insurance covers all or part. Pump supplies cost $120 to $150 per month, also with varied coverage. Additionally, more frequent blood glucose testing and insulin wastage (from reservoir changes and pump priming) will increase cost. The immediate benefit is better short-term health and a more normal lifestyle. The biggest benefit may come from decreased costs by preserving vital organ function, such as eyes and kidneys. Families are advised to investigate insurance coverage and consider costs before initiating pump treatment.

Concerns of hypoglycemia and weight gain can largely be allayed. With the use of fast acting insulin and more accurate blood glucose monitors, hypoglycemia has become less frequent on the pump compared with injections. Long acting insulins, such as NPH and Lente, may have up to a 25% variation in daily absorption, leading to unexpected, but not unexplained, episodes of hypoglycemia. In fact, recurrent nocturnal hypoglycemia is one of the current pump indications. Weight gain also was much more of a problem when Regular insulin was being used in pumps, due to the slower absorption and, therefore, need for snacks. Weight gain associated with any improvement in diabetes control will continue to be a problem. But, proper nutritional counseling as well as anticipatory guidance can greatly help to alleviate this problem. In fact, dieting for weight loss is safer and easier on a pump.

Localized skin infections with Staphylococcus are a real concern. Care must be taken to observe handwashing before insertions and to observe the site for redness or tenderness. At the first sign of infection, the cannula site must be changed. Early use of oral antibiotics is prudent. Patients are requested to seek early medical consultation if there is a concern of infection.

Rules of the Road

Once a patient has decided on insulin pump therapy, there are several issues that must be addressed to ensure success. Already, motivation and compliance issues have been discussed. Patients also

need to decide up front whether the more demanding blood glucose-testing regimen is something they can comply with. There are patients who want a "smart pump"; one that monitors blood glucose's automatically and delivers precise insulin boluses. This "set it and forget it" pump does not describe the currently available devices. All the current pumps require active participation of the patient, from blood glucose testing, to selecting the settings on the pump and counting the carbohydrate content of foods eaten.

Carbohydrate counting is the backbone of insulin pump therapy. It requires close contact for education with a dietitian and motivation on the patient's part. It takes about two months of hard work to get good at carbohydrate counting. As a "carbohydrate meal bolus" is programmed into the pump by the patient for every food ingested, successful pump use requires proficiency in carbohydrate counting.

Compliance is a real issue. It is easy to be noncompliant while using a pump. In fact, large group experiences have identified a subgroup, usually adolescents, who actually have higher Hemoglobin A1C's while on insulin pumps. Poorly counted carbohydrates, lack of blood glucose testing and missed meal boluses account for most of the problems. Sounds familiar. These are the same problems with compliance faced by patients on traditional injections.

Importantly, access to a physician and the pump trouble-shooting help-lines are critical. Patients are encouraged to have daily physician contact on initiation pump therapy. As control increases and the patient's familiarity and skill with pump management becomes sufficient, contact can drop back to traditional intervals. Many patients describe initiating pump treatment as stressful as when they initially developed diabetes. The main difference is that they also feel they are gaining a degree of control that gives them their life back again.

On the Horizon

We are seeing rapid improvements in the treatment of diabetes. The Holy Grail of "the cure" is still out there, but we are coming close. There are human use trials of implantable "smart pumps" currently underway. We are telling our patients that we believe that there are a variety of treatments in the near future that will normalize a diabetic's life. But, why wait? The current state of the art in subcutaneous insulin pump therapy is exceedingly good. It is already possible to have nearly normal blood glucose's all of the time. Embarking on insulin pump therapy does not preclude the chance of partaking in a better treatment in the future.

Our Role

Limitations being as they are, there are patients who choose either to seek their care on the civilian sector or elect not to take advantage of insulin pump therapy. We are concerned with letting our patients know that we can support insulin pump therapy. We are available on a consultative basis to discuss insulin pump treatment, and strive to meet our patients' needs.

Your Role

Please advise your patients that this form of treatment is available through the military. For those of you who may meet a pump patient in the emergency setting, please feel free to contact us on our service pager at (210) 513-7759 for assistance.

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Escherichia coli, the predominant facultative anaerobe of the human intestinal tract, plays an important role in normal physiology. However, certain strains have evolved that can cause an impressive array of disease states, including diarrhea, dysentery, urinary tract infections, septicemia, pneumonia, and meningitis. Each of these diseases is typically caused by a unique set of *E. coli* strains. These strains have developed specific virulence factors that allow them to, first, colonize a particular environment and, second, cause disease.

Enteric *E. coli* infections are an excellent example of this. There are four main categories of diarrheagenic *E. coli.* These are (1) Enteropathogenic *E. coli* (EPEC), (2) Enterotoxigenic *E. coli* (ETEC), (3) Enteroinvasive *E. coli* (EIEC), and (4) Enterohemorrhagic *E. coli* (EHEC). Two other categories, Enteroaggregative *E. coli* (EaggEC) and Diffusely adherent *E. coli (DAEC)*, have been characterized but their clinical importance is still uncertain.[1] Although the four main categories of diarrheagenic *E. coli* are separate and distinct, they have similarities with respect to pathogenesis.

(1) Critical virulence properties are encoded on plasmids.

(2) There is a characteristic interaction with the intestinal mucosa.

(3) Enterotoxins or cytotoxins are produced.

(4) Strains within each category tend to fall in certain O:H serotypes.

EPEC

EPEC was the first type of *E. coli* to be shown to cause diarrhea in humans. In the 1940's, summer diarrhea was a significant problem in Europe and North America. John Bray, a microbiologist, and John Beavan, a pediatrician, decided to study the problem. They knew that routine cultures failed to show a pathogen in most infants and hypothesized that an *E. coli* was responsible. They took an *E. coli* isolate from an infant with diarrhea and used it to immunize their pet rabbit. Serum from this rabbit was then used as an antiserum to test stools from infants with diarrhea versus controls for agglutination. The serum agglutinated the *E. coli* in 92% of diarrhea infants but only 6% of healthy controls.[2] This was found, years later, to be an O111 serogroup—a classic EPEC serogroup.

EPEC is a major cause of diarrhea in the developing world. It predominantly affects children less than 2 years old, especially the 0 – 6 month age range. In this group it may be the most common identifiable bacterial pathogen and in some studies has exceeded even Rotavirus in incidence.[3] It is not thought to be a significant problem in developed countries. However, there have been 2 severe outbreaks associated with daycare centers and routine diagnostic tests will not detect EPEC.[4] One recent study in Seattle screened all stool samples submitted for routine culture for the presence of EPEC. A variety of assays including DNA probes, adherence assays, and serotyping were used. Strains with properties consistent with EPEC (discussed below) were found more often than Salmonella, Shigella, Campylobacter, E. coli O157:H7, or Yersinia.[12] There was not a control group in this study but it suggests that EPEC may be a more common cause of diarrhea in this country than previously thought.

EPEC bind to the intestinal mucosa in a 3-step process. First, they attach loosely to the mucosa via a plasmid-encoded surface protein, or pilus. Second, genes are activated that cause dissolution of the microvilli around the EPEC organism. Third, the chromosomally located *eae* gene encodes a protein called intimin that causes intimate attachment. Additional bacterial gene products mediate further disruption of the cytoskeleton. This whole process results in the characteristic "attaching and effacing" lesion (Fig.1).

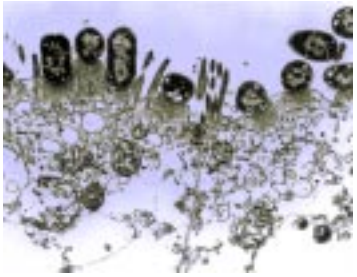

Figure 1

The mechanism of diarrhea-production is not completely clear but malabsorption secondary to loss of microvilli is most likely a major component. Some volunteer studies, however, have shown symptom onset in just a few hours. This suggests at least a secretory component. This is supported by the recent discovery of the *EspC* pathogenicity island on the chromosome of some, but not all, EPEC. The protein encoded by this gene, EspC, is excreted and has enterotoxin activity.[13] A small number of EPEC strains have been shown to produce Shiga-like toxin but the vast majority does not.[5]

The clinical illness is variable but can be quite severe. In the 1987 U.S. outbreak[4] there was an average of 8 – 12 stools/day and a mean duration of illness of 18 days. In contrast, in a Mexican community study the mean duration was only 8 days.[6] Vomiting and fever are common.

Treatment is supportive. Early feeding, especially breast feeding, should be instituted and oral rehydration used aggressively. Antibiotics may shorten the course of the disease but multiple resistance is the rule.

ETEC

Enterotoxigenic *E. coli* (ETEC) is another major cause of diarrhea worldwide. In the developing world children may have 3 or more episodes a year until age 3 – 5 years. Diarrhea in this age group is second only to acute respiratory illness as a cause of death. The WHO estimated that in 1990 approximately 800,000 children died from diarrheal disease caused by ETEC.[7]

Approximately one-third of travelers to tropical and sub-tropical areas of the world will develop diarrhea.[8] ETEC is the most frequent cause of travelers' diarrhea and has been isolated in over half of cases. ETEC induced diarrhea is also an important illness for military personnel from industrialized countries when they are deployed to less developed countries. Diarrhea was a well-documented problem for U. S. forces in Egypt[9] and U.S. forces in Saudi Arabia during Operation

Desert Shield[10]. Surveys from 1% of the 200,000 U.S. military personnel in Saudi Arabia indicated that 57% experienced diarrhea, 22% sought medical attention, and 19% were unable to perform their duties while affected. A bacterial etiology was found in 50% of cases that were investigated and ETEC represented 50% of these.[11]

ETEC cause disease by first attaching to the mucosal cell and then releasing heat-labile enterotoxin (LT), heat-stable enterotoxin (ST), or both. ETEC express antigenically distinct proteins on their surface called colonization factor antigens (CFAs) or coli surface (CS) antigens. These proteins bind to specific receptors on the mucosal cell and allow close proximity for delivery of the enterotoxins. LT and ST cause increased production of cAMP and cGMP respectively. Both result in net secretion of chloride and fluid. LT is structurally and functionally similar to cholera toxin.

Clinical disease is characterized by a short incubation period (14-50 hours) and an abrupt onset. The diarrhea is watery and without blood or mucus. Vomiting and fever are unusual. ETEC diarrhea is usually mild and self-limited but can be severe with purging rates close to that of cholera.[14]

Treatment, as with EPEC, is generally supportive. Maintenance of hydration with oral rehydration solutions can be life saving in children. Antibiotics do shorten the course of the illness but increasing antibiotic resistance is becoming a problem. Fluoroquinolones are currently the most commonly recommended agents. Bismuth and loperamide are also effective in decreasing diarrhea severity but loperamide should not be used if there is fever or the diarrhea is bloody.

Doxycycline and Septra have been shown to be effective in prophylaxis for travelers but increasing resistance would suggest the fluoroquinolones as a better choice. However, the CDC does not recommend prophylaxis. Risks include adverse drug reactions and a false sense of security. ETEC is the most frequent but not the ONLY cause of diarrhea in the traveler. The best prevention is still prudent dietary practices.

This is the end of Part 1 on E. coli enteric infections. Part 2 will finish with EIEC, EHEC (yes, that's O157:H7), EaggEC, and some historical aspects of diarrhea and the military.

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Title graphic: courtesy of Dr. Brett Finlay

Figure 1 from reference 14.

The O and the H of It

Serotyping of E. coli has played an important role in the investigation of pathogenic strains. Before more modern molecular techniques helped define specific virulence characteristics it was the main tool in differentiating pathogenic strains.

E. coli are serotyped on the basis of their O (LPS), H (flagellar), and K (capsular) surface antigen profiles. The O antigen defines the serogroup and the H antigen identifies its serotype. In a strain with a capsule, the K antigen is also used for classification. There are presently 171 O and 56 H antigens recognized.

There is a correlation between serogroup and virulence, though it is not the O, H, or K antigens themselves that cause disease. They are simply markers that correlate with certain virulence mechanisms. For example, strains classed as O111 are typically EPEC (occasionally EHEC) and K1 strains are a cause of systemic disease in infants.

The Pediatric Patient Presenting With Chest Pain Should Not Cause You To Have Chest Pain

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San Antonio Military
Pediatric Center

Chest pain is a common complaint in older children and adolescents. The differential diagnosis is broad and is usually benign. It very rarely represents a cardiac etiology in this age group. However, there are a few serious, life-threatening diagnoses that must be ruled out. It is the primary care physician's role to identify the majority of patients who only need reassurance versus the few that may need further work-up and referral to a sub-specialist.

There is usually a high level of anxiety on the child and parents' part related to the complaint of chest pain. The complaint must be taken seriously and conclusions should not be reached until a thorough history and physical has been completed. As with many complaints, the history and physical are the primary diagnostic tools necessary to make an accurate disposition. Rarely are further diagnostic tests necessary or helpful.

History

(*Red flags concerning for a cardiac etiology are in bold.)

Significant past medical history should be elicited to include specifically cardiac, pulmonary and GI diagnoses. A family history should be obtained with specific questions regarding congenital heart disease, cardiomyopathy, early coronary artery disease, hypercholesterolemia and sudden death.

Open-ended, descriptive questions regarding the chest pain should be addressed first to the child and then to the parent, if necessary. "Yes/no" questions are more likely to "lead the patient" and elicit a positive response. Ask the child to describe the quality of the chest pain (i.e.: Is it a sharp; pounding; aching; burning; or **heavy/ pressure sensation**?) If the child is having difficulty describing the pain, then ask, "What would you have to do to me to give me the same feeling?" When does the pain occur? (**Primarily with exertion** is concerning and exclusively at rest is reassuring.) How long does it last? (Seconds; **minutes**; hours or days.) Point with one finger where it hurts. (**Left**, right or both sides of the chest; **substernal** or epigastric.) Does the pain radiate anywhere? (**Left shoulder/arm** is concerning for a cardiac etiology.) How often does it occur? How long has it been occurring? **Acute** or chronic. On a scale of 1 to 10, how intense is it? Does it cause you to stop what you are doing? What makes it worse and what makes it feel better? (Worsening of pain with inspiration or movement is unlikely to be cardiac in origin and more likely to be pleuritic or musculoskeletal. Pain that is reproduced or relieved with pressing/massaging the area of discomfort is most likely musculoskeletal. **Relief obtained immediately after stopping exertion and resting** is concerning.) Is there a certain position that makes it feel better? (**Sitting up leaning forward is concerning for pericarditis**.)

Associated symptoms should be elicited. Ask, "Does anything else feel different at the time you are having the chest pain?" Then ask specifically about palpitations; lightheadedness; syncope; dyspnea; pallor and diaphoresis. If there are any of these symptoms you must established the timing of the symptoms in relationship to the chest pain. Some children will have chest pain which then makes them anxious leading to increased heart rate, shallow rapid breathing, possible lightheadedness and perioral/extremity paraesthesias consistent with hyperventilation. This must be distinguished from the child who describes **sudden onset of rapid heart rate, followed by chest discomfort, lightheadedness and/or dyspnea** which is concerning for a true tachyarrhythmia.

A history of trauma to the chest; recently starting a new physical activity or a sudden increase in physical activities would support a musculoskeletal etiology. A history of chest pain associated with meals or an acid taste in the mouth would suggest a GI etiology. A pulmonary etiology is suggested by cough, dyspnea, and/or wheezing. Fever, weight loss or fatigue

may suggest an infectious or systemic etiology. Lastly, a history of any recent stressors at home or school should be elicited. It is also helpful to ask the child/parents what they think is the source of the pain and what is their biggest concern.

Physical Exam

The physical exam is guided by the information obtained from the above detailed history. It must include at a minimum the cardiovascular, pulmonary and GI systems.

The presence of a murmur that does not meet the criteria of a benign murmur would suggest congenital heart disease (especially left ventricular outflow tract obstruction) or acquired heart disease (Rheumatic, Kawasaki's, myocarditis; early atherosclerotic disease or cardiomyopathy.) A **gallop** would suggest myocarditis or cardiomyopathy. Pericarditis should be suspected in the presence of a **pericardial rub**.

Abnormal lung sounds and/or increased work of breathing would suggest a pulmonary etiology. Epigastric or abdominal pain with palpation could be present with a gastrointestinal etiology. Reproducible pain with palpation of the chest wall is consistent with a musculoskeletal etiology. A chest deformity may indicate trauma or a musculoskeletal etiology.

Work-up and Management

The majority of patients will have a history and physical exam consistent with a benign etiology and therefore no further work-up is warranted and may actually be detrimental to the reassurance process. Explain in detail to the patient/family all of the reassuring historical and physical exam findings. There will be a group of children whose exam is completely normal and the history is benign but does not fit neatly into one of the above categories. This is considered idiopathic and should also be addressed with reassurance.

A history and/or physical exam concerning for a cardiac etiology requires a 12 lead EKG (possibly a CXR) and a consult to a Pediatric cardiologist. The urgency of this consult depends on your findings, whether it is acute or chronic and how severe the symptoms are. Questions regarding appropriate timing can be addressed with the cardiologist on call. Symptoms that occur exclusively with exertion require further work-up. The patient should be advised to avoid physical activities until the work-up is complete.

A CXR is warranted for pulmonary signs and symptoms. PFT's may be obtained if the history suggests exercise-induced asthma.

Musculoskeletal etiologies can be treated with a trial of NSAIDs (advise to take with food.). Acid/reflux symptoms can be treated with a trial of antiacids/H2 blockers.

Conclusion

Chest pain is a common but usually benign complaint in pediatric patients. This complaint cannot be adequately addressed in a quick office visit. "Red flags" must be sought to identify the few patients with significant etiologies. A detailed history and physical are the keys to making an accurate diagnosis and disposition. Most patients/families require reassurance only.

References

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Gessner I., Victorica, B., Pediatric Cardiology. A Problem Oriented Approach. WB Saunders Company. 1993. 147-151.

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Differential Diagnosis of Chest Pain

1. Idiopathic.
2. Musculoskeletal
   - **Costochondritis**
   - Muscle Strain
   - Skeletal trauma
   - Rib cage anomalies.
   - Breast abnormality
3. Pulmonary
   - **Exercise induced asthma**
   - Pleuritis
   - Pneumonia/pneumonitis
4. Cardiac
   - Left sided obstructive lesions (Aortic stenosis/ hypetrophic cardiomyopathy)
   - Pericarditis/Myocarditis
   - Aberrant coronary artery
   - Homozygous hypercholesterolemia
   - Kawasaki's Disease with coronary aneurysms
   - Tachyarrhythmias
   - Early coronary atherosclerosis
5. Systemic
   - **Tumors of the chest/mediastinum**

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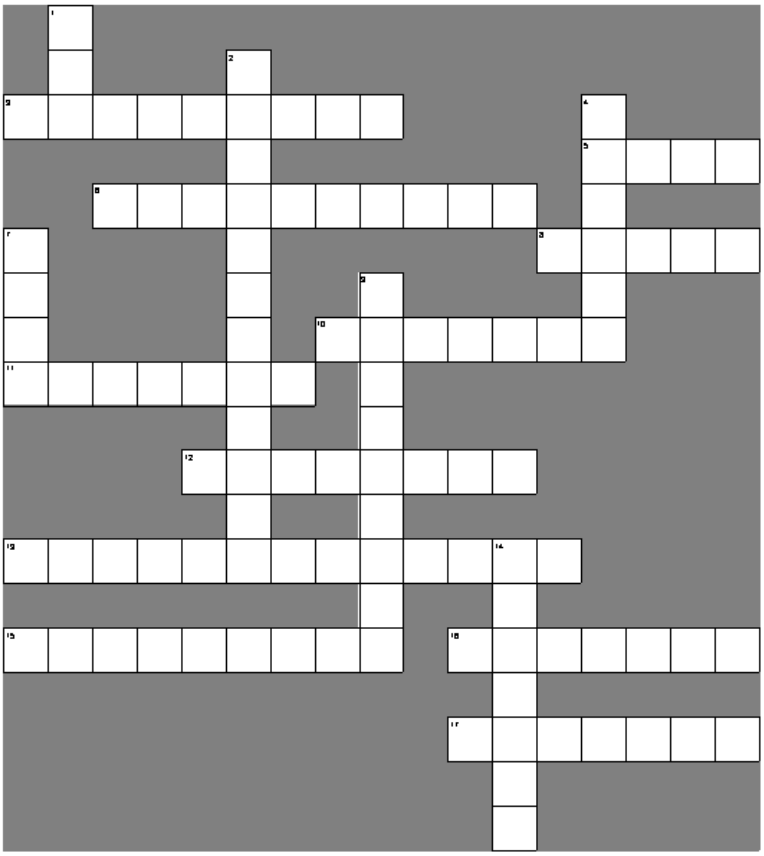
June 2001 Review Crossword



Across

3. Ratio of insulin needs to cover amount of carbohydrate eaten
5. A major cause of diarrhea in the developing world perhaps exceeding even Rotavirus in incidence
6. A third type of insulin pump setting which is a bolus of insulin designed to provide the necessary insulin to bring a patient's blood glucose back to
8. A plasma encoded surface protein in the intestinal mucosa which allows EPEC to loosely attach to the mucosa
10. Protein involved in the third step of the process of binding EPEC to the intestinal mucosa
11. Primary causative agent of dental caries in man
12. EPEC was the first type of E.coli shown to cause _____in humans
13. A cause for failure of insulin pump therapy which should be identified prior to its initiation
15. Factors that allow E.coli strains to colonize a particular environment and then cause disease
16. Grinding of teeth
17. The most important diagnostic tools for chest pain are the _____and physical

Down

1. With the insulin pump, this may occur more quickly but also may be corrected more rapidly
2. Abnormally long pulpal chambers
4. Most pediatric complaints of chest pain are _____
7. Total daily insulin (TDI) is more or less when using the pump compared to injections
9. Class III malocclusion
14. Exertional chest pain is worrisome for a _____etiology