PROBLEMS in PEDIATRIC EMERGENCY MEDICINE

Edited by

Robert C. Luten

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SERIES EDITOR

Ann Harwood-Nuss

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To my son Rocky,
to whom I am forever committed.

To Mary,
who has been my best friend.

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FOREWORD

The new series, *Contemporary Issues in Emergency Medicine,* has been created to fill a number of needs in the medical literature. Our goal is to produce bi-annual, in-depth volumes addressing major clinical topics in emergency medicine.

I have selected volume editors based on a personal bias that favors those individuals intimately familar with the complexities and scope of clinical emergency medicine. Volume topics have been selected based on a number of factors, but the dominant feature is that each represents a clinical issue which is recognized as major and problematic for the practicing emergency physician. The text will be a blend of didactic and practical information organized in a format designed to enhance clinical application. The chapter format will be consistent, featuring discussions on differential diagnosis, clinical manifestations, emergency department evaluation and management, the use of routine and special studies, indications for admission, the role of sub-specialty consultants, and finally, proper disposition with emphasis on recommendations for followup, referral, or transfer.

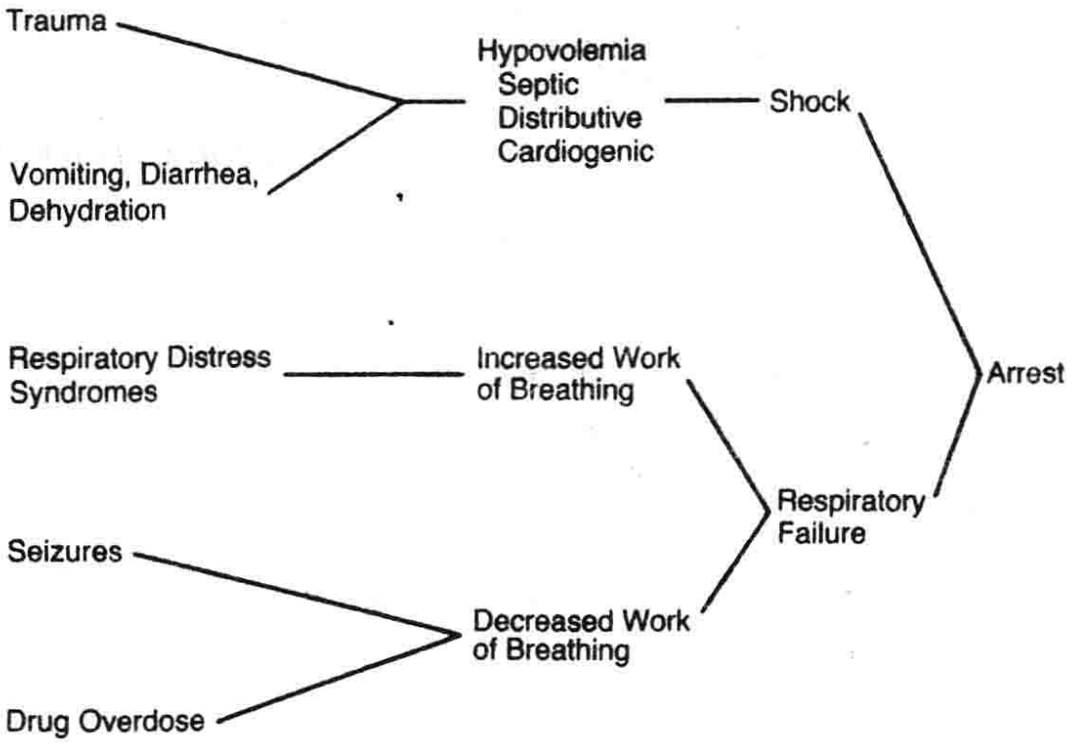
In summary, there exists a variety of methods available for the acquisition and maintenance of current knowledge. However, each of these has its limitations. The advantages of a biannual volume devoted to a specific clinical issue are timely publication and in-depth review of a major topic, blended with practical recommendations for a patient care. I would hope that our series will be used as both a major resource for understanding a specific topic, as well as a clinical tool in the care of the emergency patient.

Ann Harwood-Nuss, M.D.

PREFACE

The entity of pediatric emergency care has been evolving rapidly within the specialties of emergency medicine and pediatrics. This is appropriate since both specialties are involved in the care of children and will continue to be involved in the future. Although research is needed in this area, there is already a wealth of existing knowledge and experience within the two parent specialties. This book consolidates elements of this body of knowledge into a functional educational package written by thoughtful, creative authors.

There are many common disease entities which account for the majority of morbidity and mortality in children. These entities follow two common pathways: shock and respiratory failure. If allowed to progress unabated, both of these pathways terminate in cardiac arrest.



Whereas pediatric life support courses have concentrated more on terminal endpoints as shown on the right half of the above figure, this particular text concentrates more on the presenting symptoms or diseases as shown on the left side. In addition to these specific areas, general topics related to the management of pediatric emergencies are also included.

In this volume the authors address specifically the most difficult or problematic areas related to each disease entity rather than take the more traditional, all-inclusive academic approach to a given topic. Hopefully clinicians will find this volume not only interesting reading but essential to their daily practice of emergency medicine. The book is divided into three sections. The first section deals with general concerns related to pediatric emergency medicine. The chapter on recognition of the sick child serves as an introduction to the book and is followed by guidelines for the use of laboratory studies in the evaluation of the pediatric patient and a chapter on how to optimize the care of children in situations which might be less than optimal. This is followed by a chapter on medical-legal considerations in dealing with pediatric emergencies and a thoughtful chapter on the global problem of child abuse in our society. The subsequent material is divided into a trauma section and a medical section. Included for completeness along with multiple trauma is a chapter on minor trauma and also chapters on evaluation of the pediatric cervical spine and pediatric fractures. Included in the medical section are chapters on the management of dehydration (a frequent precursor of shock), as well as common disease entities of the respiratory failure pathway, specifically seizures, ingested toxins, and respiratory distress syndromes.

It is the intent of this volume to focus on clinical problems related to the care of the pediatric patient in the emergency situation. I wish to thank all the contributors for their untiring and diligent efforts to accomplish this goal. I also wish to acknowledge Ms. Jo-Reid Nichols for her help in preparing this volume and Dr. Robert Wears for his ever-willing attitude to help and offer his thoughtful suggestions.

Robert C. Luten, M.D.

CONTENTS

General Section

Trauma Section

Medical Section

1 Recognition of the Sick Child

Robert C. Luten

Recognition of serious disease is critical to the timely initiation of effective treatment. Recognition encompasses knowledge not only of the presence of disease but, in many cases, of the progression of disease from a relatively benign form to a potentially more severe form. The most sophisticated of treatment modalities are useless if one does not realize the need for their application. This chapter provides an overview of the process of evaluation and recognition of disease in children. It focuses first on the primary caretaker of children in the emergency situation and that person's unique capabilities. Second, we look at the patient who is being evaluated and the spectrum of disease and clinical dilemmas that this patient presents. Last, we examine the process by which this evaluation is accomplished. This chapter serves as an introduction to subsequent chapters that deal with specific disease entities.

THE PRIMARY CARETAKER OF CHILDREN

As of 1985, approximately 5,500 undifferentiated emergency departments saw approximately 75 million patients per year. One-third of these patients, or approximately 25 million, were children. This is in stark comparison to the number of free-standing pediatric emergency departments (50), and the number of patients seen in these emergency departments (approximately 1.5 million) (National Association of Children's Hospitals and Related Institutions, American College of Emergency Physicians, personal

communications). Even if one includes all pediatric residency programs whose emergency departments might or might not be staffed by pediatricians 24 hours a day, the total proportion of children seen initially by pediatricians is still small. Over the past few years, these numbers have changed little, and it remains a fact that the overwhelming majority of children are seen initially in an undifferentiated emergency department, and by nonpediatricians. It is the opinion of this author and others[1] that the two types of physicians who are the primary caretakers of children—pediatricians and emergency physicians—differ in their ability to take care of pediatric emergencies. Emergency physicians are very good at treating obvious emergencies, such as cardiac arrest. They are also capable in airway management and other procedural emergencies; however, they may lack some of the recognition skills needed to manage more subtle emergencies; for example, they may have difficulty identifying early meningitis or early dehydration in a child with vomiting and diarrhea. Pediatricians, on the other hand, are very good at recognizing these subtle emergencies—i.e., have good recognition skills—but tend to be deficient at trauma care and some of the procedural and obvious emergencies (Table 1-1).

It was recently noted that the training in some emergency medicine programs was deficient in coverage of pediatric emergencies.[2] This has been recognized as a problem by emergency physicians and has been addressed with the proposal of an expanded curriculum,[3] expansion of the study guide in emergency medicine, and a cooperative effort between the American College of Emergency Physicians (ACEP) and the American Academy of Pediatrics (AAP) to improve the care of children through the development of an advanced pediatric life-support course. There is also some evidence that the care of infants with severe illness by emergency physicians may be similar to the standard of care that would have been given by pediatricians in certain circumstances.[4]

The AAP has also dedicated its effort to improve the pediatrician's ability to care for the emergent pediatric patient. Besides the cooperative efforts mentioned above, some 15 pediatric emergency medicine fellowship programs have been developed, most of which are based in pediatric programs and which address the needs of pediatricians to care for children with emergent problems.

In the future, economic forces will probably support expansion of facilities dedicated exclusively to the care of children. These facilities will probably be staffed by physicians specifically trained to take care of pediatric

Table 1-1. Relative Strengths and Weaknesses of the Primary Caretakers of Children in Emergency Situations

	Emergency Medicine	Pediatrics
Strengths	Obvious emergencies Procedural emergencies	Subtle emergencies (recognition skills)
Weaknesses	Subtle emergencies (recognition skills)	Trauma Obvious emergencies Procedural emergencies

emergencies. This raises the question, Who is better equipped to take care of children?—the emergency physician who is an expert at emergencies but who rarely sees critically ill children, or the pediatrician who is an expert in the care of children but who rarely manages emergencies? The question is probably moot. Even with expansion of purely pediatric facilities, children will always be cared for in both environments and by both types of physicians. It would be impossible to separate out all children and direct them to a pure pediatric facility. Indeed, the bulk of children will still be cared for by the general emergency physician in the future. It is for this reason that cooperation must exist between the two parent organizations, ACEP and AAP; more importantly, educational efforts directed toward the emergency care of children should be directed at the needs of both types of caretakers.

THE PATIENT

In defining the emergent pediatric patient, we must consider first the spectrum of the normal progression of disease and then look specifically at the clinical problems encountered by the physician caring for children with these diseases.

The Spectrum of Disease

Most pediatric illnesses are benign and self-limited. Of those that do progress further, there are two common pathways—shock syndromes and respiratory failure syndromes—both of which, if allowed to progress, will result in cardiac arrest (Fig. 1-1). The emergency physician is presented with an extremely diverse patient population; the primary job is to pick out the sick child or the child with a potentially serious disease from this large group of undifferentiated patients. Second, and equally important, the emergency physician must be able to recognize when a given patient is progressing further into shock or respiratory failure, necessitating intervention to prevent further progression to the arrest state. Once a child has arrested, the physician's primary responsibility is to optimize resuscitative techniques in an effort to increase survival and produce a good neurologic outcome. Over the past few years, strides have been made in educational efforts centering around the two final common pathways, diseases associated with these pathways, and the management of the patient in cardiac arrest (APLS course). This volume takes one step backward to the recognition of the child who is at risk of progression or sick, followed by recognition of the further progression of these children to a more serious form of their disease. Only when the child is recognized as being different from the undifferentiated patient or as being sick can diagnostic workup and therapy be begun. This same principle applies to the treatment of shock or respiratory failure; that is, it can only be initiated if the clinician recognizes that this syndrome is developing in a given patient.

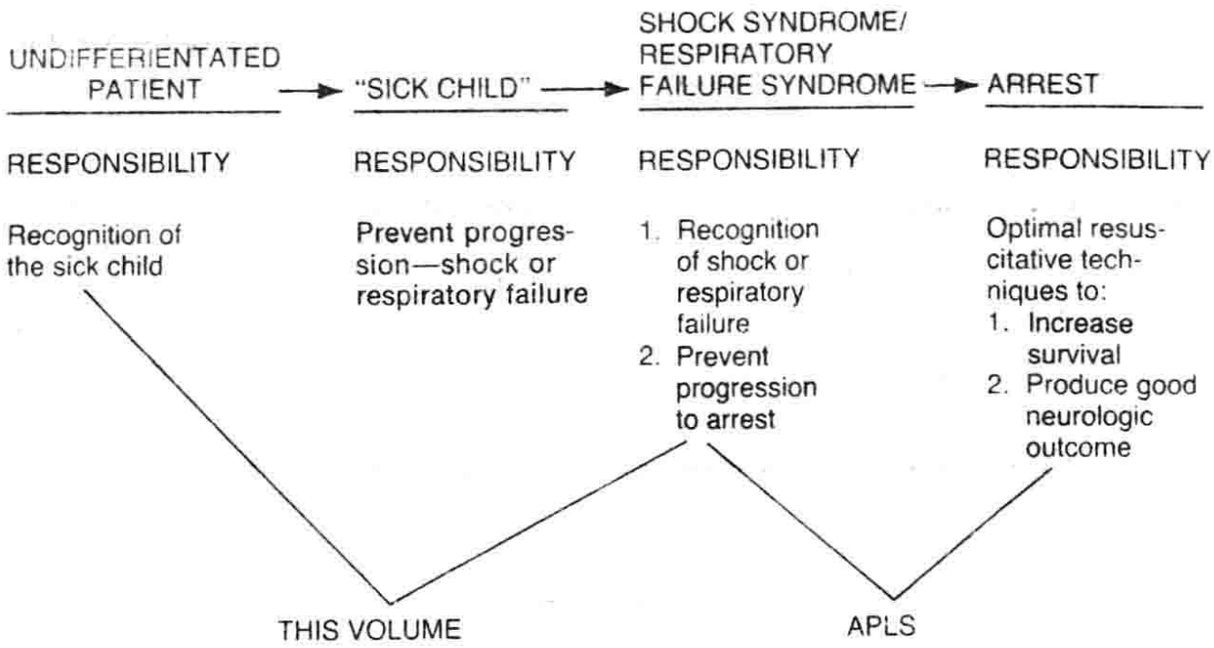


Fig. 1-1 Potential progression of the undifferentiated pediatric patient. Note caretaker's responsibility at each point in the progression.

The Clinical Problem

The term *sick child* is an arbitrary one. From the standpoint of clinical decision-making, and for purposes of this chapter, it covers that group of patients who appear to have the potential for progression to a serious outcome if left untreated. They usually require either some sort of further evaluation or consultation, or both, for ultimate disposition (Table 1-2). From a clinical point of view, we can exclude from the sick group some subsets of patients. The child with a runny nose who is running around the room laughing and screaming, who otherwise does not have any risk factors, such as temperature elevation or age (temperature greater than 104°F, age less than 2 years old), is obviously not sick. Some of these children might require some degree of laboratory evaluation prior to disposition, but it is usually rather clear cut, or the laboratory is only confirmatory in nature, for example, the well-appearing infant with a positive chest radiograph. The obviously severely ill child should also be excluded, i.e., the moribund, comatose infant because, although he is severely ill and has the potential for progression to a poor outcome, he does not represent a recognition problem and appropriate therapy is not delayed. The final exclusion is the child who might appear well but who we know is at high risk of a serious disease, as determined by certain risk factors. These children usually do not pose a clinical recognition problem, since they are recognized by virtue of their risk factors. An example would be a sickle cell patient with a high fever or the newborn infant with a low-grade fever. Once identified, the evaluation, treatment, and disposition are relatively straightforward in most cases. The key is knowledge of the high-risk categories. The pediatrician tends to be cognizant of these high-risk patients, and the emergency physician to a

Table 1-2. Subgroups of Children Presenting as Sick

Group 1	
Severe form of common, usually benign disease *Example:* Gastroenteritis that progresses to dehydration and shock	Appropriate treatment and follow-up can reduce the number of patients in this category. Early recognition of progression of disease and appropriate intervention are keys to reducing morbidity and mortality. All these entities are heralded by logical progression of events or warnings. Problems particular to common diseases in this category are presented in subsequent chapters.
Group 2	
Early form of a serious disease *Example:* meningitis[a] Child who appears clinically ill and requires further diagnostic intervention to formulate a diagnosis (irritable infant who has a positive LP)	These ill-appearing children do not improve with observation and ultimately have specific diagnosis confirmed by laboratory evaluation (positive LP).
Group 3	
Child who appears clinically ill, but for whom serious disease is ruled out by evaluation *Example:* a negative LP (fussy infant who has a negative LP)	These children represent the bulk of children clinically classified as sick. Even though, they are by definition retrospectively classified. They usually require some sort of evaluation (LP), observation, or consultation for ultimate disposition. The size of this subset is inversely proportional to one's clinical experience with children.

[a] The exception is the child who appears clinically well, but who later develops serious disease. These occult conditions are currently the subject of much research and investigation. Many of these children may therefore fall into the category defined as high risk by virtue of age and temperature elevation (less than 2 years, temperature greater than 104°F).

lesser extent. Conversely, there are patients with whom the pediatrician is relatively unfamiliar and with whom the emergency physician has more experience. An example would be the patient who ingests a tricyclic antidepressant who, unlike other overdose patients, is at extremely high risk of rapid deterioration and needs more expeditious intervention than the routine overdose (i.e., immediate lavage versus the slower decontamination route using ipecac).

In the ill-appearing or sick group, three patient categories tend to be identified. The first is a severe form of a common, usually benign disease, such as gastroenteritis that may be progressing to dehydration and shock. The second is an early form of a serious disease, such as meningitis (see

Table 1-2). The child appears clinically ill and requires further diagnostic intervention to arrive at the correct diagnosis. An example would be the irritable infant who has a positive lumbar puncture. For the first group, appropriate treatment and follow-up of the disease on initial presentation can reduce the number of patients in this category. Early recognition of progression of disease and appropriate intervention are the keys to prevention of morbidity and mortality. All the entities in the first group are heralded by a logical progression of events or warnings. An example would be the infant who presents with vomiting and diarrhea. We know that if the process does not resolve, there is potential for progression to dehydration and then to shock. The clinician is thus alerted to the potential pathway a given patient might follow; this aids in the recognition of that progression. Problems particular to many of these common diseases and their recognition are presented in subsequent chapters. The second group, children with early forms of serious disease, do not improve with observation and usually have their ultimate specific diagnosis confirmed by laboratory evaluation. An obvious exception to this group has been the subject of much research over the past few years—the child who has a serious disease but who appears clinically well. These occult conditions are well described in the pediatric literature. Many authorities would classify these children in the high-risk group, that is, as children who appear well but who may be defined as high risk by virtue of certain risk factors, such as age and temperature (under 2 years of age and having a temperature above 104°F). Some consider laboratory evaluation clinically useful in identifying these patients. The chapter on laboratory use addresses this issue.

The third category of ill-appearing children includes those who appear clinically ill but for whom serious disease is ruled out by subsequent evaluation. An example would be a fussy infant who has a negative lumbar puncture. This group represents the bulk of children initially classified clinically as sick. These infants usually require some sort of evaluation, such as lumbar puncture, observation, or consultation for ultimate disposition. The size of this last subset of patients is usually inversely proportional to the physician's clinical experience with children (Fig. 1-2). The more experience a clinician has with children, the more easily he or she can decide, on a clinical basis, whether a child looks well or severely ill and the smaller the proportion of sick children or children requiring further evaluation. This reduction in the total number of sick children is therefore the result of reducing the size of group 3. It also follows logically that the physician who is less familiar with children will rely on more laboratory tests (with a higher percentage of negative lumbar punctures and chest radiographs) than will the more experienced physician. The fact that group 3 is large and even outnumbers the number of patients with serious disease should not be necessarily alarming to the physician caring for children. Several studies have demonstrated the need for acceptance of a high degree of initial false-positive results in the clinical assessment of ill-appearing children in order to pick up the acceptable percentage of truly sick infants.[5] Further evidence is demonstrated by the fact that the vast majority of lumbar punctures done

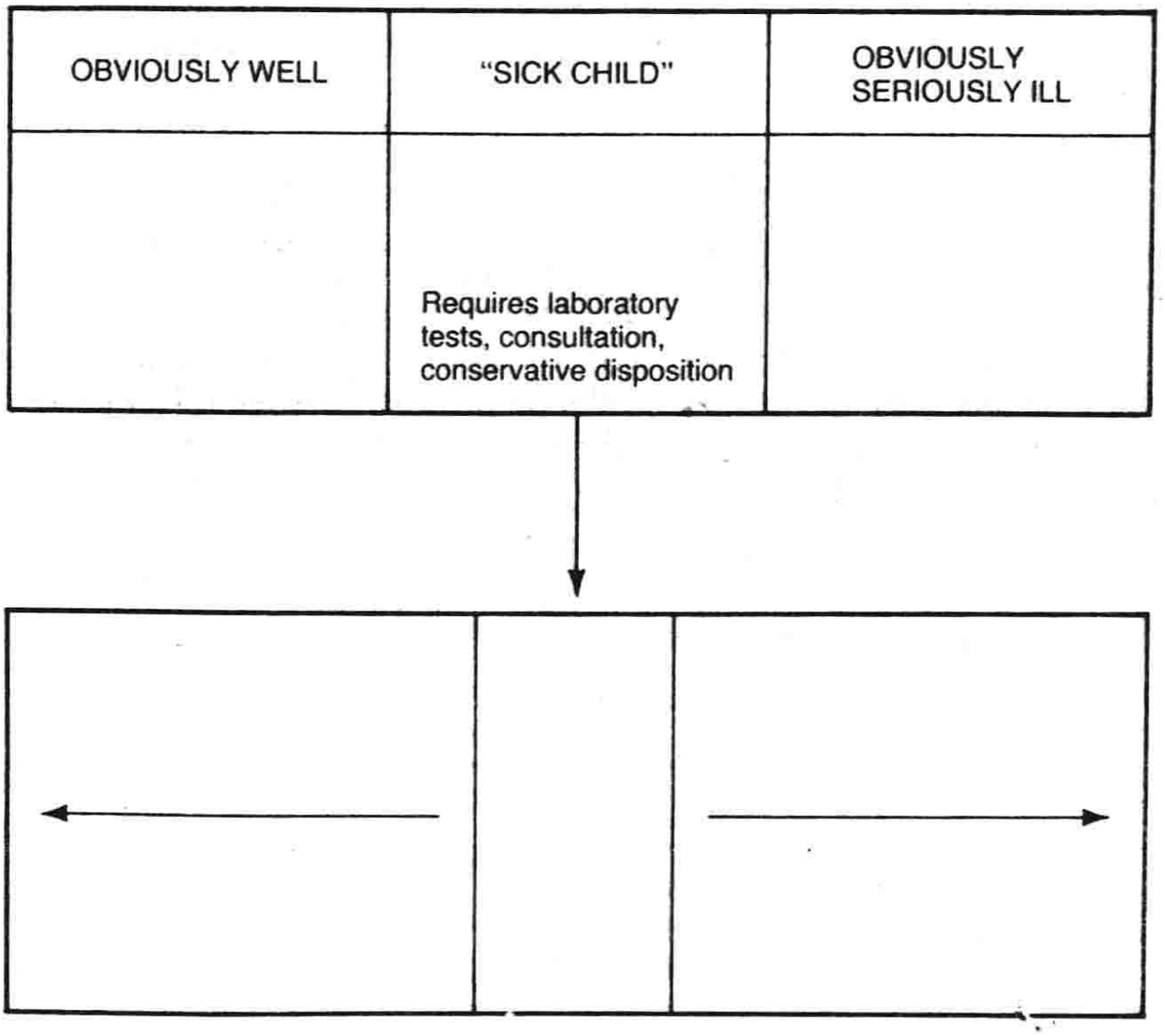


Fig. 1-2 Evaluation categories. With experience, more children will be put into the well or seriously ill categories, thus reducing the subset of sick children. At the same time, fewer laboratory tests and less consultation are required.

on children are negative,[6] even when trained, experienced pediatricians were evaluating sick infants. In other words, these are children who initially appeared clinically ill or sick but who had a negative workup (group 3).

Although laboratory evaluation in many cases ultimately decides the final disposition of sick patients, many children can also be eliminated from the sick category by judicious observation. It has been shown that children with high temperatures, in a strange environment, will act irritable and fussy and indeed will appear more ill than they actually are. In a review of the evaluation of febrile infants, McCarthy[7] described a technique called optimal observation. According to this principle, the infant is given antipyretics to lower temperature and is then placed in the lap of the mother (a comfortable environment) and then is reevaluated. Many infants who initially appeared sick will subsequently appear well. If this is done judiciously in the questionable case, many children can be spared the pain and suffering associated with further diagnostic intervention.

THE PROCESS

This section deals with the process of emergency department evaluation of children. The components of this evaluation are history, observation, physical examination, and laboratory testing (Fig. 1-3). The emergency department evaluation differs from an inpatient evaluation or even the outpatient clinic type of evaluation, in that the amount of time or effort required on one or the other facets of this process might be more or less exaggerated. Although the components of the evaluation for the child and the adult are similar, the relative value of each category in terms of its ability to identify the sick patient is different when applied to the pediatric patient versus the adult patient. Indeed, if the same model is used to evaluate both children and adults, it will most certainly not be effective.

First, let us examine these four variables in relation to the evaluation of pediatric patients. The history has always been considered a crucial part

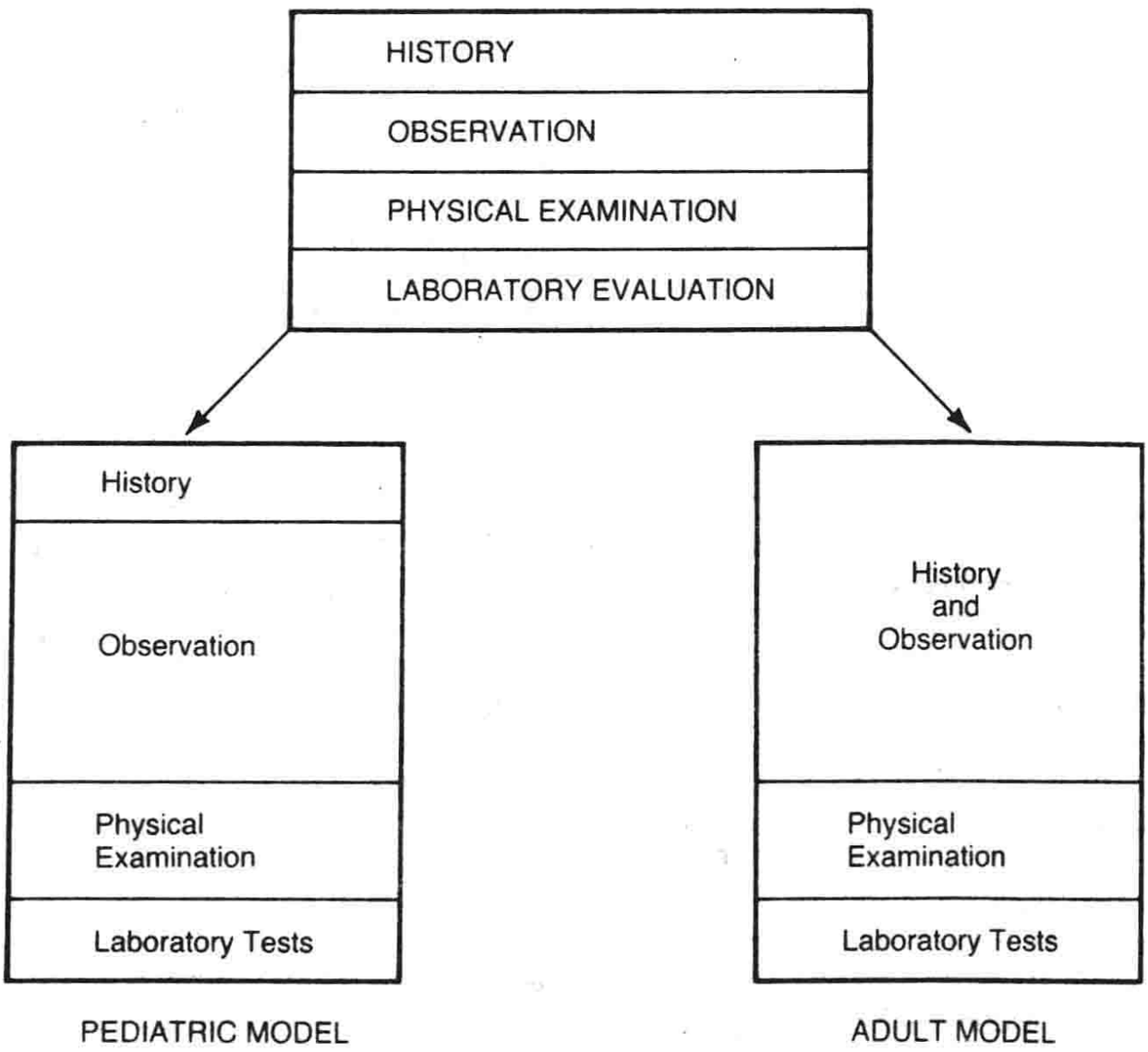


Fig. 1-3 Evaluation component models.

of the evaluative process. However, it has limited use in small infants, since the history of these diseases is so nonspecific (e.g., a urinary tract infection presenting as vomiting and diarrhea). The fact remains that the history, although valuable, is often suboptimal in the emergency department situation. Contributing to a less than optimal history are such factors as lack of familiarity with the parents, presentation of a child in an emergency situation who is not accompanied by a caretaker, or the distraught state of mind of the parents. More important than historical evaluation is observation of the patient for such signs as playfulness, eye contact, and attention to environment. Studies that have compared the relative value of observation variables with historical information in predicting serious disease show that observation of the acutely ill child is more predictive of serious disease than are historical variables.[6,9] These studies also show that if a child is assessed as appearing well, in most cases the child does not have a serious disease.[6,10] Furthermore, patients who are judged ill might or might not actually be ill, as demonstrated by the high number of false-positive results encountered in evaluating the sick child. Much work has been done to determine which of the observation variables are most important in identifying serious disease.[5,8,10] Toward that effort, observation scales and scoring systems have been developed to identify sick children. These scoring scales are global in nature and unrelated to specific organ systems. They have been applied across the board to many different diseases, and not just one specific disease. The point is that the general appearance of an infant as defined by many variables, regardless of the underlying disease being considered, is predictive of the presence of serious disease. Table 1-3 is an observation scale developed at Yale University.

Much work has also been done recently researching the value of laboratory tests in the evaluation of febrile infants. Although the laboratory evaluation has its place in the overall evaluation of an infant, the most common mistake made by physicians is to overemphasize the use of laboratory tests and to depend on them primarily to diagnose severe disease in children. It cannot be overemphasized that the combination of all the essentials of the process is needed to decide whether an infant is sick.

Perhaps the single most important facet of the work that has come out in the past few years concerning the evaluation of the febrile infant, however, is the importance of observation in detecting the seriously ill pediatric patient. Indeed, it has been shown[10] that physical findings that would normally denote serious disease, when combined with an ill-appearing infant, do portend serious disease, but these same findings in a child who appears well do not necessarily imply serious disease. A study of a large group of patients[11] also showed that 20 of 26 children with serious disease were identified on the basis of observation alone (sensitivity of 76 percent). When combined with physical examination, 24 children were identified (sensitivity of 92 percent). Clearly, observation is important in evaluating the pediatric patient, but all facets of the evaluation need to be combined, and no single facet itself is sufficiently predictive of serious disease.

Table 1-3. Predictive Model: Six Observation Items and Their Scales

Observative Item	1 Normal	3 Moderate Impairment	5 Severe Impairment
Quality of cry	Strong with normal tone OR Content and not crying	Whimpering OR Sobbing	Weak OR Moaning OR High pitched
Reaction to parent stimulation	Cries briefly then stops OR Content and not crying	Cries off and on	Continual cry OR Hardly responds
State variation	If awake → stays awake OR If asleep and stimulated → wakes up quickly	Eyes close briefly → awake OR Awakes with prolonged stimulation	Falls asleep OR Will not rouse
Color	Pink	Pale extremities OR Acrocyanosis	Pale OR Cyanotic OR Mottled OR Ashen
Hydration	Skin normal, eyes normal OR Mucous membranes moist	Skin, eyes normal AND Mouth slightly dry	Skin doughy OR Tented AND Dry mucous membranes AND/OR Sunken eyes
Response (talk, smile) to social overtures	Smiles OR Alerts (≤2 mo)	Brief smile OR Alerts briefly (≤2 mo)	No smile Face anxious, dull, expressionless OR No alerting (≤2 mo)

(Reproduced with permission from McCarthy PL, Sharpe MR, et al: Predictive values: Observation scales to identify serious illness in febrile children. Pediatrics 70(5):802, 1982.)

Returning to Figure 1-3, the adult versus pediatric model, it is clear that observation is a large and extremely important part of the evaluative process of children. In the obviously well-appearing infant, observation may be incorporated either consciously or subconsciously into other facets of the evaluative process. For the questionable infant, observation becomes a separate and important diagnostic aid. However, in the evaluation of the adult patient, observation usually is not done separately. In the evaluation of an adult, observation probably represents an overall gestalt and is probably done subconsciously sometime during the history-taking.[12] Application of this adult approach to children is doomed to failure. The tendency of the adult physician, when confronted with the child, is to apply his adult principles of examination. This fact, combined with time exigencies of a busy emergency department, frequently leads the physician to rush quickly from history to physical examination, thereby precluding any gathering of obser-

vational data. In order for a child to be observed, he must feel comfortable in his environment and not threatened. Using the adult approach, therefore, may preclude that ability. Second, if the examination is also done in a rush, and time is not taken to gain the confidence of the child, even findings on physical examination will be masked. It is impossible to palpate the abdomen, test for neck stiffness, or listen for rales in the lungs of a child who is upset and crying. In summary, the typical adult model of evaluation cannot be used in the evaluation of infants. A different approach, with an emphasis on observation, must be taken when evaluating children, if the emergency physician expects to recognize serious disease.

SUMMARY

Recognition of the presence of serious disease is essential if therapy is to be effective. Many common diseases that normally do not portend a serious outcome, if not recognized early and treated appropriately, may indeed progress further. Some of the less common serious diseases may not present in a clinically obvious fashion. This chapter addresses some of the problems in recognition and treatment of many of the diseases encountered in the emergency department. The process of recognition of serious disease in children is complex and is not the same process used in evaluating adults.

REFERENCES

1. Fleisher G, Ludwig S: Preface. p. viii. In Fleisher G, Ludwig S (eds): Pediatric Emergency Medicine. Williams & Wilkins, Baltimore, 1983
2. Ludwig S, Fleisher G, Henrelig F, et al: Pediatric training in emergency medicine programs. Ann Emerg Med 114:170, 1982
3. Ansch SM, Weigand JV: A pediatric curriculum for emergency medicine training programs. Ann Emerg Med 15:19, 1986
4. Sacchetti A, Carraccio C, et al: Community hospital management of pediatric emergencies: Implications for pediatric emergency medical services. Am J Emerg Med 4:1, 1986
5. McCarthy PL, Jekel JF, et al: History and observation variables in assessing febrile children. Pediatrics 65:6, 1980
6. Guraraj VJ, Russo RM, Allen JE, et al: To tap or not to tap? What are the best indicators for performing an LP in an outpatient child? Clin Pediatr 12:488, 1973
7. McCarthy PL: Controversies in pediatrics: What tests are indicated for the child under 2 with fever? Pediatr Rev 1:2, 1979
8. McCarthy PL, Jekel JF, et al: Further definition of history and observation variables in assessing febrile children. Pediatrics 67:5;1981
9. Waskerwitz S, Berkelhamer JE: Outpatient bacteremia: Clinical findings in children under two years with initial temperatures of 39.5°C or higher. J Pediatr 1981

10. McCarthy PL, Sharpe MR, et al: Observation scales to identify serious illness in febrile children Pediatr 70:5, 1982
11. McCarthy PL, Lembo RM, et al: Predictive value of abnormal physical examination findings in ill-appearing and well-appearing febrile children. Pediatrics 76:2, 1985
12. Bolinger RE, Ahlers P: The science of pattern recognition. JAMA 233:12, 1975

2 Problems in the Use of the Laboratory

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Laboratory and radiologic studies can be of great help or great hindrance to the practice of pediatric emergency medicine. At their best, they can establish diagnoses, define severity, and quantitate response to therapy. At their worst, they can mislead,[1] delay, cause needless pain and radiation exposure, add to cost, and even be responsible for clinical deterioration.[2] This chapter attempts to define a few general guidelines for the use of the laboratory in the evaluation of pediatric emergency patients. A few selected laboratory studies are discussed in detail.

PREDICTIVE VALUE AND EMERGENCY ROOM DECISION-MAKING

Evaluation of the value of a laboratory test in a given clinical setting is complex. In addition to such factors as cost, time delays, radiation exposure, pain, and clinical risk, the physician's decision-making process must also consider the predictive value of a given test—the probability that a positive test indicates disease or that a negative test indicates absence of disease. The predictive value must, in turn, be weighed against the risk of over- or underdiagnosis. In general, the greater the severity of disease A, the greater the importance that a test for disease A has a high negative predictive value (false-negative results are less acceptable than false-positives). By contrast, the greater the risk/cost/pain of therapy for disease A, the greater the im-

portance of a high positive predictive value (false-positive results are less acceptable than false-negatives).

Because the role of the emergency room is to identify and stabilize emergency conditions, it follows that high negative predictive values will nearly always be essential if a test is to have high utility in the emergency department. In addition, since many interventions in the emergency department carry risk, high cost (admission), or substantial discomfort, high positive predictive values will often also be extremely important when evaluating a test's utility in the emergency department.

Analysis of the predictive value of a laboratory or radiographic study was described by Galen[3] and applied to the emergency department setting by McCabe.[4] The predictive value of a study is a function of the study's inherent sensitivity and specificity as well as the population to which it is applied. Sensitivity is the likelihood that a patient with condition X will have a positive test. Specificity is the likelihood that a patient without condition X will have a negative test.

Table 2-1. Calculation of Predictive Values

Disease X is known to have an incidence of 5%. The sensitivity and specificity of serum porcelain (SP) for disease X are known to be 60% and 90% respectively. The following distribution of 100 patients can be expected and predictive values determined:

	Disease X		
	Present	Absent	Totals
SP+	3	9	12
SP−	2	86	88
Totals	5	95	100

Incidence = 5/100 (5%)
Sensitivity = 3/5 (60%)
Specificity = 86/95 (90%)
Positive predictive value = 3/12 (25%)
Negative predictive value = 86/88 (98%)

For disease Y, incidence is 1%, sensitivity and specificity of SP are both 90%

	Disease Y		
	Present	Absent	Totals
SP+	9	99	108
SP−	1	891	892
Totals	10	990	1000

Incidence = 10/1000 (1%)
Sensitivity = 9/10 (90%)
Specificity = 891/990 (90%)
Positive predictive value = 9/108 (8%)
Negative predictive value = 891/892 (99%)

Table 2-1 illustrates the derivation of the positive and negative predictive values of a test when sensitivity, specificity, and incidence of illness in a given population are known. Note in the second example that despite a 90 percent sensitivity and a 90 percent specificity, the positive predictive value of the serum porcelain (SP) test is only 8 percent. The example illustrates the strong effect of incidence of disease on the calculation of predictive values.

There are two problems with the application of predictive value analysis to emergency medical decision-making. The first is that sensitivity, specificity, and incidence of disease parameters are often not precisely known for a given laboratory study relative to a given emergency condition. Second, even when these values appear to be reasonably well defined in the medical literature, they do not necessarily apply to the population for which they are selected for use by a given emergency physician. For example, white blood cell (WBC) count specificity and sensitivity data derived from an unscreened population of emergency department patients with abdominal pain may have limited relevance when applied to a subpopulation of patients with abdominal pain preselected for testing by an emergency physician. Here again, tests with very high predictive values will best overcome this problem because extremely high predictive values are least affected by the incidence of disease and, thus, application to selected subpopulations of patients.

When specificity, sensitivity, and incidence are not all well defined or when either sensitivity or specificity is either known or suspected to be relatively low, predictive value theory can still occasionally help to better define the value of a particular test. Two situations that occur commonly are illustrated in Table 2-2. In the first situation, the incidence of disease is low. The example illustrates that in this circumstance positive predictive value is most useful.* Furthermore, in this situation, high specificity is most essential for a high positive predictive value. In other words, when suspicion of disease is low and many patients are being tested who probably do not have the disease, a test is needed with a very low incidence of false-positive results. A routine ankle radiograph to differentiate sprain from fracture is an excellent example. It is a test with high specificity (extremely low false-positive rate) applied to a situation in which the incidence of disease is low (virtually all swollen ankles presenting to the emergency department). The fact that its sensitivity is not extremely high (Salter I fractures are missed) does not negate the value of the test. By contrast, this model would question the use of C-reactive protein greater than 20 to identify occult bacteremia in the febrile toddler (sensitivity 100 percent, specificity 75 percent).[5] Given the low incidence of the disease, the lack of high specificity of the test will result in a very high incidence of false-positive results and, consequently, overuse of antibiotics, the high sensitivity of the test notwithstanding.

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* To clarify the effect of incidence of disease on a test's predictive value, this analysis ignores the issue of severity of disease and invasiveness of treatment. In fact, despite a low incidence, if severity of disease is sufficiently great, negative predictive value may still be crucial.

Table 2-2. Impact of Incidence of Disease on Predictive Values

Disease X has a low incidence of 5% in the population tested. Given the nature of disease X, if no test were available to refine decision-making, the usual course of action would be to assume absence of disease and manage conservatively. In such situations, a positive test result might affect management and a negative would not. Consequently, in this situation positive predictive value is more important that negative predictive value. Assuming a moderate sensitivity of 80% it is clear from the examples below that the specificity must be extremely high (99% vs. 90%) in order for the test to have a high positive predictive value:

	Disease X		
	Present	Absent	Total
Test+	40	10	50
Test−	10	940	950
Total	50	950	1000

Sensitivity = 80%
Specificity = 99%
Positive Predictive Value=40/50=80%

	Disease X		
	Present	Absent	Total
Test+	40	95	135
Test−	10	855	865
Total	50	950	1000

Sensitivity = 80%
Specificity = 90%
Positive Predictive Value=40/135=*30%*

Disease Y has a high incidence of 60% in the population tested. In this situation disease Y would be assumed to be present in the absence of a test to refine decision-making. Hence, in this situation it is negative predictive value that is most important. The following illustrates that negative predictive value is most dependent on a test's sensitivity when incidence is high:

	Disease Y		
	Present	Absent	Total
Test+	594	80	674
Test−	6	320	326
Total	600	400	1000

Sensitivity 99%
Specificity 80%
Negative Predictive Value 98%

	Disease Y		
	Present	Absent	Total
Test+	420	80	500
Test−	180	320	500
Total	600	400	1000

Sensitivity 70%
Specificity 80%
Negative Predictive Value 64%

In the second situation described in Table 2-2, the incidence of disease is high. Here, it is the negative predictive value that is most likely to affect decision-making. The example illustrates that when incidence of disease is high, the negative predictive value is most affected by a test's sensitivity rather than its specificity. For example, many studies would indicate that the sensitivity of the WBC count for appendicitis is quite high, although its specificity is not.[6] When applied to a preselected population of patients (selected by the emergency physician) with a high incidence of appendicitis, rather than as a screen applied to all patients with abdominal pain, the high sensitivity value of the test renders it quite useful.

In summary, in the emergency department, tests with high positive or negative predictive values, or both, are most useful. Tests that have high

Table 2-3. Frequency of Laboratory Tests Performed in a Pediatric Emergency Department

Test	*Frequency* (per 100 patients)
Complete blood count	24%
Chest radiograph	12%
Blood culture	12%
Electrolytes	10%
Long Bone radiograph	9%
Urinalysis	8%
Throat culture	6%
Erythrocyte sedimentation rate	5%
CSF studies	4%

Other tests ordered with a frequency of at least .5% or greater listed in decreasing order of frequency: abdominal radiographs, stool culture, arterial blood gases/venous blood gases, sinus radiographs, drug levels, skull radiographs, clotting studies, drug screening.
Other tests of importance are carboxyhemoglolin, methemoglolin, sickle cell screening, HCG, ECG, stool smear for WBCs, conjunctival smear for gram negative diplococci.

specificity (few false-positive results) will be most likely to have high positive predictive values and will be most useful when suspicion of disease is low. Tests of high sensitivity (few false-negative results) will be more likely to have a high negative predictive value and will be most useful when suspicion of disease is high. Table 2-3 lists several commonly performed tests in the pediatric emergency department (Simon JE, unpublished data). Not surprisingly, given the above analysis, most have high sensitivity or specificity or both. A major exception is the complete blood count (CBC), usually performed to obtain the WBC count.

THE WHITE BLOOD CELL COUNT AND THE EVALUATION OF THE FEBRILE TODDLER

The value of the WBC count in the evaluation of febrile illness has been studied extensively. Its sensitivity and specificity for bacterial illness have been shown to be low.[7] Specifically when applied to the evaluation of the febrile toddler, the WBC count as a predictor of bacteremia has been shown to have a sensitivity in the range of 36 percent and specificity in the range of 62 percent.[8,9] The incidence of bacteremia in toddlers with fever greater than 39.5° and no obvious source is relatively low, approximately 5 percent.[7] Applying the above analysis (value of predictive values relative to incidence of disease), therefore, for a test to be of value in this setting it must have a high positive predictive value. As illustrated in Table 2-4, the positive predictive value of a WBC count greater than 15,000 for occult bacteremia is only 5 percent. The negative predictive value of 95 percent would, at first, appear to be useful, particularly when the potentially severe complications of occult bacteremia are considered.[10] However, in this particular case, the

Table 2-4. Predictive Value of the White Blood Cell Count for Bacteremia

	Bacteremia		
	Present	Absent	Total
WBC > 15,000	19	361	380
WBC < 15,000	31	589	620
Totals	50	950	1000

Incidence 50/1000 (5%)
Sensitivity 36%
Specificity 62%
Positive Predictive Value 19/380 (5%)
Negative Predictive Value 589/620 (95%)

95 percent negative predictive value is no improvement over clinical assessment which, given an incidence of bacteremia of 5 percent, can predict the absence of bacteremia with 95 percent accuracy.

BUN AND SODIUM IN THE EVALUATION OF HYDRATION STATUS

Sensitivity, specificity, and incidence data are often not precisely known for a laboratory test relative to a given clinical condition. Nevertheless, before interpreting a piece of laboratory data, estimates of these parameters must be made if the laboratory data are to be used intelligently. The use of BUN in the diagnosis of dehydration is an example wherein precise data are unavailable. However, using reasonable assumptions and available data, it is possible to estimate specificity, sensitivity, and incidence parameters sufficiently well to evaluate the positive and negative predictive values of BUN. The specificity of BUN greater than 20 for dehydration is easiest to estimate. Since elevation of BUN in acutely ill previously healthy children due to causes other than dehydration is extremely unusual, the specificity of this test can be assumed to be high. For purposes of this discussion, therefore, the specificity of BUN greater than 20 for dehydration will be assumed to be 98 percent. The test's sensitivity is not so impressive. In a recent study by Listernick et al.[11] of 29 dehydrated children, the mean BUN was only 20, with a standard deviation of 8. Although the spread of the data was not provided in this article, assumption of a normal curve distribution would imply that the sensitivity of BUN greater than 20 for dehydration in children is only 50 percent. This is not surprising when one considers that even with total anuria the BUN rises at a rate of only 1 mg percent per hour.[12] Thus, a child who rapidly becomes dehydrated over the course of several hours will not have time to develop a significantly elevated BUN, even if dehydration is severe. Furthermore, a reduction in glomerular filtration rate by 50 percent results in only a doubling of BUN.[13] Consequently, if a given child's normal BUN is only 8, it is clear that

Table 2-5. Predictive Value for the Blood Urea Nitrogen for Dehydration

	Dehydration		
	Present	Absent	Total
BUN > 20	25	1	26
BUN < 20	25	49	74
Totals	50	50	100

Incidence (50%)
Sensitivity (50%)
Specificity (98%)
Negative Predictive Value 49/74 (67%)
Positive Predictive Value 25/26 (96%)

	Dehydration		
	Present	Absent	Total
BUN > 20	10	2	12
BUN < 20	10	78	88
Totals	20	80	100

Incidence 20%
Sensitivity 50%
Specificity 98%
Negative Predictive Value 78/88 (89%)
Positive Predictive Value 10/12 (83%)

significant dehydration could occur without an elevation of BUN to a value greater than 20.

To complete our analysis, we must make an assumption about the true incidence of dehydration among children judged by an emergency physician to be sufficiently likely to be dehydrated to warrant a set of electrolytes, including a BUN. Let us assume an incidence of 50 percent for an experienced emergency physician and 20 percent for a July intern rotating through an emergency department, to illustrate again the effect of incidence of disease on predictive value analysis. Table 2-5 illustrates that the negative predictive value of BUN greater than 20 would be only 67 percent for the experienced emergency physician, not much better than 50 percent based on chance selection after clinical criteria have identified a population with a 50 percent risk of dehydration. On the other hand, for the intern, the negative predictive value would be 89 percent. Unfortunately, this value is achieved at the expense of testing many children for whom dehydration could be ruled out clinically. In fact, for this reason the intern would fail to diagnose as many dehydrated children as the experienced emergency physician, assuming that both regarded a BUN of greater than or less than 20 as definitive.

A test that is often obtained along with the BUN is the serum sodium. Although serum sodium is usually regarded as a means of assessing type of dehydration, rather than assisting in the diagnosis of the presence or ab-

sence of dehydration, it is probably more useful in this regard than the BUN, given the above analysis of the low negative predictive value of BUN. In particular, the serum sodium can be used to sharpen clinical assessment of degree of dehydration. A high-normal or frankly elevated serum sodium implies conservation of extracellular fluid (ECF) at the expense of intracellular fluid volume (ICF). Since most clinical signs of dehydration relate to depletion of ECF, an elevated serum sodium warns the clinician of the risk that clinical assessment may have underestimated the degree of dehydration. Conversely, a low serum sodium suggests that clinical assessment might have overestimated the degree of dehydration.

ARTERIAL BLOOD GASES

The job of the emergency physician is to recognize and stabilize emergency conditions. Reduced to basics, this translates into the recognition and stabilization of actual or impending respiratory failure, circulatory failure, or reversible central nervous system (CNS) deterioration. The arterial blood gas (ABG) provides valuable information in the assessment of all three of these conditions, and does so quickly. PO_2 and PCO_2 values greatly assist in evaluation of both respiratory status and therapy for increased intracranial pressure (ICP) aimed at decreasing cerebral blood volume. The pH and HCO_3^- values are of assistance in assessing the severity of poor perfusion states. The predictive values of the various portions of the ABG are high but not necessarily 100 percent. The agitation produced when performing an arterial puncture can alter the ABG values, unless the arterial puncture is almost immediately successful.

Aside from the issue of predictive value, there is a risk that must be considered before ordering an ABG for a pediatric emergency patient. In the child with impending respiratory failure, the agitation produced or the restraint required during the performance of an arterial puncture can increase the child's respiratory distress and even precipitate respiratory arrest.[2] Similarly, any stimulus, but particularly a painful stimulus, such as repeated arterial punctures, can exacerbate increased ICP. One solution is an indwelling arterial line. However, these are difficult to place and carry their own risks, particularly in an emergency department. Another solution is the use of transcutaneous oxygen saturation monitoring coupled with venous blood gases drawn from an indwelling intravenous line, assuming that the risks of agitation and restraint when placing an intervenous line are deemed acceptable—not necessarily the case for a patient with epiglottitis, for example. Transcutaneous oxygen monitoring has been shown to be extremely accurate. The acid-base parameters of a venous blood gas are similar to those of the ABG, except in situations of peripheral circulatory compromise, provided that the venous blood gas sample can be obtained without use of a tourniquet.

In summary, not even the ABG with its wealth of information should be ordered indiscriminately by the emergency physician. It too can mislead or actually contribute to clinical deterioration.

PLAIN RADIOGRAPHS

The plain radiograph is one of the most frequently ordered tests for pediatric emergency patients (see Table 2-3). The radiation risk of a plain radiograph is thought to be low but not nonexistent. Plain radiographs are costly and, in many emergency departments, time consuming. Adhering to well-defined criteria for ordering plain radiographs would reduce radiation exposure to small children, help control emergency department costs, and improve efficiency in the emergency department. Criteria for ordering plain radiographs have now been developed for many clinical situations.[14-16]

Skull Radiographs

A committee of the Center of Devices and Radiological Health, a division of the US Department of Health and Human Services, recently reviewed the available data on the clinical value of skull radiographs in head trauma.[17] The committee concluded that patients could be divided into three groups on clinical criteria: high, moderate, and low risk of intracranial injury. The committee further concluded that plain skull radiographs would add little to the evaluation of patients in the high-risk group because these patients required computed tomography (CT) scanning regardless of the results of plain radiography. Furthermore, the committee concluded that the low-risk group (accounting for 50 percent of those radiographed in most series) also received no benefit from plain skull radiographs. The incidence of skull fracture in the low-risk group was 1 percent. The risk of intracranial sequelae was zero but estimated at 1 in 2,500 because of statistical limitations. Criteria defining the low-risk and high-risk groups are listed in Table 2-6. The committee made no definite recommendations regarding plain ra-

Table 2-6. Low-and High-risk Criteria for Significant Head Injury

Low Risk	High Risk
Asymptomatic	Depressed Level of Consciousness
Non-progressive headache	Focal Neurological Signs
Dizziness	Penetrating Skull Injury
Scalp Hematoma	
Scalp Laceration	
Scalp Contusion	
Absence of Moderate or High Risk Criteria	

Table 2-7. Moderate Risk Criteria for Significant Head Injury

History of Loss of Consciousness
Progressive Headache
Alcohol or Drug Intoxication
Unreliable History
Age less than 2 years (unless trivial)
Post-traumatic Seizure
Vomiting
Multiple Trauma
Facial Trauma
Possible Penetrating Skull Injury
Suspected Child Abuse

diographs for patients in the moderate-risk group (Table 2-7). They suggested, however, that many in this group would be best managed by extended close observation followed by CT examination if there was either deterioration or failure to improve under observation. The two situations identified for which skull radiographs without CT scanning might be appropriate management were (1) suspected child abuse (for documentation purposes), and (2) suspected depressed skull fracture without clinical signs of intracranial injury (trauma with a pointed or edged object).

Chest Radiographs

A study presented by Gessner and Turkewitz[14] at the 1987 Ambulatory Pediatric Association meetings suggests that radiation exposure to diagnose pneumonia in pediatric emergency patients might be cut in half without significant loss in diagnostic accuracy. Specifically, 129 patients and their chest radiographs were studied to determine whether the lateral chest radiograph added significantly to the overall interpretation of the chest radiographs of pediatric patients presenting with suspected acute pneumonia. The conclusion of the study was that the positive and negative predictive values of a single frontal view of the chest versus those of a two-view study of the chest did not differ. [Next, someone needs to perform a similar study of the kidney-ureters-bladder (KUB) versus the KUB and upright of the abdomen in children presenting with abdominal pain. In fact, KUB versus no KUB might also prove to be an interesting study.]

Long Bone Radiographs

Brand and co-workers[15] studied a set of high-yield criteria for determining the need for long bone radiographs in a mixed population of children and adults presenting with musculoskeletal trauma. Rivera et al.[16] revised the

criteria listed below and studied their application to pediatric patients only.

Upper extremity trauma
  Gross deformity
  Point tenderness
Lower extremity trauma
  Gross deformity
  Pain on motion

The negative predictive value of the upper-extremity criteria listed was only 75 percent—not very good given the above analysis because of the relatively high incidence of fractures among children presenting to an emergency department with the chief complaint of upper extremity trauma. On the other hand, the negative predictive value for the criteria listed for lower extremity injury was 97 percent. Therefore, the absence of gross deformity or pain on motion may obviate the need for radiological evaluation of lower-extremity injury. The numbers in this study were small, however.

ELECTROCARDIOGRAM

The electrocardiogram (ECG) is not performed nearly as often on pediatric as on adult emergency patients. Furthermore, when it is performed, positive findings are infrequent. For example, in a study of chest pain in children, Selbst et al.[18] found that the ECG was performed in only 30 percent of pediatric patients presenting with chest pain. The ECG contributed to the diagnosis in only 4 percent of these, or only 1 percent of the total. Nevertheless, the ECG is a noninvasive test with very high positive and negative predictive values for several emergency conditions in children, principally emergency dysrhythmias. Other than chest pain, presentations for which an ECG should be considered include syncope, unexplained episodes of irritability or lethargy, congestive heart failure, and an unexplained seizure, particularly in a patient with a family history of unexplained sudden death.[19]

URINALYSIS/URINE CULTURE

The limitations of a urinalysis and even a urine culture in the diagnosis of urinary tract infection in infants and children have been well documented.[20] Pyuria may be the result of several pathophysiologic processes including, but not limited to, dehydration, chemical irritation, administration of oral poliovaccine, and gastroenteritis, in addition to urinary tract infection.[20] Also, bacteremia is not necessarily associated with pyuria. Indeed, even symptomatic urinary tract infection may occur without pyuria.[21] Pryles and Lustik[20] studied the relationship of urinalysis findings to urinary tract infections and concluded that the presence or absence of readily

seen bacteria in an unspun urine specimen correlated best with the diagnosis of urinary tract infection. Unfortunately, routine urinalysis in most hospitals only examines the spun sediment—a test that is not well standardized despite its frequent use.

Another confounding variable is the method of urine collection. Bagged urines are easily contaminated. Culture results from bagged urines should be confirmed by means of another collection method before treatment is initiated. Clean catch urine is useful but difficult to collect under age 4. Suprapubic aspiration yields reliable results, but the technique is not without risk.[22] Catheterization is traumatic for toddlers and risks introduction of infection.

Where does this all leave the emergency physician who is confronted with a febrile child? Several recent studies have indicated that the incidence of bacteruria is low in febrile children.[23,24] For instance, in a study of 664 children, Bauchner et al.[23] found bacteruria in only 11 for an incidence of 1.7 percent. (This study probably overestimated incidence of bacteruria in the study population because cultures were performed primarily on clean voided specimens. Furthermore, colony counts greater than 10,000 were regarded as significant.) An additional finding of the study was that 9 of the 11 episodes of bacteruria occurred in infants under 1 year of age. The two older patients were both females. In a study by Roberts et al.,[29] all urinary tract infections identified in febrile children under 2 years of age occurred in females as well. On the basis of these studies, the following approach would appear reasonable when evaluating an infant or child with fever in the emergency department:

Age greater than 1 year, nontoxic

No urine examination is indicated for boys in this category. For girls, parents should be instructed in the proper collection of a bag or clean voided early morning specimen at home. This should be returned and cultured. All positive cultures should be repeated in a clinic or private office setting on a specimen collected by clean void technique under supervision or by catheterization.

Age greater than 1 year, toxic

Catheterized specimens are indicated if no other identifiable source of infection is present. Immediate interpretation should be based on microscopic bacteruria in the unspun urine.

Age less than 1 year, nontoxic

Bag specimens for culture and examination for bacteruria in the unspun urine are indicated. If microsopic bacteruria is found, a second sample obtained by bladder tap or catheterization should be obtained for culture. Treatment should be considered at that point in a child under 6 months of age because of the potential risk of septic complications. Between 6 months and 1 year, treatment may be individualized. If a bag specimen cannot be obtained, bladder tap or catheterization is indicated for all infants under 6 months of age and for all

female infants under 1 year of age without other source for fever. For males between 6 months and 1 year of age, urinalysis and culture in this category is not necessary unless unexplained fever persists.

Age less than 1 year, toxic

All infants in this age group warrant a bladder tap or catheterization for urinalysis and culture.

STOOL SMEAR

For most children presenting with diarrhea, it is not critical to rule out bacterial etiology in the emergency department prior to discharge. If there is reasonable suspicion based on acute onset of diarrhea without vomiting, as well as high fever and blood in the stool,[25] a stool culture is performed, and the child is treated conservatively pending the result. However, in very small infants with diarrhea, it is critical to rule out a bacterial etiology because of the greater suspectibility to septic complications, particularly when the infectious process involves a colitis. In this situation, a stool smear for WBCs can be invaluable.[25] The absence of WBCs has a negative predictive value of 97 percent for bacterial infection with colitis. The presence of WBCs in the stool suggests bacterial, allergic, or amebic etiology. This finding in a very young infant justifies admission or extremely close outpatient follow-up until a definite diagnosis is established.

CONJUNCTIVAL SMEAR

Conjunctivitis in an infant under 2 weeks of age warrants a scraping of the conjunctiva for gram-negative diplococci. If present, gonorrhea ophthalmia must be assumed pending culture results and the child treated appropriately.

OTHER CONSIDERATIONS

One justification often used for obtaining a laboratory test is that it will strengthen the chart from a medical-legal point of view. Predictive value analysis assists in the evaluation of this argument as well. Tests with low positive predictive values have high false-positive rates. A positive result of such a test will therefore often be ignored in favor of evidence thought to be more definitive at the time of initial evaluation. The positive test result can, nevertheless, be used by a skillful attorney to impress a jury that care was deficient. ("Why did you order the test in the first place, doctor?") For example, an elevated WBC count as part of a fever evaluation can, unfortunately, be misused in court to question the failure to perform a lumbar puncture on

a child, the analysis above notwithstanding. Studies with high false-negative rates are safer in this regard but not beyond the use of a shrewd attorney to impugn a physician's care.

A final reason often used to justify a laboratory evaluation is to reassure a parent. This can be difficult reasoning to resist. Certainly, if the test carries any measure of risk, however small, such as radiation exposure, there is sufficient reason to resist a parents's request. Also, if a test is known to have a high false-positive rate, such as a WBC count in a febrile child, this would also be reason to resist a parent's request, because it may well fail to reassure. Furthermore, to submit to the parent's request reinforces the parent's thinking that a child requires a WBC count with every febrile illness, making the job of subsequent physicians more difficult when attempting to reassure the parent. To say that one never yields to a parent's request, however, would be unrealistic. In the final analysis, the goal is an optimal outcome for the child, not purity of medical evaluation. Unfortunately, there are occasional parents whose compliance can only be gained after a "test" has reassured them of the validity of one's medical judgment.

ALTERNATIVES TO LABORATORY TESTING

When clinical evaluation cannot definitively rule in or out an emergency diagnosis and no laboratory study is available with sufficient positive or negative predictive value to be of much help, the alternatives are treatment, consultation, discharge with close follow-up, and observation. Observation in the emergency department itself or in an emergency department holding area is often overlooked as a means of assessment in pediatric emergency medicine. Holding areas have traditionally been used primarily for short courses of therapy for such diagnoses as asthma[26] and dehydration.[11] However, a few hours of observation can assist greatly in the diagnostic evaluation of pediatric emergency patients. Most pediatric emergency conditions have a rather predictable course. After 1 or 2 hours of observation and a dose of acetaminophen, the irritable febrile infant often becomes a happy, smiling, playful infant, ruling out serious infection other than possibly occult bacteremia. Similarly, the evaluation of the lethargic head trauma victim or the irritable accidental poisoning victim will often be clarified after a few hours of observation.

SUMMARY

The role of the emergency department is to recognize and stabilize emergency conditions. In this setting, it is highly desirable that laboratory studies have either high positive or negative predictive values, or both. When suspicion for disease is low or therapy invasive, a test with a very high positive predictive value is needed. When suspicion for disease is high

or severity of disease is high, a test with high negative predictive value is needed. In general, the negative aspects of laboratory/radiography testing are more compelling in pediatric patients compared with adult patients. For example, pain is a prime consideration when dealing with a pediatric patient. Radiation exposure may have greater consequences for young patients. Pediatric patients are at greater risk for clinical deterioration as a result of the agitation produced by a procedure. Therefore, tests that do not add to the medical evaluation of a pediatric emergency patient by virtue of a clear analysis of their utility should not be performed. An alternative to testing when tests of sufficient positive or negative predictive value are not available is observation of the course of illness over a few hours in an emergency department holding area.

REFERENCES

1. Casscells W, Schoenberger A, Graboys TB: Interpretations by physicians of clinical laboratory results. N Engl J Med 299:999, 1978
2. Singer JI, McCabe JB: Epiglottitis at the extremes of age. Presented at the First International Conference of Emergency Medicine, London, 1986
3. Galen RS: Application of the predictive value model in the analysis of test effectiveness. Clin Lab Med 2:685, 1982
4. McCabe JB: Decision making in laboratory test studies. Emerg Med Clin North Am 4:1, 1986
5. Putto A, Ruushanen O, Meurman O, et al: C-reactive protein in the evaluation of febrile illness. Arch Dis Child 61:24, 1986
6. Davis EA, Werman HA, Rund DA: Use of the leuckocyte count and differential in the evaluation of abdominal pain. Am J Emerg Med 4:482, 1986
7. Rasmussen NH, Rasmussen LN: Predictive value of white blood cell count and differential cell count for bacterial infection in children. Acta Paediatr Scand 71:775, 1982
8. Waskerwitz S, Berkelhamer JE: Outpatient bacteremia: Clinical findings in children under two years with initial temperature 39.5 degrees C or higher. J Pediatr 99:231, 1981
9. McCarthy PL, Jekel SF, Dolan TF: Comparison of acute-phase reactants in pediatric patients with fever. Pediatrics 62:716, 1978
10. Carroll WL, Farrell MK, Singer JI, et al: Treatment of occult bacteremia: A perspective randomized clinical trial. Pediatr 72:608, 1983
11. Listernick R, Zieserl E, Davis AT: Outpatient oral rehydration in the United States. Am J Dis Child 140:211, 1986
12. Royer P: Pediatric Nephrology. WB Saunders, Philadelphia, 1974
13. Edelmann CM: Pediatric Kidney Disease. Little, Brown, Boston, 1978
14. Gessner HW, Turkewitz D: Role of the lateral roentgenogram in diagnosis of pneumonia in outpatient pediatric patients. Presented at the Twenty-seventh Annual Meeting of the Ambulatory Pediatric Association, Anaheim, 1987
15. Brand DA, Frazier WH, Kohlhepp WC, et al: A protocol for selecting patients with injured extremities who need x-rays. N Engl J Med 306:333, 1982
16. Rivera FP, Parish RA, Mueller BA: Extremity injuries in children: Predictive value of clinical findings. Pediatrics 78:803, 1986

17. US Department of Health and Human Services: The Selection of Patients For x-ray Examination. US Government Printing Office, Washington DC, 1986
18. Selbst S, Ruddy R, Clark J, et al: Prospective study of chest pain in children. Presented at the Twenty-seventh Annual Meeting of the Ambulatory Pediatric Association, Anaheim, 1987
19. Garson A: Medicolegal problems in the management of cardiac arrhythmias in children. Pediatr 79:84, 1987
20. Pryles CV, Lustik B: Laboratory diagnosis of urinary tract infection. Pediatr Clin North Am 18:233, 1971
21. Geisinger JF: Intermittency of pyuria at the level of the renal papillae. J Urol 25:649, 1931
22. Morrell RE, Duritz G, Oltorf C: Suprapubic aspiration associated with hematoma. Pediatr 69:455, 1982
23. Bauchner H, Phillips B, Dashefsky B, Klein J: Prevalence of bacteruria in febrile children. Pediatr Infect Dis J 6:239, 1987
24. Roberts KB, et al: Urinary tract infection in infants with unexplained fever: A collaborative study, J Pediatr 103:864, 1983
25. DeWitt TG, Humphrey KF, McCarthy P: Clinical predictors of acute bacterial diarrhea in young children. Pediatr 76:551, 1985
26. Willert C, Davis AT, Herman JJ, et al: Short-term holding room treatment of asthmatic children. J Pediatr 106:707, 1985

3 Optimizing Pediatric Emergency Care with Sub-optimal Resources

Joseph Simon

The concept for this chapter was conceived during a conversation with a pediatrician from a small community who was implicated in a malpractice case involving a child with epiglottitis. In was clear from the conversation that this pediatrician and the emergency physician who was also named in the legal action both clearly understood the pathophysiology and appropriate treatment of epiglottitis. In particular, they both recognized the desirability of quickly establishing an artificial airway. However, because they did not feel technically capable of establishing an artificial airway in a patient with epiglottitis, and because they could not identify any other physician in their small community who did feel capable of performing this difficult procedure, they contacted a tertiary care pediatric center approximately 75 miles away and requested that an air-transport team be dispatched by this center to assume responsibility for the child. The tertiary care center refused because they were unwilling to transport a child with epiglottitis without an artificial airway (their team also lacked the skill to intubate or perform a tracheostomy on an epiglottitis patient). The pediatrician and the emergency physician therefore attempted to intubate the child without success. The child deteriorated during the attempt. Again, the managing physician contacted the tertiary care center and again transport was refused. The pediatrician and emergency physician then elected to transport by local ground rescue unit to the tertiary center. During transport, the child arrested and could not be resuscitated. Did these physicians make the correct decision? Was the decision of the tertiary care center appropriate? Could advance planning have improved this child's chances for survival?

The answers to these questions are difficult. Most medical research is conducted at medical centers with abundant medical resources. Consequently, it is particularly difficult to find answers to these questions in the medical literature. In particular, the relative risks of various courses of management, none of which is regarded as ideal, are not well researched. This chapter discusses general solutions to the problem of suboptimal medical resource availability. A few selected situations are then discussed in detail, using information available in the literature to speculate on the optimal course of management for each situation.

SCOPE OF THE PROBLEM

Fortunately for children, both mortality and the incidence of life-threatening illness and injury in the pediatric age group are low. Unfortunately for children, a consequence of this low mortality and low incidence of serious disease and injury is that physicians' experience in the management of life-threatening pediatric problems is also limited. To illustrate the problem, imagine a hypothetical community called community A of 100,000 people served by two emergency departments and eight emergency physicians. Twelve infant deaths, excluding neonatal deaths, would be expected to occur in this community each year.[1] Assuming a 20 percent successful resuscitation rate (high) for emergency department resuscitations of pediatric patients and assuming that one-half of 12 deaths involved an emergency department resuscitation, the number of infant cardiopulmonary resuscitations conducted in community A's two emergency departments can be estimated at eight, or four per facility, or one per emergency physician per year.

As another example, community A would be expected to have two cases per year of intussusception, one per emergency department per year or one per emergency physician every 4 years.[2]

Another consequence of this low incidence of serious illness in the pediatric age group is the limited number of pediatric subspecialists trained to care for critically ill pediatric patients. Community A would be unlikely to possess either a pediatric radiologist experienced in barium enema reduction of intussusception or a pediatric surgeon. In fact, the 1985 to 1986 Directory of Medical Specialists in the United States devotes 310 pages of small print to the listing of general surgeons as compared with a total of only 400 pediatric surgeons for the entire country.

In summary, when a pediatric emergency occurs outside a major metropolitan area, optimal resources to manage the emergency are often not going to be available. Furthermore, many of those physicians who are called on to manage these pediatric emergency situations will lack that critical level of experience with pediatric emergency situations essential to optimal provision of care despite otherwise excellent training and expertise in adult emergency care.

COMPENSATING FOR SUBOPTIMAL RESOURCES

Recognition

The first step toward solving a problem is recognizing that a problem exists. Any emergency department caring for children must accurately assess its capabilities in the following areas:

1. *Pediatric airway management:* ability to establish an artificial airway in the pediatric patient with upper airway obstruction, availability of flexible and rigid bronchoscopy for the management of both lower and upper airway obstruction
2. *Pediatric respiratory failure:* availability of physicians and intensive care unit personnel knowledgeable and experienced in ventilator management of pediatric patients, including sedation and restraint of pediatric patients, and pediatric pulmonary toilet
3. *Pediatric dysrhythmias, congestive heart failure, shock:* availability of physicians knowledgeable and experienced in these areas, including pediatric echocardiography, ability to gain rapid venous access, including central venous access, in sick infants and children
4. *Pediatric central nervous system resuscitation:* availability of pediatric cranial computed tomography (CT) capabilities; availability of a neurosurgeon capable of decompressing intracranial mass lesions in pediatric patients, establishing intracranial pressure monitoring and managing ventricular shunt malfunctions
5. *Pediatric surgical emergencies:* availability of a surgeon experienced and skilled in the management of pediatric bowel obstruction and pediatric multiple trauma; availability of an anesthesiologist skilled and experienced in the emergency anesthetic management of infants and children
6. *Appropriate equipment, laboratory, and radiological support:* for the resources listed above

Other pediatric resources are obviously desirable. This list attempts to outline only those resources for which very prompt or emergent availability might be life saving.

Maximizing Availability of Key Resources

The next step is to work toward maximum availability of those community resources that do possess needed skills. For example, separate call schedules listing only physicians interested and skilled in the care of pediatric patients might be developed by an emergency department. This would serve the dual purpose of concentrating pediatric experience among a few specialists, improving the likelihood that these physicians will remain current in pediatric emergency care within their field. For example, this might serve as an inducement to a physician to pursue appropriate continuing

medical education experience in pediatric emergency care. In fact, one incentive a hospital might use to gain participation in a pediatric subspeciality call schedule might be formal support for pediatric continuing medical education activities for those who participate. Nonphysician resources should not be overlooked when developing plans for the management of pediatric emergencies. For example, selected intensive care unit nurses or respiratory therapists might be identified as more skilled in the care of infants and children. Often the only inducement necessary to gain the ready availability of these personnel will be the recognition of their expertise.

Protocols

The third step is the development of protocols for the management of selected pediatric emergency situations. Aside from providing immediate direction to emergency department personnel, protocols serve at least three other purposes. First, protocols can serve as an effective component of an emergency room's in-service educational program. Second, by involving key resources in the community in the writing of the protocols, the level of cooperation of those resources with the management of a given pediatric emergency situation when it arises is solidified. Finally, a protocol, if followed, can be a protective instrument when legal action is threatened or pursued, particularly with respect to institutional liability. Protocols should be specific. In particular, they should not only provide direction regarding key decisions in management but also provide nurses with direction in the areas of monitoring and appropriate equipment/medication to assemble. Do not's as well as do's should be included in the protocols.

One concern regarding detailed protocols is that they limit individualization of medical care. They do not. Deviation from a protocol is and should be permitted provided that there is detailed documentation in the chart of the reasons for variation from the protocol. On the other hand, protocols can be used effectively to limit the choices of a physician who is not prepared to document appropriate reasons for deviation from protocol. Finally, all protocols should define indications for patient transfer to a tertiary pediatric facility.

Transfer Agreements

The Accreditation Manual published by the Joint Commission on Accreditation of Hospitals recommends that hospitals that lack tertiary care capability in a given area develop formal transfer agreements with hospitals that do provide such care.[3] In particular, any hospital that lacks tertiary care capability for selected or all pediatric patients should have a transfer agreement with a pediatric tertiary center. These agreements can greatly expedite the emergency transfer of patients,[4] define responsibilities

during transfer, and clarify transfer procedures. These agreements can guarantee that referrals from a given emergency department will be accepted without regard to the patient's financial status. On the other hand, these agreements can provide a measure of security to the tertiary institution that referral will not be based upon financial considerations. One aspect that these agreements should, however, not attempt to define is the medical indications for transfer.[5] These indications should be incorporated into the referring hospital's protocols. The tertiary pediatric receiving hospital should guarantee acceptance of any patient which the referring hospital desires to transfer subject only to considerations of resource availability at the receiving institution. Figure 3-1 is an example of a transfer agreement that incorporates many of these characteristics.

Pediatric Interhospital Transport Systems

When protocol calls for transfer and a tertiary pediatric facility has accepted the infant or child in transfer, the next step is rapid and skilled transport to the receiving facility. Referring institutions should work with receiving institutions and with the various transport resources within an area to identify, train, and support a transport system that has special expertise in the area of pediatric transport. Guidelines for such a system have recently been developed by the American Academy of Pediatrics.[5] Ideally, the system should be exclusively a pediatric transport system. In most areas, however, practical considerations will dictate that the system will share many components with a general transport system. One aspect that should not be shared, however, is the medical directorship of the pediatric system.[5] Also, it is desirable to have at least one member of the transport team who possesses special expertise in the care of the critically ill/injured infant or child.

If the urgency of the situation does not permit the delay inherent in a round-trip transport by a pediatric transport team dispatched from the receiving tertiary center, then local transport resources must be available that at least possess appropriate equipment and operate under appropriate protocols for the management of pediatric patients. Local hospitals and emergency departments should work with local transport services to guarantee at least this minimum level of pediatric transport expertise. In these situations, arrangements should also be made, when necessary, for a physician from the local community with pediatric critical care knowledge to accompany the local EMS team during transport. Unless these arrangements are made in advance, it can be very difficult to assemble a transport team quickly with the maximal skill available in the community and with appropriate equipment.

Finally, it must be recognized that stabilization before transport from one hospital to another is not necessarily equivalent to stabilization before admission to the same hospital. Transport conditions such as noise, vibra-

Transfer Agreement Between _____ and _____

To facilitate the transfer of patients between _____ Hospital (hereafter referred to as "_____") and _____ (hereafter referred to as "_____"), the following procedures are set forth and agreed upon by the two institutions. The guiding principle of these procedures is to maintain the best interest of the patient in mind while transferring the patient from one institution to another.

1. The decision to transfer a patient shall be made jointly between the attending physician or his designate at _____ and the receiving physician or his designate at _____. The joint decision will assure that adequate facilities are available at _____ for a patient who is properly prepared for the transfer.

2. A copy of the complete medical record, including x-rays and laboratory reports, must be transferred with the patient. In addition, the following information should be available prior to the patient's transfer: (a) medical authorization; (b) patient's or guardian's consent; and (c) proper patient identification.

3. Patients who require a burn unit for their inpatient care shall not be transferred to _____.

4. The physician sending the patient and the appropriate physician at _____ shall determine the necessity for medical or support personnel to accompany the patient and the appropriate mode of transportation. (Land ambulance or helicopter.)

5. When a patient does not require a physician to accompany the patient, _____ shall provide appropriate support personnel during the transfer. In this case, the source and mode of transportation should consider returning personnel to _____.

6. When a physician from _____ does not accompany the patient in transit, _____ shall assume medical responsibility for the patient until the patient arrives at the Emergency Department of _____. Upon such arrival, _____ will assume medical responsibility for the patient.

7. When a physician from _____ accompanies the patient in transit, _____ shall assume joint medical responsibility for the patient once the patient has been determined clinically stable for transportation and has departed from _____. Upon arrival at the Emergency Department of _____, _____ will assume medical responsibility for the patient.

Fig. 3-1 Sample form of a transfer agreement (*Figure continues.*)

8. _____ is not responsible for any costs associated with the transportation of a patient by land or by air. Such costs shall be billed by the organization providing the transportation to the responsible party of the patient.

9. _____ encourages the utilization of medical communication between the vehicle transporting the patient and the Emergency Service's radio at _____.

10. All patients transported to _____ shall be received in the Emergency Department of the hospital, unless other arrangements have been made by the receiving physician at _____.

11. _____ and its physicians agree to provide appropriate information to the referring physician and hospital about the progress and disposition of the patient after the transfer is completed and for the duration of the patient's stay.

12. _____ agrees to accept patients under age 18 without regard for the patient's financial or third-party funding status.

13. _____ _____ agrees to make the decision regarding the transfer to _____ vs. admitting the patient or transferring to another institution without regard for the patient's financial status or third-party funding.

14. In the event either party in its own unilateral judgement determines to perform under this agreement and corrective efforts made in good faith by both parties are unsuccessful, either party may terminate this agreement upon the giving of thirty (30) days prior written notice without penalty.

15. This agreement shall be in effect for a period of one (1) year beginning on the date of signature below. It shall be automatically renewed on an annual basis, unless either party expresses their intent in writing to terminate or renegotiate the agreement at least sixty (60) days prior to the renewal date.

_____ _____

Director, Emergency Services _____

_____ _____

Aministrator, _____ Administrator, _____

_____ _____

Date Date

Fig. 3-1 (*Continued*).

tion, temperature, and limited space often dictate different stabilization measures than would otherwise be indicated. The subject of pediatric transport has been reviewed elsewhere.[6] Pediatric emergency care hotlines or the medical control physician from the pediatric transport system can be useful resources to tap for advice on appropriate stabilization prior to transport.

Pediatric Emergency Care Hotlines

Many states, regions, and tertiary care pediatric centers have taken the initiative to establish the availability of telephone consultation on pediatric emergency cases. For example, in the state of Georgia, the Emergency Health Section of the Department of Human Resources has funded an 800 number located at a pediatric emergency department in the state, staffed by pediatric emergency attending physicians. This line provides emergency physicians and pediatricians throughout the state with immediate round-the-clock access to consultation. The line can be used to obtain recommendations on the management of children who do not necessarily require transfer or recommendations on the stabilization of pediatric patients prior to transfer. These hotlines, however, are not a substitute for aggressive advanced planning and protocol development by local hospital emergency departments. The advice that can be provided by these hotlines will be limited by the availability of local resources. At a minimum, accurate knowledge of availability of local resources must be provided to the consultant to permit this consultant to assist in the development of sound medical judgments.

In-service Education/Quality Assurance Evaluation

Finally, local emergency facilities that are only infrequently challenged by a critically ill or injured child should emphasize pediatric emergency care in their in-service training and quality assurance evaluation. Although this might, at first glance, appear contradictory, a strong case can be made for experience as the best teacher. Consequently, when experience is lacking, extra emphasis must be placed on other modalities of maintaining a medical skill or a body of medical knowledge. Periodically providing physicians and nurses with the opportunity to obtain clinical experience in a high-volume pediatric emergency department is another way of supplementing an emergency room's limited exposure to critically ill or injured infants and children.

In summary, there are a number of steps that an emergency department, or more generally, a local community can take to compensate for the limited availability of resources for the care of pediatric emergency patients. Frequently, there are more resources than are immediately apparent. One dedicated physician can promote through the development of proto-

cols, pediatric call schedules, transfer agreements and other means the mobilization of a community's resources to a level which will be more than adequate for the large majority of pediatric emergency patients.

EPIGLOTTITIS

The case described earlier of a child with epiglottitis provides a classic example of a pediatric emergency for which optimal resources simply cannot be expected to be available at every community emergency department. Many recent articles advocate establishment at prompt provision of an artificial airway, by intubation or tracheostomy, as the optimal care for a child with epiglottitis.[7–9] However, intubating a child with edematous supraglottic structures or performing an emergency tracheostomy on a struggling three year old requires specialized skills—skills that are difficult to maintain when the need for them arises only infrequently. Furthermore, attempting either of these procedures carries the risk of irreversible respiratory arrest if unsuccessful. What is the solution for the small community hospital emergency department that must, on rare occasion, manage a child with epiglottitis?

The first step is to identify, if possible, either an anesthesiologist or surgeon, or both, within the community who possess the needed skills. If present, their availability should be solicited. However, the limits of their availability need to be clearly defined for their own medical-legal protection.

If such resources are not available, the next step is to develop a protocol that maximizes all other aspects of the care of a child with epiglottitis, short of establishing an artificial airway. For example, this protocol should minimize the child's agitation, provide oxygen if it does not produce agitation, encourage the sitting position in a parent's lap, and provide for close monitoring and the availability of needle cricothyroidotomy equipment at the bedside. In particular, an IV line is no substitute for an artificial airway and may aggravate the situation. Therefore, the protocol should either prohibit establishing an IV line—a reflex act for most medical personnel when faced with a sick patient—or should clearly authorize IV placement only if placement without causing the child to become agitated is possible.

The key question that this epiglottitis protocol must address is disposition. Should an artificial airway be attempted by a physician who is uncomfortable with this procedure? Should the child be admitted and managed without an artificial airway? Should transfer without an artificial airway be attempted? Management without an artificial airway has been studied. Glicklich et al.[10] used dexamethasone and otherwise conservative management in 40 of 53 patients successfully. Another interesting aspect of this study was that bag-mask-valve ventilation was a successful temporizing measure when respiratory failure occurred. This approach has been confirmed by others.[11,12] Costigan and Newth[13] also attempted conservative

management of 40 selected cases out of a total of 170. Only 2 of the 40 patients subsequently required an artificial airway. In both studies, however, conservative management was attempted in a situation in which placement of an artificial airway was readily available in the event of clinical deterioration. To choose this course of action in a community hospital without the in-house capability of emergency airway management of a patient with epiglottitis would be ill advised. However, both studies suggest that rapid transport of a child without an artificial airway who is early in the course of epiglottitis might be a viable option, given that many children with epiglottitis have been successfully managed without an artificial airway. Furthermore, the first study indicates that deterioration during transport might be successfully managed by someone skilled in bag-mask-valve ventilation of the pediatric patient. (See Chapter 12 for further discussion of epiglottitis.)

In summary, when the resources to provide an artificial airway to a child with epiglottitis are simply not available a reasonable alternative is a protocol that calls for:

1. Minimal agitation
2. Oxygen, if tolerated, in the sitting position
3. Close monitoring
4. Rapid transport to a nearby facility accompanied by the local physician who is most skilled in a bag-mask-valve ventilation of children and who is familiar with the technique of needle cricothyroidotomy; or transport via a pediatric transport team dispatched by a tertiary pediatric center with these same skills (If the child's respiratory status is rapidly deteriorating then attempting an artificial airway at the referring hospital may be the only viable option.)

Arrangements with the receiving facility and participating local physicians and local EMS services should be well formulated in advance to facilitate rapid transport.

INTUSSUSCEPTION

Intussusception is another pediatric emergency that requires specialized management resources. Yet intussusception occurs so infrequently that it is difficult for a smaller community to develop and maintain these resources. It is generally accepted that the optimal management of intussusception is prompt barium enema reduction under the supervision of a surgeon who is prepared to operate quickly in the event that barium reduction fails.[14,15] If these resources are not available, rapid transport must be arranged. The problem that this poses is the increased risk of bowel ischemia or necrosis engendered by the delay involved in transport to another facility. Can this risk be reduced through medical management at the referring hospital prior to transfer? The answer is unclear. However, two forms of

therapy warrant consideration for inclusion in local protocols dealing with intussusception. The first is aggressive fluid therapy to maintain perfusion to the splanchnic bed. Several factors may conspire to magnify the compromised perfusion to the intussusceptum that results from its anatomic distortion. First, the infant may be dehydrated from repeated vomiting and poor intake. Second, blood may be lost through already ischemic bowel producing the classic currant jelly stool.

Finally, there is evidence that endogenous opioids may be secreted in large amounts in some patients with intussusception.[16] These opioids may produce vasodilation and shunting of blood away from the splanchnic bed. Consequently, a fluid bolus of 20 ml/kg of saline or Ringer's lactate followed by additional isotonic fluid to restore and maintain extracellular fluid volume would seem appropriate prior to transfer. Indeed, many protocols call for aggressive fluid therapy prior to an attempt at barium reduction even when transfer is not involved.[14,15]

Another approach to improving perfusion to the intussusceptum is reduction in the tone of the smooth muscle of the bowel. Administration of glucagon is known to have this as one of its pharmacologic effects. In fact, glucagon has been used as a substitute for barium reduction with limited success[17] and as an adjunct to barium reduction with more apparent success.[18] Haase and Boles[19] studied the effects of glucagon in experimental intussusception in puppies. He found that it resulted in "easier reduction, and an easier return of normal vascular supply as measured by color and by arterial pulsations."

In summary, a reasonable protocol for the management of intussusception at a community hospital emergency department would be:

1. Nasogastric decompression
2. One or more boluses of 10 to 20 ml/kg of Ringer's lactate followed by continuous infusion of Ringer's lactate of at least a maintenance rate
3. Administration of 0.05 mg/kg of glucagon by the IV or IM route
4. Rapid transport to an appropriate tertiary care facility by personnel skilled in the diagnosis and management of circulatory compromise in infants

OCCULT BACTEREMIA

Five percent of infants between the ages of 6 and 24 months with temperatures greater than 39.5°C and with no obvious source of infection will have positive blood cultures, usually *Pneumococcus,* occasionally *Hemophilus influenzae.*[20] This phenomenon of occult bacteremia of infancy has been the subject of much study.[20–22] Unfortunately, no one has as yet identified a serum porcelain level that identifies infants with occult bacteremia with sufficient positive and negative predictive value to be of much use. Consequently, after obtaining appropriate blood cultures on infants with high

fever, one is faced with the alternatives of either treating all such infants empirically with antibiotics or withholding treatment pending culture results. The best course of action is vigorously debated. The most compelling data in favor of expectant antibiotic therapy has been provided by Carroll et al.[23] In their study of 96 infants, 10 of which were bacteremic, a randomized double-blind prospective study method indicated that expectant therapy could prevent occult bacteremia from developing into meningitis. By contrast, several retrospective studies do not support this conclusion.[24,25] One variable that is never discussed in the conclusions of these studies is the laboratory methodology used in the processing of a blood culture. Culture technologies vary greatly in their speed and accuracy. Radioisotopic techniques will usually identify *Pneumococcus* and *Hemophilus influenza* bacteremia within 12 hours.[26] Other techniques may require up to 48 hours before yielding reliable results. Although no data are available on the rapidity with which occult bacteremia typically evolves into meningitis it would seem prudent for an emergency room which does not have the support of rapid blood culture techniques to adopt a protocol of expectant antibiotic therapy for occult bacteremia. The antibiotic used in Carroll's study was Bicillin CR 50,000/kg IM, followed by penicillin V 100 mg/kg/day orally divided into four doses. Unfortunately, this does not adequately cover the occasional infant with *Hemophilus influenza* bacteremia. Nevertheless, it is the only prospectively studied antibiotic regimen for which there is evidence of efficacy at this time. On the other hand, the numbers in Carroll's study were small. Consequently, the choice of another antibiotic regimen, such as oral amoxicillin, would be difficult to criticize.

In summary, an emergency room must consider the capabilities of its laboratory support as it establishes policies and protocols for the management of the various emergency conditions it will be confronted with. In the case of occult bacteremia of infancy, a reasonable protocol for an emergency department without the support of rapid blood culture techniques would be as follows:

1. Culture of all infants aged 3 to 24 months without an obvious source of infection and with a temperature measured at home or in the emergency room of greater than 39.5°C
2. Amoxicillin, 50 to 75 mg/kg/day divided in three doses for 3 days, pending blood culture results
3. Reexamination within 24 hours by a physician
4. Reevaluation within 72 hours when culture results are known (phone evaluation may be permissible in some cases)

VASCULAR ACCESS

One of the greatest challenges in pediatric emergency care is establishing rapid vascular access in a poorly perfused infant. It has been well documented that even physicians experienced in pediatric care employing cut-

down and central venous access techniques, in addition to percutaneous techniques, often fail to adequately meet this challenge.[27] How does a community hospital emergency department with its low volume of critically ill small infants develop and maintain its skill in this area? If the hospital has a neonatal intensive care unit or a separately staffed pediatric inpatient service, one solution is to arrange for nurses from these areas to respond to the emergency department when a critically ill or injured infant arrives. These nurses will often possess considerable skill at establishing rapid venous access in sick infants. However, even nurses with considerable experience with the task can have difficulty. An old solution to the problem that has recently been revived and extensively reviewed is vascular access via an intraosseous route.[28,29] A stiff, large-bore, styleted needle is placed in the marrow space of the anterior tibia or distal femur. This technique is simple and reliable and requires only seconds to perform. A recent study by Kanter et al.[27] of the effect of incorporating intraosseous needle placement into a protocol for rapid vascular access demonstrated a reduction in mean time to successful vascular access from 10 to 4.5 minutes. Even more significant, this study documented a reduction from 40 to 10 minutes in the maximum delay experience before vascular access was achieved after incorporation of intraosseous infusion into standard protocol. The technique is safe. All resuscitation medications, including continuous infusions of pressors[30] and rapid fluid boluses, can be administrated through an intraosseous needle.

Every emergency department, and in particular those that lack either a large number of critically ill infants to maintain their skill at vascular access or other in-house resources that can be of assistance, should develop a protocol for vascular access incorporating the intraosseous route. An intraosseous needle should be readily available. Although little skill is needed to place an intraosseous needle, in-service training using various manikins (chicken legs work well) should be conducted regularly to develop familiarity with the procedure. Ongoing quality assurance studies in an emergency department should routinely identify every critically ill infant in which vascular access required more than 5 minutes. Failure to employ intraosseous infusion in an infant so identified should trigger careful review.

HEAD TRAUMA

Trauma deaths account for more deaths between the ages of 1 and 14 years than all other causes combined.[31] Severe head trauma accounts for 60 to 80 percent of this mortality.[31] Optimal resources for the management of the pediatric trauma victim in general, and the pediatric head trauma victim in particular, have been described by Ramenofsky.[32] Included in these resource guidelines, which are currently being used with various modifications to designate institutions as separate pediatric trauma centers, are such resources as a pediatric neurosurgeon, pediatric anesthesiologist, pediatric operating room team, and pediatric intensive care unit. These resources will not be available to most emergency departments. One solution

to the problem is for emergency departments to cooperate with a regional trauma system that directs severely injured pediatric patients directly from the scene of an accident to those hospitals in the area that possess appropriate resources for the management of the pediatric trauma victim in general and the pediatric head trauma victim in particular. Unfortunately, in many communities no hospital will possess all the needed resources. Consequently, many emergency departments will be called upon to manage a pediatric head trauma victim without the availability of optimal resources for the immediate surgical management of such a patient. Is suboptimal care inevitable for these children?

In a study of 146 children with severe head injury conducted by Bruce,[33] only 26 children (18 percent) had CT evidence of a mass lesion for which a definitive neurosurgical procedure was a consideration. (Six epidural hematomas, 11 subdurmal hematomas, and eight intracerbral hematomas). Several of these were managed nonsurgically, and several of those that were treated surgically did not require emergency surgery. In another study by Bruce the most common CT finding in the pediatric patient with severe head injury was bilateral, diffuse cerebral swelling.[34] The treatment for this group, and for many other children with identified mass lesions, consisted of control of intracranial pressure primarily with hyperventilation and, in refractory cases, with osmotic agents.

In summary, a large majority of pediatric head injured patients will not require immediate neurosurgery. Instead, optimal initial management of these infants and children consists of controlled intubation, hyperventilation, and fluid restriction unless shock is a complicating factor. Therefore, an emergency department that manages severely head-injured pediatric patients should develop a protocol that includes the following components for any child with a marked alteration in level of consciousness or a deteriorating level of consciousness:

1. Controlled intubation using paralyzation, sedation, and lidocaine to prevent exacerbation of increased intracranial pressure during the procedure
2. Hyperventilation to a PCO_2 of less than 25 using continuously monitored exhaled PCO_2 to control ventilator settings
3. Fluid therapy with controlled boluses of Ringer's lactate only as needed to treat shock (IVs should be placed at KVO or to heparin seal at other times.)
4. Rapid transport after stabilization to an appropriate tertiary care facility with pediatric neurosurgery, pediatric anesthesiology, and pediatric intensive care capabilities.

SUMMARY

Critically ill infants and children require a high level of specialized pediatric medical and surgical resources for their emergency care. Because of the smaller number of pediatric emergency cases, however, these special-

ized pediatric resources are less readily available than similar resources for critically ill adult patients. Emergency departments that must manage an occasional critically ill pediatric patient with limited resources must recognize potential problem areas and devise appropriate strategies for these situations. Steps need to be taken to maximize the availability of limited specialized pediatric resources. Protocols that, in turn, maximize the effectiveness of these resources should be developed and in-serviced frequently to compensate for limited clinical experience. If a given emergency department in a region possesses clearly superior resources for the management of critically ill/injured children then other emergency departments should cooperate with the development of a regional EMS plan for the direct transport of the most critically ill/injured children to this emergency facility. Transfer agreements between community emergency departments and tertiary pediatric centers should be developed so that transfer is expedited. Finally, interhospital pediatric transport systems need to be developed, preferably as separate systems but as subsystems of larger systems if necessary, in order to ensure rapid, safe transport of the critically ill pediatric patient to a tertiary pediatric center, when necessary.

REFERENCES

1. Fleisher GR, Ludwig S: Textbook of Pediatric Emergency Medicine. Williams & Wilkins, Baltimore, 1982
2. Smith IM: Incidence of intussusception and congenital hypertrophic pyloric stenosis in Edinburgh children. Br Med J 1:551, 1960
3. Accrediation Manual for Hospitals: Joint Commission on Accrediation of Hospitals, 1986
4. Simon JE, Smookler S, Guy B: A regionalized approach to pediatric emergency care. Pediatr Clin North Am 28:677, 1981
5. Guidelines for Air and Ground Transportation of Pediatric Patients. Committee on Hospital Care. American Association of Pediatrics. Pediatrics 78:943, 1986
6. Ludwig S: Pediatric Emergencies: Churchill-Livingstone, New York, 1985
7. Davis HW, Gartner JC, Galvis AG, et al: Acute upper airway obstruction: Croup and epiglottitis. Pediatr Clin North Am 28:859, 1981
8. Lewis JK, et al: A protocol for the management of acute epiglottitis. Clin Pediatr 17:494, 1978
9. Fried MP: Controversies in the management of supraglottitis and croup. Pediatr Clin North Am 26:931, 1979
10. Glicklich M, Cohen RD, Jona JZ: Steroids and bag and mask ventilation in the treatment of acute epiglottitis. J Pediatr Surg 14:247, 1979
11. Hannallah R, Roseales JK: Acute epiglottitis: Current management and review. Can Anaesth Soc J 25:84, 1978
12. Adair JC, Ring WH: Acute epiglottitis. Anesth Analg 54:622, 1975
13. Costigan DC, Newth CJL: Respiratory status of children with epiglottitis with and without an artificial airway. Am J Dis Child 132:139, 1983
14. Welsch KJ, et al: Pediatric Surgery. 4th Ed. Year Book Medical Publishers, Chicago, 1986
15. Ein SH, Stephens CA: Intussusception: 354 cases in 10 years. J Pediatr Surg 6:16, 1971

16. Tenenbein M, Wiseman NE: Early coma in intussusception: Endogenous opioid induced? Pediatr Emerg Care 3:22, 1987
17. Ficher JK, Germann DR: Glucagon-aided reduction of intussusception. Radiology 122:197, 1977
18. Hoy GR, Dunbar D, Boles T: The use of glucagon in the diagnosis and management of ileocolic intussusception. J Pediatr Surg 12:939, 1977
19. Haase GM, Boles T: Glucagon in experimental intussusception. J Pediatr Surg 14:664, 1979
20. Waskerwitz S, Berkelhamer JE: Outpatient bacteremia: Clinical findings in children under 2 years with initial temperatures of 39.5°C or higher. J Pediatr 99:231, 1981
21. McCarthy PL, et al: Bacteremia in children: An outpatient clinical review. Pediatrics 57:861, 1976
22. Teele DW, et al: Bacteremia in febrile children under 2 years of age: Results of blood cultures of 600 consecutive febrile children seen in a "walk-in" clinic. J Pediatr 87:227, 1975
23. Carroll WL, et al: Treatment of occult bacteremia: A prospective randomized clinical trial. Pediatrics 72:608, 1983
24. Myers MG, et al: Complications of occult pneumococcal bacteremia in children. J Pediatr 84:656, 1974
25. Bratton L, Teele DW, Klein JO: Outcome of unsuspected pneumococcemia in children not initially admitted to the hospital. J Pediatr 90:703, 1977
26. La Scalea LJ, et al: Advantages of BACTEC hypertonic culture medium for detection of H. Influenza Bacteremia in children. J Clin Micro 17:1177, 1983
27. Kanter RK, Zimmerman JJ, Strauss RH, Stoeckel KA: Pediatric emergency venous access. Am J Dis Child 140:132, 1986
28. Iserson KV, Criss E: Intraosseous infusions: A usable technique. Am J Emerg Med 4:540, 1986
29. Rosetti VA, Thompson BM, Miller J, et al: Intraosseous infusion: An alternative route of pediatric intravascular access. Ann Emerg Med 14:855, 1985
30. Berg RA: Emergency infusion of catecholamines into bone marrow. Am J Dis Child, 138:810, 1984
31. Mayer T, Walker ML, Johnson DC, et al: Causes of morbidity and mortality in severe pediatric trauma. JAMA 245:719, 1981
32. Ramenofsky M, Morse TS: Standards of care for the critically injured pediatric patient. J Trauma 22:921, 1982
33. Bruce DA, Gennarelli TA, Langfitt TW: Resuscitation from coma due to head injury. Crit Care Med 6:254, 1978
34. Bruce DA, Alavi A, Bilanink L, et al: Diffuse cerebral swelling following head injury in children: The syndrome of malignant brain edema. J Neurosurg 54:170, 1981

4 Medical-Legal Aspects

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It is no longer possible to practice medicine in a pediatric emergency department without serious consideration of the medical-legal risks involved in this practice. While a pediatrician in private practice is not likely to be part of a lawsuit, it must be remembered that physicians in the emergency department are the focus of a disproportionate number of such suits. One study showed that 16 percent of all hospital lawsuits involved the emergency department.[1] About one-half to two-thirds of these suits resulted from misdiagnosis.[1,2] The most common error made by physicians in the emergency department is a grossly deficient examination related to the chief complaint.[1] Another important error that results in lawsuits is failure to order appropriate diagnostic studies.[1] Specific diagnoses are particularly troublesome and are often the subject of malpractice suits. For instance, failure to diagnose meningitis or appendicitis are two leading pitfalls for those treating children in the pediatric emergency department. This is not surprising because such conditions can be fatal or can result in serious morbidity if they are not diagnosed and treated promptly. However, other errors such as missed fractures or failure to find a foreign body in a wound do not usually result in serious morbidity, yet they too are major causes of malpractice suits.[1] Most likely this is because the public expects physicians to make such diagnoses, regardless of the outcome.

LITIGATION IN THE EMERGENCY DEPARTMENT: ASSOCIATED FACTORS

Many experts believe that the recent explosion in malpractice suits against physicians is because patients are aware of medical advances, and therefore, expect physicians to always be correct. In the emergency department, there are other reasons why lawsuits evolve so frequently.[3] For instance, most pediatric emergency departments have an impersonal registration system, unlike the warm atmosphere of a private pediatrician's office. Parents are quickly told to fill out forms, sit in one location, and then move to another waiting area. Privacy is often limited, and an overworked clerical staff is likely to be abrupt or discourteous at times. Moreover, patients usually endure a few hours of waiting in a dull seating area before they can see the physician. The actual visit with the doctor is often very brief as compared with the time spent waiting, and the encounter may seem impersonal when an overwhelmed physician hurries off to see the next child. It is difficult to establish good rapport with a patient who is not previously known to the emergency physician, and it is unlikely that the emergency physician will be as well informed about the child as the private pediatrician who has followed the patient for years. All these factors can make a family angry, and this anger is likely to be directed at the emergency physician if the ill child does not have a good outcome. Anger is a major force in the initiation of a lawsuit.

Other hazards confront the emergency physician as well. These doctors have no control over which patients come to the facility, and thus many patients or parents are under the influence of drugs or alcohol when they are interviewed. Some are abusive or uncooperative, providing an incomplete or unreliable history. Also, such patients may provoke negative feelings from the physicians or nursing staff, which could affect their judgment or decision making. Furthermore, the emergency department staff usually works in an environment of excessive noise, high pressure, high volume, and stress. The staff often endures long hours that can affect judgment and record keeping. Despite this, they must treat critically ill children with rapid decisions that have broad consequences.[3] The chances for error are understandably high. Factors in the emergency department that can lead to a malpractice suit against an emergency physician are listed below (Table 4-1).

Table 4-1. Factors in the ED Responsible for Malpractice Actions

Long waiting times
Impersonal atmosphere
Relatively brief visit with physician
Difficulty establishing rapport
High volume of patients
Long hours for staff
Excessive noise level
Critically ill patients treated

Most lawsuits against physicians are settled out of court or are dropped completely. Only about 10 percent actually reach a jury verdict.[3] However, this should not be terribly reassuring to the emergency physician. The strain of the lawsuit is still burdensome. Also, the number of jury awards that exceed $1 million has increased dramatically in recent years. Physicians in the pediatric emergency department must remember that they can be sued for just about any cause.[3] Physicians can face civil charges or sometimes criminal prosecution for their errors. They can be held accountable if they make a mistake or if others working with them (i.e., nurses, respiratory therapists) act incorrectly. They can be liable for not doing enough for a patient, for doing something incorrectly, or for doing too much for a patient against the patient's or guardian's wishes. The physician can be accountable for releasing too much information about a child or for failing to report information about the child when indicated. The physician is responsible for the outcome of the patient as well as the child's family or other contacts who might suffer from a missed diagnosis. The physician may be responsible for faulty equipment used in the emergency department or perhaps for faulty medical records prepared by other physicians.

Needless to say, treating children in the emergency department is fraught with risks. Even experienced pediatricians and emergency physicians are likely to be sued. Only about one-third of legal actions involve inexperienced or unsupervised house staff and moonlighters.[1] Studies have also shown that weekends, holidays, evenings, and nights are the most hazardous times to practice in the emergency department. Such time periods account for more than 80 percent of malpractice actions.[4] This may be due to chronic understaffing at these times.

There is no way to eliminate completely all legal actions against the emergency department. Mistakes and poor outcomes are inevitable. However, knowledge about certain risk-management issues may limit the number and expense of some lawsuits. This chapter focuses on some of these important risk-management issues.

THE MEDICAL RECORD

Risk management attempts to identify areas in which lawsuits frequently occur and to take steps to avoid future problems and thus future litigation. Perhaps the most important risk-management issue is that of good medical records in the emergency department. A well-designed chart can remind the emergency physician to examine the child completely or to pay particular attention to details in the history.[3] It is important to note that litigation that involves children may not take place for several years after treatment because of the extended statute of limitations. Thus, it may be impossible for a physician to recall what was said or done for a particular child. The physician is forced to rely entirely on what is written in the medical record. This document can prove to be a friend or a foe to the physician, depending on how well it was prepared at the time of treatment.

Each chart should therefore be prepared as if it were to serve as the basis for defense of a lawsuit, because one can never predict which case will end up in litigation. In general, information that is not legibly written on the chart will be difficult to justify in the future. Insurance companies have paid millions of dollars annually in injury awards and out-of-court settlements for cases in which good medical care was provided but not substantiated in the medical records.[5]

The Joint Commission on Accreditation of Hospitals (JCAH) has several requirements for adequate charting in the emergency department. If physicians strictly followed their criteria, the medical record would serve them well. One such requirement is that the patient be completely and accurately identified on the records. Each page of the chart should clearly display the patient's name and record number. Also, the time the child arrived in the emergency department and the means of the patient's arrival must be included. For instance, it is important to note whether the child was brought by ambulance or in the parent's car, as this may suggest the seriousness of the complaint in some cases. Any prehospital care received by the child should also be recorded in detail. Certainly, the history of the present illness must be described completely but concisely. This is often a stumbling block for physicians in the emergency department because the excessive volume of patients limits their time for recording information. While physicians should not write a lengthy novel for each patient, they must record any information that seems important to the chief complaints. A few extra moments to record details about the child's recent diet or activity will not lead to a severe delay in patient flow. Instead it could save the physician from many hours in court and will thus be time well spent. A complete physical examination must be recorded for each child. This should include a complete set of vital signs to be recorded repeatedly when appropriate. Also, any diagnostic or therapeutic orders must be clearly written. They should include the exact time the order was written, since minutes or hours are important in the management of a sick child. If such orders are not carried out promptly, the child may suffer grave consequences, and the records must reflect this timely process. The emergency physician must also record clinical observations of the patient. Moreover, any laboratory test results or reports of procedures performed must be documented. This often presents a problem for the emergency department staff, since some laboratory test results do not become available until long after the patient has left the emergency department. Still, the physician is responsible for recording this information on the chart, as it may have a bearing on the child's outcome. The medical record must also include the diagnostic impression formulated by the doctor, as well as the final disposition of the patient. Any follow-up instructions given must be recorded in detail. These instructions are best put in writing for the patient, but the physician should also go over them verbally and see that the family understands. It is helpful to have the parents sign a statement indicating that they understand the instructions given. Finally, if the patient leaves against medical advice, this must be

Table 4-2. JCAH Requirements for Charting in the ED

Patient identification
Time and means of arrival
Emergency care prior to arrival
History of illness
Physical findings, vital signs
Diagnostic and therapeutic orders
Clinical observations
Reports of procedures/tests
Diagnostic impression
Condition on discharge
Final disposition
Leaving AMA

clearly documented, according to JCAH. Table 4-2 summarizes the JCAH requirements for charting in the emergency department.

In addition to the JCAH requirements, charts in the emergency department can be improved by adhering to several suggestions. First, the record should display a concerned professional attitude toward the child and family.[6] While some emergency department charts may use a checklist system in an effort to save time, physicians are reminded to add details of important positive or negative findings on the physical examination. A chart with the entire examination merely checked off as normal will not impress a jury that the physician examined the child carefully. In addition, it is important to document any consultants called in on a particular case, as well as when they were called. Not only does this demonstrate concern for the child's welfare and the desire to provide proper treatment, it also shares the responsibility of a poor outcome. Similarly, if a child is transferred to the care of another emergency physician (i.e., when one doctor completes a shift), the time of this transferred care should be noted in the chart.

If the chief complaint is an injury, the medical record should reflect how the injury occurred and whether the history is consistent with the physical examination.[7] It may be best to use a picture or diagram to describe the trauma in detail. It is also important to list any previous injuries the child may have suffered. The physician should note any concerns of child abuse and should record whether the case was reported to local authorities as such.

Furthermore, the chart should not contain any insensitive terms, such as "F.L.K." or "gomer." Such terms used to describe "funny-looking kids" or undesirable patients will undoubtedly show that the physician is uncaring or unsympathetic, should this chart be reviewed for litigation. Likewise, one should avoid such unnecessary terms as "inadvertently" or "by error."[8] If a medication was given erroneously, this must be documented in the chart, but there is no need to advertise the error. Also, it is important that the staff (i.e., nurses, consultants, physicians) not engage in battles on the record.[6] Blatantly expressing disagreements in the chart will not serve anyone well

Table 4-3. Recommendations for Charting in the Emergency Department

Demonstration of a concerned, professional opinion
Thought process at examination
Specific time of orders, procedures
Consultants called
Instructions given
Injuries with diagrams

Table 4-4. Items to Avoid in the Emergency Department Chart

Blacking out or erasures
Engaging in "battles" on paper
Use of insensitive terms (i.e., F.L.K.)
Use of unnecessary terms (i.e., inadvertently)
Alteration of the chart after completion

if the case comes to litigation. Instead, the chart should reflect agreement among all caretakers whenever possible, and inflammatory remarks should not be written down.

Finally, any errors in the chart should be corrected appropriately. Physicians understandably make mistakes while writing in the chart. However, they should not attempt to cover up these mistakes by blacking out words or phrases. This tends to arouse suspicion. Instead, a single line through the error, which is then initialed and dated, is a better way to correct a mistake.[6] Similarly, one should never attempt to add or subtract from the record after the case is involved in litigation, as this will undoubtedly seem a deliberate and dishonest cover-up. Obviously, the chart should never be altered once the patient's attorney has a copy of it. However, one never knows exactly when the chart has been requested for review. Thus, it is best never to alter a chart once it leaves the emergency department. Such tampering can result in loss of the lawsuit, the physician's license, as well as other criminal charges.[9] Tables 4-3 and 4-4 summarize these additional recommendations for charting in the ED.

CONSENT FOR TREATMENT

Another troublesome area for physicians caring for children in the emergency department is the issue of consent for treatment. Young children are often brought to the emergency department by siblings, extended family members, day care workers, camp counselors, school teachers, or babysitters. Many temporary custodians to not have authority to give consent for medical care. Even written documentation of authority does not always

substitute for verbal consent from parents.[10] The physician must determine whether immediate treatment is indicated before parental consent can be obtained. This presents an additional challenge to the complicated task of managing an ill or injured child. In general, treatment without consent of parents is allowed in order to prevent serious injury or death.[11] The greater the risk of harm to the child, the more justified is treatment without consent of the legal guardian. What is considered a true emergency such that parental consent is not needed varies from state to state. Almost all states recognize severe bleeding or respiratory difficulty as emergencies requiring immediate care. Some indicate that any condition that presents a threat to "life and limb" or "life and health" constitutes an emergency.[12] Still other states consider that any painful condition should be promptly treated, even without parental consent.[12] Most have such vague statutes concerning this issue that the well-meaning physician is protected. Certainly unnecessary or elective procedures are unjustified without the parent's permission, but the physician should not withhold treatment that is rational and life-saving. In some cases, a guardian could bring suit against a hospital for delivering unauthorized care. This, however, is usually less disastrous and less costly than withholding care when it is needed immediately. Moreover, physicians should not appear more concerned about potential litigation than about the health of the child, so reasonable care should not be delayed in an emergency situation.[11]

Hospital staff should make every effort to contact the guardian as soon as the child arrives in the emergency department, even in a true emergency. Such attempts should be documented in the medical record.[10] Parents should be requested to come to the emergency department, but if this is not possible, consent for treatment may be given over the telephone. In such cases, a nurse, clerk, or physician should serve as a witness for the consent by listening on another phone extension.[11] If consent cannot be obtained prior to treatment in an emergency, it is still beneficial to attempt to reach the parents afterward. This will maintain rapport with the family and help with discharge instructions and follow-up care.[6] If parents cannot be found despite valiant efforts, a close relative should be contacted.[10]

If parents are legally separated or divorced, it is important to obtain consent from the custodial parent when this can be determined.[11] If the situation is clearly not an emergency, it is best to withhold treatment until the custodial parent can be reached. In general, the courts hold that parents have the right to authorize treatment for their young children, and physicians can be sued for violating this right.[12] Again, if the situation is not clear, it is better to err on the side of treatment or to contact a hospital lawyer for help.

Adolescents who present for care without their parents provide an additional challenge for the emergency physician. Many of these patients do not want their parents informed that they are at the hospital. Most states allow physicians to treat these patients if they are "emancipated minors." An emancipated minor is one who has been married or is pregnant, who has

Table 4-5. Examples of Emancipated Minors

High school graduate
Pregnant (currently, previously)
Married (currently, previously)
Self-employed
Living independently
Served in armed forces

been graduated from high school, or who lives independently of parents.[6] In some states, minors who are self-employed or self-sufficient or who have served in the armed forces are also considered emancipated even if they live at home. These criteria also apply to adolescents who run away from home or are otherwise alienated from their parents, regardless of the parents' feelings about the child's "emancipation."[13] Some states specify a minimum age at which these laws apply, which can vary from age 14 to 18 years. In some states, a minor is still emancipated after a marriage dissolves, or after a pregnancy ends, but others require parental consent for medical treatment after a teenage pregnancy terminates.[12] Table 4-5 lists examples of emancipated minors.

Even if an adolescent does not meet the criteria for an emancipated minor, some states allow medical care to be provided to those over 15 years of age under the "mature minor doctrine." This says that treatment for the benefit of a minor may be given if the minor is "sufficiently mature to understand the nature of the procedure and its consequences."[12,13] This doctrine has been used to allow a teenager to receive a smallpox vaccine voluntarily or to allow a finger laceration to be repaired without parental consent.[11,12]

Finally, most states allow adolescents to receive medical care for problems that might lead to a delay in care if parental consent were required. For example, pregnancy-related problems, contraception issues, or sexually transmitted diseases may be treated without parental consent.[10] Likewise, teenagers can seek treatment for drug- and alcohol-related problems without notifying their parents.[13] In some states, the child must be older than 12 years of age before treatment is allowed, but other states have no age requirement. It is believed that if parental consent were needed for treatment of these problems, treatment would be avoided and the child as well as society may suffer. Table 4-6 lists certain medical problems that may be treated in the emergency department without parental consent.

While the law allows the emergency physician to deliver care to children without parental knowledge, it is best to discuss in depth with the adolescent the medical and ethical problems that result from such care. It should be noted that if information about drug or alcohol use is obtained from a teenager, the physician has the legal right to convey this information to a parent against the wishes of the minor. However, the physician may elect not to do so if such action is likely to interfere with treatment. Other-

Table 4-6. Medical Problems That May Be Treated in the Emergency Department Without Parental Consent

Any true emergency
Pregnancy
Sexually transmitted disease
Drug or alcohol abuse
Contraception issues

wise, the physician and social workers in the emergency department should point out the benefits of discussing medical problems with the parents, and the teenager should be told that a hospital bill may be sent to the legal guardian. The parent may refuse such a bill, leaving the adolescent or hospital to assume the costs of treatment.[13] The parent may then demand an explanation from the child or the physician about the bill. However, if the adolescent insists on privacy, this should be respected.

LEAVING THE EMERGENCY DEPARTMENT AGAINST MEDICAL ADVICE

In some situations, a legal guardian may be present but may refuse to give consent for treatment or may wish to leave the emergency department against medical advice. Likewise, an emancipated minor may also refuse a certain procedure or treatment. An informed rational parent or patient has the right to seek alternative care in many cases.[14] This desire to leave the emergency department may occur when patients or parents are angry about the way they have been spoken to or treated thus far in the emergency department. They may then direct their anger toward the physician and refuse a painful procedure.[15] Other patients or parents wish to leave against medical advice because they are afraid of what is likely to happen to them or their children.[15] Parents may have uncertainty or fear of a serious diagnosis. They may have fears regarding previous hospital experiences. They may fear that young physicians are "practicing" or "experimenting" with their children. Some may have heard misinformation from others and may therefore be afraid of a procedure such as a spinal tap. Or, they may be concerned about other children at home and how their lives will be disrupted by admission to the hospital. Those who do not understand English will be particularly frightened. Such fears will frequently cause a teenager or parent to wish to leave the emergency department or to seek a second opinion. Still others will refuse a procedure such as a blood transfusion on religious grounds.

Such patients are a significant headache for the emergency physician. It is certainly easy simply to let the patient or parents leave the emergency

department and go elsewhere for care. In some cases, an angry staff member may actually invite the family to go elsewhere, especially if the parents have been abusive. However, it should be noted that obscene language by the patient or parent does not justify improper release of the child from the hospital.[16] Also, no one is likely to benefit when a patient leaves against medical advice. The emergency department staff always feels a sense of frustration or failure when their recommendations are ignored. Moreover, the hospital and the emergency physicians may encounter a lawsuit if the patient suffers after leaving the emergency department without proper treatment, even if he/she left voluntarily. Most importantly, the child may continue to suffer from persistent or worsening symptoms that have not been treated. Thus, the emergency physician must make every effort to convince them to stay in the facility for a complete evaluation and treatment.[14] This requires a great deal of sensitivity, compassion, and diplomacy, as well as awareness of the law.

The emergency physician should first try to determine why the parent or patient wishes to leave the emergency department. If they are angry or afraid, they must be allowed to express their concerns without interruption. The physician should remain courteous, humble, and flexible, if possible.[17] There is no advantage to threatening patients or agitating them further with security guards, unless they are absolutely needed to maintain order. Certainly, the staff should not direct anger at the family or challenge them to sign out against medical advice. Instead, additional family members, a familiar private doctor, a trusted friend or nurse, or a religious leader should be contacted to help calm the family and ease their fears.[15] A competent translator should be found if a language barrier exists. Certainly a staff member who made the family angry should apologize or avoid further contact with them.[11] If no progress can be made with the above measures, it may be helpful to have a social worker speak to the family or let another physician in the emergency department try to establish a better rapport with them.

In all cases, the physician must spend a considerable amount of time to ensure that the patient or parents understand all the risks and benefits of the recommended treatment and of alternative treatments. They must understand the risks involved in refusing this treatment. They should be given time to reflect on this information in a low-pressure atmosphere, so that they can reach a rational decision.[15]

Despite valiant efforts, it is sometimes impossible to convince parents or teenagers to accept the recommendations of the emergency department staff. If a procedure is refused, such as a spinal tap, the physician may decide to manage the child without benefit of the procedure. For example, the child can be admitted to the hospital for antibiotics with the assumption that meningitis exists.

If the parent or teenager insists on leaving the emergency department, they should be asked to sign a statement that releases the emergency department staff and hospital from all liability. This "against medical advice

form" may shift some responsibility for a poor outcome to the parents but does not entirely limit the physician's liability,[6,15] as parents can always claim later they did not completely understand the doctor's recommendations.

On the medical record, the physician should record any limited history or physical examination that was obtained as well as the clinical impression. The parents' reasons for leaving should be documented, as well as the risks and benefits of treatment as it was explained to the parents. The parents should be told, and it should be documented, that they can return to the emergency department at any time for further treatment.[15] If the parents refuse to sign this release form, this must also be documented. Another staff member should sign the forms and chart bearing witness to the events that took place.[4] The items which must be documented for patients who leave the emergency room against medical advice are listed in Table 4-7.

In some circumstances, a patient cannot be allowed to leave the emergency department regardless of personal or parental wishes. In a true medical emergency, in which additional time or the act of transport would jeopardize the life of the child, immediate medical care is justified regardless of the parent's wishes.[6,18] It is still best to win their cooperation but, if they refuse, the child should be treated and the case reported as child neglect.

If a blood transfusion is refused on religious grounds, court permission can be obtained in life-threatening situations. Most courts will override religious beliefs on behalf of minors if life or health is in danger. Likewise, if child abuse is suspected, the patient cannot be released from the emergency department unless the home is thought to be safe. Regardless of the parents' request, if the perpetrator is not known and the emergency physician is concerned about the child's safety, security guards should be called to prevent the parents from taking the child. The hospital lawyers or administrators or a judge on emergency call should be contacted to obtain a temporary restraining order. The conversation with the judge should be recorded on the chart, and written permission from the judge should be forthcoming within the next 48 hours.[14]

Finally, if a teenager or guardian of a young child is under the influence of drugs or alcohol or is mentally impaired, it is not possible for them to

Table 4-7. Required Documentation for Patients Who Leave Against Medical Advice

Risks/benefits of treatment
Risks/benefits of alternatives
History and physical examination
Impression
Reasons for leaving emergency department
Invitation to return to emergency department
Signatures: parents, physician, witness

Table 4-8. Conditions Where Leaving Against Medical Advice Is Not Permitted

Life-threatening emergency
Suspected child abuse
Incoherent parents/patient

understand the risks and benefits of treatment. Thus, they must be restrained, if needed, to allow treatment to be given. It is best to assume that a patient's bizarre behavior is part of a medical illness until proved otherwise. The physician may attempt to gain their cooperation, but it is pointless to engage in lengthy conversation with a disoriented patient or parent. A potentially violent patient should be interviewed in a room with an open door. The hospital staff may use reasonable force to keep patients from harming themselves or others.[17] In fact, the staff is liable if the patient leaves the emergency department and suffers or hurts someone else.[4] While a lawsuit for battery could result from physical restraint in a situation in which a patient is erroneously thought to be incoherent, the risks of allowing the patient to leave are still greater. Conditions in which patients cannot leave the emergency department are summarized in Table 4-8.

TELEPHONE ADVICE

The last medical-legal issue to be discussed here involves the dangers of giving advice to patients or parents over the telephone in the pediatric emergency department. While pediatricians in private practice encourage their patients to call for advice, this invitation should be discouraged in the emergency department. Parents may prefer to call the emergency physician for advice about their child because it is a convenient way to receive care. Certainly, parents would like to avoid bundling up an ill child and waiting for public transportation to take them to the hospital.[19] This is especially true when they have other children at home to care for as well. Moreover, it is less expensive to receive care by telephone, because transportation costs are avoided and usually no hospital bill is generated.

While physicians and hospitals must be sympathetic to the needs of families that have no other source of care, it remains unwise for the emergency physician to give medical advice over the phone. Certainly, many trivial complaints could be managed in this way, but some serious illnesses can mimic an apparently trivial complaint. For instance, the vague symptoms of meningitis cannot be easily distinguished from a simple gastrointestinal virus over the phone.

It is often impossible to deliver good medical care by telephone. Certainly, it is difficult to get a complete history by phone. Emergency physicians are often distracted by the noise and sick patients around them, hence they may not devote full attention to the caller. Likewise, parents may

Table 4-9. Disadvantages of Giving Telephone Advice in the Emergency Department

Complete history is unlikely.
Physical examination is impossible.
Documentation is difficult.
Instructions are misunderstood.
Other ill patients in the emergency department are distracting.
Remuneration is rare.

forget to relate important historic facts, and physicians may neglect to ask certain questions if the child is not present in front of them.[19] For instance, a parent who is most concerned about symptoms of fever or vomiting may neglect to describe a rash that seems insignificant. The physician may neglect to ask about this specifically without seeing the rash. Several authorities have recommended that certain screening questions can determine the severity of a child's illness.[20] However, these questions are often misleading or unreliable.

Moreover, it is obviously impossible to perform a physical examination over the telephone. Trying to distinguish croup from epiglottitis by listening to the child breathe into the phone receiver is not a rational approach. Certainly parents cannot be expected to distinguish a petechial rash from a viral exanthem or a benign pharyngitis from a peritonsillar abscess. Diagnosis of a rigid abdomen or an abnormal chest examination likewise requires the skill of a well-trained physician.

Thus, it seems extremely hazardous to give advice about treatment of a child with an incomplete history and without a physical examination. Furthermore, it is difficult to document these conversations adequately, so it would be impossible to defend the case should a poor outcome result. Undoubtedly, the physician stands a better chance in court if a full evaluation including vital signs is carefully recorded in an organized manner. Finally, emergency physicians who do not know the caller cannot be confident that their advice has been understood. Follow-up evaluation of these patients is difficult when there are so many other sick patients in the emergency department to care for.

Consequently, telephone advice should be limited. It may be given in an extreme emergency, such as an accidental poisoning.[8] These patients should always be told to come to the emergency department immediately after initial measures are taken at home. They should be advised to call for emergency transport if this seems indicated, or the hospital should arrange this. Such a conversation should be documented if possible.[19] Likewise, telephone advice may be given to a patient who just left the facility and who needs further clarification of instructions. An additional entry should be made in this patient's chart. The child should be brought back to the emergency department if the phone call does not easily clarify the situation.[19]

Otherwise, patients and parents who call should be told: "I'm sorry, I

cannot give you any medical advice over the telephone. I would be happy to evaluate your child if you bring your child to the hospital."[19] This should be said in a caring, polite way, as this policy is really in the best interests of all involved. If this statement is given consistently, the phone conversations do not necessarily require documentation. Physicians should caution nurses and other staff members against supplying further advice to patients over the phone. Such persons usually have less training than the physician and could provide wrong or misleading information. Table 4-9 reviews disadvantages to giving medical advice over the telephone.

SUMMARY

The practice of pediatric emergency medicine has numerous legal hazards. Some of these are unavoidable, and anyone who cares for children in the emergency department is at risk for litigation. However, if the emergency physician is aware of some important risk-management issues, the number and cost of some lawsuits may be significantly reduced.

REFERENCES

1. Trautlein JJ, Lambert RL, Miller J: Malpractice in the emergency department—Review of 200 cases. Ann Emerg Med 13:709, 1984
2. Ricci JA, Lambert RL, Steffes DG: Pediatrics and professional liability. Pediatr Emerg Care 2:106, 1986
3. Korin J, Selbst SM, Torrey S: Medicolegal issues in pediatric emergency medicine. Pediatr Emerg Care 1:48, 1985
4. Rogers JT: Risk Management in Emergency Medicine. Emergency Medical Foundation, American College of Emergency Physicians, Dallas, 1985
5. George JE, Quattrone MS: The E.D. record: Legal implications. Emerg Physician Legal Bull 10:2, 1984
6. Bergen CL: Legal aspects of emergency department pediatrics. p. 1201. In Fleisher G, Ludwig S (eds): Textbook of Pediatric Emergency Medicine. Williams & Wilkins, Baltimore, 1983
7. Solomons G: Trauma and child abuse—The importance of the medical record. Am J Dis Child 134:503, 1980
8. Vaccarine JE: Medicolegal considerations in emergency care. p. 734. In Wilkins EW (ed): MGH Textbook of Emergency Medicine. Williams & Wilkins, Baltimore, 1978
9. Korin JB: Altering records now hazardous to your license. Emerg Physician Legal Bull 5:1982
10. Selbst SM: Treating minors without their parents. Pediatr Emerg Care 1:168, 1985
11. George JE: Consent to treatment. p. 35, In Law and Emergency Care. CV Mosby, St. Louis, 1980
12. Brown RH, Truitt RB: The right of minors to medical treatment. DePaul Law Rev 28:289, 1979

13. Hoffman AD: Legal issues in child health care. p. 521. In Hockelman RA (ed): Principals of Pediatrics. Health Care of the Young. McGraw-Hill, New York, 1978
14. Selbst SM: Leaving against medical advice. Pediatr Emerg Care 2:266, 1986
15. Purdie FRJ, Honigman B, Rosen P, et al: Prudent handling of patients signing out against medical advice. ER Rep 3:73, 1982
16. Johnson R, Trimble C: The (expletive depleted) shouter. J Am Coll Emerg Physicians 4:333, 1975.
17. Levy RL, Gale MS: Interactional approach to the difficult emergency department patient. J Am Coll Emerg Physicians 6:17, 1977
18. Gouge RL: Responsibilities and liabilities for emergency care service. J Am Med Wom Assoc 33:423 1978
19. Selbst SM, Korin J: The telephone in pediatric emergency medicine. Pediatr Emerg Care 1:108, 1985
20. Brown JL: Telephone Medicine—A Practical Guide to Pediatric Telephone Advice. CV Mosby, St. Louis, 1980

5 Child Abuse: Causes and Solutions

Stephen Ludwig

Child abuse is a term used frequently in the emergency department. Since the awakening of the medical community to this problem in the mid-1960s, there has been an explosion of information concerning the recognition and reporting of abuse.[1–3] In addition, the many faces of abuse have been seen and reported in the medical literature.[4,5] Physicians have been educated concerning their medical, legal, and moral obligation to report suspected abuse. Several large monetary awards have been granted to plaintiffs who claimed that their physician's negligence in failing to report abuse resulted in more severe injury. In some emergency departments, abuse is such a frequently made diagnosis that physicians are instructed to consider every traumatic injury for its abuse potential.

Beyond a recognition of abuse, an acceptance of its pervasiveness in our society and a commitment to report suspected cases in a way prescribed by local guidelines, the emergency physician must also consider the root causes of abuse and the potential solutions to this serious health problem. The emergency physician often bears the greatest stress in making the diagnosis of abuse and initiating the report. Thus, the emergency physician should strive to understand and remediate the causes of abuse. Ludwig[6] stressed identification and reporting. This chapter discusses causes and solutions. Obviously, the solutions are not easy ones for if they were children would no longer be beaten, killed, raped, abandoned, or neglected as they are in ever-increasing numbers. But we should recall that at certain points in time,

solutions to poliomyelitis, tetanus, and smallpox did not seem feasible. As a profession and as a society, we must stop reading the newspaper headlines and simply shaking our heads in horror and disbelief. Action is required.

WHAT IS ABUSE?

One place to begin is to consider what is meant by the term *child abuse*. Often in the emergency department we use the term as if it were a specific diagnostic term, much like the diagnostic term *pneumonia*. However, child abuse is not a diagnosis, it is a category of disease more akin to the term *infectious disease*. Child abuse is a broad term indicating that injury has occurred to a child as a result of family dysfunction. The injury is the result of either actions on the part of the parents (or persons acting in the parental role) or inactions.

Thus, child abuse as a category of diseases has many subelements. The elements are frequently delineated further by indicating the kind of injury—physical, sexual, emotional, or neglect. Further delination may be indicated based on the parents' intent—premeditated, intentional/repetitive, intentional, overreactive, unintentional/ignorant, and act of omission. We need to understand abuse with greater depth of understanding. Without this depth of understanding, we will not be able ultimately to apply proper treatment. For example, we understand infectious diseases as a category of disease. Furthermore, we understand pneumonia as a pathophysiologic process in addition to the specific causative organisms (e.g., pneumococcal). By having all these levels of understanding, we know how to administer a specific treatment and how to provide more general supportive care. When it comes to child abuse, our knowledge remains at the most superficial level. We have not penetrated deeper levels of understanding nor any specific therapies. As physicians, we find satisfaction in applying treatments that cure a problem. The diagnosis of child abuse has brought little satisfaction thus far. We are in the preantibiotic era as far as the analogy.

For now, child abuse is a symptom of family dysfunction that produces injury in a child. This is a broad definition, but one I find practical in the setting of the emergency department. When facing a given case, I ask myself: (1) is there an injury? (2) is it demonstrable in a child? (3) is it the result of family (parental) dysfunction? If the answers to these questions are yes, I have diagnosed the category of disease we call child abuse. What caused it? and What can we do to treat it? remain unanswered questions.

CAUSES OF ABUSE

When the causes of abuse are identified, they can be broken into four categories indicating the source of the cause. The etiologic factors are societal, community-based, family based, or individual/personal. In each case of

child abuse, there may be one, two, or many causal factors. Some theorists would consider the cause of child abuse to reside solely in the personal category. They would claim that the responsibility for abuse rests in the hands of a weak person, a defective parent—the abuser. If this were true, we would be able to produce a profile of the abusive parent and have the ability to pick out the abusive parent from a group of "normals." Thus far, researchers have not been able to do so.[7] I believe this is because there is no abusive profile. A more cogent theory would indicate that any person placed under the correct set of life circumstances could abuse a child. With this construct, the entire responsibility for abuse does not rest with the individual alone, and we may examine other societal, community, and family-based factors.[8] The following sections explore each of the possible etiologies.

Societal Factors

It is clear that some societies have the tendency to produce more abuse than others. Cross-cultural studies show that some groups raise their children in a child-oriented fashion, while children in other societies grow up in a hostile, "grow up if you can" environment. For unknown reasons, child abuse seems to thrive in Western industrialized societies. Perhaps it is the value that such societies place on productivity and material goods that unknowingly devalues nonproducers in the society. Perhaps children are abused because they are takers rather than producers or because they have no inherent economic value as they once did in an agricultural society. They also have no political value.

In our own society, at least three factors are central to the problem of abuse. The first is a societal tolerance of violence in all forms. The Surgeon General of the United States, Dr. C. Everett Koop, stated that family violence was the number one health problem facing the United States during the remainder of this century.[9] With violence so much a part of societal practice, it is no wonder that some befalls children. In any urban emergency department, the effects of violence are dealt with on a continual basis. Even when we examine specific forms of violent injury, such as death due to handguns, we find children being swept into the mortality statistics.[10]

A second societal factor is our economic system. David Gil and others have indicated that with the extremes of our current economic system a large number of children are abused by virtue of being born into families of poverty.[11] Such children are not physically abused but economically abused. The societal economic system keeps them from being able to reach their potential. Surely this cause of abuse is a broad one, and changing our capitalistic system is for most people out of the question. Yet is it important to recognize that the system produces poverty, and the level of poverty that some experience is injurious to the health of their children.

A third societal factor is an acceptance of corporal punishment as the standard child discipline. Sweden has taken a societal position—"Don't hit your children." In the United States, you will often hear the position

stated—"Hit your kid once a week, whether he needs it or not." We have accepted corporal punishment as the first response when our child acts out rather than recognizing that it may on occasion be the last response. Corporal punishment is like societal drinking, just about everyone does it. Unfortunately, like drinking, some people go too far. This results in abuse. Many cases of abuse seen in the emergency department have had their beginning with a parent who tried to teach their child right from wrong. Corporal punishment is initiated and then escalated until severe harm is done to the child.

Community Factors

Several community factors also contribute to child abuse.[12] Perhaps the most important of these is social isolations. Social isolation is a fact of American community life-style. The belief that "A man's home is his castle" has been taken so literally that many American families are building walls and digging moats. Within our communities, there is little to bring people together. Thus, families are isolated. When stresses overwhelm them, there are few people around who know of a family's distress and even fewer who are willing to help. Most families that are having trouble coping are likely to find rejection from neighbors for not conforming to community standards rather than the help they need.

As a part of our social isolation, we isolate children with their parents for the first 3 years of life. We keep children at home and certainly discourage their presence in public places until they are civilized. There have been an increased number of preschool programs, but these are generally available for mothers who work or who go to school. Some mothers and fathers would benefit from sending their children to day care and preschool from the time of child's birth, even though they are neither working nor going to school. By the time the child has reached age 3, many of the problems of parenting have been solved or the child has already been abused.

The other community factor that must be considered is our human services delivery system for children with special needs. These children are often the victims of abuse. In our community, a mother recently shot and killed her 3-year-old retarded child because she saw no other way out. This is abuse by an individual, and the mother was charged with murder. It was also abuse by a community. For children who have sustained minor forms of abuse, the community also has a responsibility to provide services that will protect the child while preserving family continuity. This can only be done at great initial expense to a community. Failure to provide community-based family treatment results in separation of greater numbers of children from their families. The alternatives to family care are foster care and institutional care, which are themselves often damaging and more costly in the long term.

Familial Factors

The family unit has clearly been weakened during the twentieth century. There are more single parent families, more divorces, more alternative family units, and more families becoming less unified than ever before. The extended family that lives geographically close enough to share in common tasks is slowly disappearing. These factors are major contributors to abuse.[13] Child-rearing is a difficult, anxiety-filled, often unrewarding task. It becomes much easier when shared by two parents or by an entire extended family unit. Early in this century, the average family had at least three adult members, and often a grandparent, aunt, or uncle would be living in the house. Today the work of those three adults is being managed by one, and that one often is trying to work outside the home. The family as the basic child-rearing unit has been devalued, and no other social structure has moved in to fill the void.

Individual

There are still many individual factors that lead to abuse. Although the stresses may come from without, the control or loss of control still is centered in the parent. If the parent is weakened by other factors such as drugs or alcohol, abuse more likely occurs. Parenting is a demanding job, often confusing, often challenging, often numbing. If an adult has weakened self-esteem or is lacking self-assurance, parenting will bring out these weaknesses more strongly. Thus, the crying child will be interpreted as "he hates me," and retaliation will result.

Kempe and Helfer have put forth two theoretical models for child abuse.[2,12] Their first model indicates that the important elements of abuse are (1) a parent with the proclivity to abuse, (2) a child who is seen by the parent as different or special, and (3) a crisis that sets off an explosive abuse reaction between the already existing elements. Kemp and Helfer contend that the problem with abusive parents is that they have weak self-control, lack of knowledge of child development, unrealistic expectations, and a poor parental model from their own childhood. The second theoretical construct is called the "world of abnormal rearing." In this model, Kempe and Helfer show the cyclic nature of abuse: poor childhood experiences leading to poor self-image, poor peer relationships, poor mate selection, and ultimately poor parenting. Both models give us good examples of individual contributions to abuse.

Other individual causes of abuse have to do with parental psychopathology. Some parents are depressed or psychotic or have inadequate personalities. Other parents are psychopathic or sociopathic, for whom violence against children is part of a more pervasive violent approach to life. Retarded adults in some cases appear to be excellent parents, particularly for small infants and toddlers. However, some retarded parents are unable to

consider or meet the needs of their children. In all, these causes of abusive behavior account for only a small number of cases. Estimates indicate that in only 5 to 10 percent of child abuse cases can parental psychopathology be identified.

SOLUTIONS

Just as the causes of abuse can be broken into four problem levels, so are the solutions. Some of the solutions are global and cannot be accomplished by the emergency physician. The solutions stressed in this section are primarily those the emergency department physician can modify directly or support through professional or public action.

Societal Solutions

As well-respected members of society, we must advocate for change on a societal level. For many reasons, we must advocate for handgun control. This step alone would save the lives in many children and decrease the overall level of violence in our country. Our first goal should be the reduction of the child homicide rates that have increased sixfold since the 1930s.

In addition, we should advocate for the diminution of corporal punishment in the society. This can be accomplished by a national referendum condemning corporal punishment, as has been done in Sweden. This sets an ideal for parents to work toward and states clearly that as a society we are opposed to big people physically injuring little people. The abolishment of corporal punishment can also be attached in each emergency room waiting room as well as the supermarket and school. Whenever we see corporal punishment occurring in public, we can be sure that more and more severe injury is occurring at home. It is easy to walk away from this form of public display or to intellectualize it and extol the virtue of maintaining individual privacy. However, in walking away and not interceding, we are passively condoning abuse. Corporal punishment must be severely restricted or stopped. As physicians who work with children who are uncooperative, frightened, and overstressed, we must not fall into the trap of allowing the parent to be violent with the child in order to make our job easier. Thus, "Hold still for the doctor or I'll beat you" should be discouraged. In rare circumstances, the physician or nurse who is overworked and overstressed may lose control and do something to hurt a child. This must be sternly dealt with. There can never be an excuse for child abuse by a professional. Every emergency department policy and procedure manual should have a policy statement condemning this behavior and outlining a course of administrative action to be taken.

As far as the economic structure leading to abuse, it would be inappropriate for this paper to attempt to suggest any governmental reform or

global changes in our political system that would redistribute the wealth of society. However, physicians must never forget that poverty is a factor that causes ill health and that poverty must be modified in order to treat many medical conditions. For the purposes of emergency physicians, I would suggest a semiannual follow-up visit conducted at the home of one of your patients. Be aware how your patients live. This is something I have done over the past 10 years. It has given me an important perspective on societal effects on health and health care, made treatment plans more realistic, encouraged a more empathetic approach, and instilled a broader view of the physician's role.

Community Solutions

The emergency physician must have a knowledge of the community and community resources.[15] The emergency physician should not drive to work and drive home without knowing the community environment surrounding the emergency department. If the emergency physician does not have this awareness, it is incumbent on other staff members to bring such knowledge to the emergency department. The emergency department is one of the strongest community resources for social problems in addition to its medical role. It is a fact of our current life-style that people who have problems of any kind come to the emergency department. Social work services should be available 24 hr/day. This is particularly important for cases of child abuse. Social workers provide person-to-person services to the child and parents, but of equal importance are their link to community institutions and resources. Emergency services must include emergency social work services.

Other community solutions that can be encouraged and supported by the emergency physician include (1) promoting support for community-based child-protective services; (2) enhancing communication between child protective services, police, and the hospital; (3) encouraging the use of crisis nursing, day care centers, emergency babysitters for parents in crisis; (4) developing a child-abuse hot line and support groups for parents experiencing difficulty in parenting, and (5) providing increased services for special needs patients.

Family Solutions

The emergency physician needs to develop a view of child abuse as a symptom of family dysfunction.[16] Thus, in order to evaluate child abuse, one must evaluate the state of family. The emergency physician needs to view all children in terms of their place within the family and the family's protection and nurturing of them. This perspective is difficult to achieve. In its practical application, the emergency physician does not simply see and repair a laceration but sees a child and notes:

What was the cause of laceration?
Was this a preventable injury?
Was this an isolated injury?
Are there signs of inflicted injury or lack of adequate supervision?
Has the family been performing in other areas?
Is the child adequately nourished, clean, and clothed?
How are the parents treating the child?
Are they supportive, comforting, kind?

The family perspective also works in the other direction. For example, when seeing an adult patient with a drug or alcohol problem, evidence of violent behavior, or psychiatric emergencies, the emergency physician needs to inquire about other family members, especially children. Where are they? Who is taking care of them? Are they having any problems? Gaining this family perspective is crucial to identifying high-risk situations that can be helped before more serious symptoms and family malfunction occur.

Personal Solutions

Personal solutions can best be accomplished by the early effective reporting of abuse.[17] Many studies documented that fatal abuse is often preceded by more minor abuse manifestations. If these are recognized and reported, more severe injury may be averted. Reporting abuse and helping the abused child will help to break the cycle of abuse and avoid the world of abnormal rearing. All parents need help, not more social pressure, to have their children meet some hypothetical TV model. Children must also be considered individuals with their own set of human and legal rights.[18]

SUMMARY

Child case management is often difficult, as it is emotionally charged, unpleasant, tied to bureaucratic systems, and on occasion to a court appearance as a witness. All these factors make us wish that we did not have to deal with abuse. Abuse may never be eliminated completely. It is a basic response that occurs in all animal species when circumstances bring it forth. Our role as emergency physicians and as people is to try to understand the factors that bring out this destructive response that adult humans have to their offspring. Once we fully understand the causes of the problem of child abuse, we can begin the difficult process of addressing the solutions.

REFERENCES

1. AMA diagnostic and treatment guidelines concerning child abuse and neglect. JAMA 254:796, 1985
2. Kempe CH, Helfer R: The Battered Child, 3rd ed. University of Chicago Press, Chicago, 1980

3. Ludwig S: Child abuse. p. 1027. In Fleisher G, Ludwig S (eds): Textbook of Pediatric Emergency Medicine. Williams & Wilkins, Baltimore, 1983
4. Ellerstein N (ed): Child Abuse and Neglect: A Medical Reference. John Wiley & Sons, New York, 1981
5. Shaw A: Are you sure it's child abuse? Contem Pediatr 3:92, 1986
6. Ludwig S: Child abuse. p. 107. In Pediatric Emergencies, Clinics in Emergency Medicine. Churchill-Livingstone, New York, 1985
7. Gelles RJ: Child abuse as psychopathology: A sociological critique and reformulation. Am J Orthopsychiatry 43:611, 1973
8. Fromm E: The Anatomy of Human Destructiveness. Holt, Rinehart & Winston, New York, 1973
9. Koop CE: Presented at the Annual Meeting of the American Academy of Pediatrics, New York, 1981
10. Bergman A, et al: Changing spectrum of serious child abuse. Pediatrics 77:113, 1986
11. Gil DG: Violence Against Children: Physical Abuse in the U.S. Harvard University Press, Cambridge, MA, 1979
12. Kempe HC, Helfer RE: Child Abuse and Neglect: The Family and the Community. Ballinger, Cambridge, MA, 1976
13. Finkelhor D, et al: The Dark Side of families: Current Family Violence Research. Sage Publications, Beverly Hills, CA, 1983
14. Ludwig S: The abused patient—current therapy. In Emergency Medicine. BC Decker, New York, 1987
15. Martin HP: The Abused Child: A Multidisciplinary Approach to Developmental Issues and Treatment. Cambridge, MA, 1976
16. Morris MG, Gould RW, Matthews PJ: Toward prevention of child abuse: Some social signs of abusive parents. Children 55, 1964
17. Morris J, et al: To report or not to report: Physician's attitudes toward discipline and child abuse. Am J Dis Child 139:194, 1985
18. Wilkerson AE (ed): Rights of Children. Temple University Press, Philadelphia, 1974

6 Problems in the Management of Multiple Trauma

Joseph J. Tepas III

Each year approximately 9,000 children under age 14 years and another 26,206 adolescents and youths between 15 and 24 years of age die as a result of accidental injury.[1] Considered in light of a 24 to 40 percent incidence of preventable death in injured children, this fact becomes tragedy and clearly mandates an understanding of the unique problems in caring for the injured child.[2] Just as with all other aspects of pediatric care, proper assessment and management of the injured child require more than an application of adult principles to a smaller organism.

Children have common patterns of injury, unique physiologic responses, and special needs on the basis of size, maturity, and psychosocial development. To ignore these requirements is to invite disaster and, more importantly, will cheat our most precious natural resource. This chapter first reviews the common problems and errors in the care of the pediatric trauma victim and then reviews optimal trauma management and its rationale.

THE CHILD AS A TRAUMA VICTIM

Considering the child as a trauma victim, many unique characteristics must be addressed. The incidence of blunt versus penetrating trauma is highest in the pediatric population. Analysis of the National Pediatric Trauma Registry (NPTR) data identified blunt trauma as the mechanism of

injury in 89 percent of cases. Falls were the most common cause of injury (39 percent), closely followed by vehicular related accidents (38 percent). Injury was accidental in 87 percent of cases, sports related in 4 percent, and the result of assault in 4 percent. Multisystem injury is the rule rather than the exception (mean 2.1 diagnoses per patient); therefore, all organ systems must be assumed to be injured until proved otherwise. Although there may be minimal external evidence of injury, potentially significant external derangement of every major organ must be excluded.

The unique anatomic characteristics of the pediatric population require special considerations in assessing and treating the pediatric trauma victim. The child's smaller size produces a smaller target to which are applied linear forces from fenders, bumpers, and falls. Because of diminished body fat, increased elasticity of connective tissue, and close proximity of multiple organs, these forces are less well dissipated, and a more intense energy is applied to multiple organs. The skeleton of a child is incompletely calcified, contains multiple active growth centers, and is more resilient, making it less able to absorb kinetic forces applied during a traumatic event. A situation may be produced wherein significant internal derangement has occurred in the face of apparently minimal external damage.

The ratio between a child's body surface area to body volume is highest at birth, diminishing throughout infancy and childhood. As a result, thermal energy loss becomes a significant stress factor in the smaller child. While this problem may not be life-threatening of and by itself, it frequently provides additional stress to a child who may be hypotensive and in severe pain. If allowed to develop, severe hypothermia will frequently initiate irreversible cardiovascular collapse.

The psychological ramifications of caring for an injured child can also present a major challenge. Especially in the very young child, emotional lability frequently leads to a regressive psychological behavior when stress, pain, or other perceived threats intervene on the child's environment. The child's ability to interact with unfamiliar individuals in strange environments is usually limited, making history taking and cooperative manipulation extremely difficult. An understanding of these characteristics and a willingness to cajole and sooth an injured child will frequently be the most effective means of achieving good rapport and a more comprehensive assessment of the child's physiologic state.

A final consideration in dealing with pediatric trauma is the effect that injury may have on subsequent growth and development. Unlike the adult, the child must not only recover from the effects of a traumatic event but must also continue along a normal process of growth and development. The effect of injury on this process, especially in long-term disability, growth deformity, or abnormal subsequent development both physiologically and psychologically, cannot be underestimated. Children sustaining even minor injury may have prolonged disability either in cerebral function, psychological adjustment, or organ-system disability.[3,4] At the moment, it is unknown how many of the 36 percent of injured children with functional impairment at discharge have long-term disability. It is certain, however, that the cost of

correcting these problems will be staggering and lifelong. The effect of inadequate or inappropriate care during the immediate post-traumatic period may have consequences not only on the child's survival, but perhaps as importantly on the quality of the child's life for years to come.

IMMEDIATE LIFE THREATS

The ultimate result of care for the injured child in regard to mortality, morbidity and disability is largely determined by the quality of care rendered during the first minutes after injury. By far the most common error that occurs at this time is failure to diagnose a potentially lethal injury as the result of an unorganized, chaotic assessment. Clearly, a systematized approach using a well-understood protocol is the best defense against overlooking an injury that may be rapidly fatal or that may provide unnecessary morbidity or disability if inadequately managed during the immediate post-injury period. As in the adult, the three most common causes of immediate death secondary to injury are hypoxia, overwhelming central nervous system (CNS) trauma, or massive hemorrhage. Lack of efficient triage to the most appropriate center for treatment can compound any or all of the above problems.

Hypoxia

Initial airway management must focus not only on the confirmation of adequate patency but on the need for supplemental oxygen as a component of therapy for CNS injury or hypoperfusion as well. Confirmation of airway patency does not preclude the need for supplemental oxygen. This is especially true in cases of closed head injury, in which the patient may be somewhat obtunded and may be provided with a significantly improved potential for functional recovery if adequate cerebral oxygenation can be supported. If at all possible, patients in whom airway manipulation will be required for definitive management should be hyperventilated with mask O_2 prior to initiation of any attempt at intubation. Obviously, in the case of a child whose airway is completely obstructed, needle cricothyroidostomy with high-flow (15 L/min) O^2 delivered through the cannula should be an initial consideration prior to more definitive airway management. In the pediatric age group, supplemental oxygenation is clearly a modality that is easily obtainable by most persons caring for children and carries absolutely no potential for morbidity or longterm detrimental effect.

Brain Injury

Children who have sustained massive brain injury may have a degree of injury that will be rapidly lethal, regardless of management technique. Nevertheless, provision of adequate cerebral circulation and oxygenation

must begin as soon as the patient is identified. In the most severely injured child, these efforts may have no positive effect at all, whereas in the child whose injury is potentially reversible in the acute phase, this therapy may make a significant contribution toward a more complete functional recovery. Children sustaining closed head trauma frequently present with a mild degree of obtundation and have sustained a period of unconsciousness that is not always recorded during the patient's initial evaluation. Clearly, a history of loss of consciousness is one of the most important prognostic parameters that will indicate degree of potential central nervous system injury that the patient may have sustained. Likewise, incomplete documentation of a baseline neurologic status, including response to sensory stimulation, motor function, and cranial nerve function, is a common shortcoming in initial pediatric trauma care. Since CNS injury is an ongoing spectrum of disease that begins with an initial depolarization of CNS neurons and then proceeds along a potentially recognizable course of secondary edema and hypoprofusion, the absence of an adequate baseline assessment makes ongoing follow-up and assessment of intervention extremely imprecise and difficult.

Hemorrhage

Fortunately, the experience recorded in the NPTR indicates that the incidence of children presenting to emergency facilities in rapidly fatal hypovolemic shock is relatively low. Most children sustaining this type of injury die on the scene or are dead on arrival in the emergency room. In those children who present in an agonal state, the most common problem encountered is inadequate restoration of red blood cell (RBC) mass lost as the result of hemorrhage. Patients in this category clearly require massive boluses of crystalloid solution (Ringer's lactate) as expeditiously as possible. Unfortunately, they are frequently transfused with blood that is old and cold, so that oxygen-carrying capacity is minimally restored. The best means of handling this particular problem is rapid resuscitation with crystalloid and immediate transfusion with packed RBCs reconstituted with warm normal saline. This permits a degree of control over the viscosity of the transfused solution as well as an opportunity to warm the transfused RBCs as effectively and quickly as possible.

The concept of evolving shock must also be considered in any child who has sustained blunt trauma. While the percentage of children presenting in frank hypovolemic shock is relatively low, the incidence of children sustaining hypotension secondary to a less severe injury is to be considered the rule rather than the exception. These children may have minimal change in their initial vital signs and progress to profound hypovolemia within a short period of time. Accordingly, any child sustaining blunt trauma must have vital signs carefully monitored in order to ensure that this does not occur and that inadequate resuscitation does not set the stage for a profound cardiovascular collapse at a time relatively remote from the time of injury.

An additional common error seen in initial evaluation of the injured child is the tendency to overresuscitate the patient with fluids once venous access has been secured. Given the potentially high incidence of closed head injury and blunt trauma in regard to the relatively low incidence of severe hemorrhagic shock, it becomes obvious that gross overresuscitation of a child with a closed head injury may be more detrimental than effective and may actually worsen the evolving cerebral edema. Accordingly, careful assessment of the child's vital signs and even more compulsive evaluation of the effect of therapeutic intervention must be the primary consideration for the child during the immediate period after presentation to the emergency room. A final common pitfall in initial management of the injured child relates to inappropriate transfer decisions in regard to disposition and ultimate care.

Triage

The number of trauma centers capable of treating the severely injured child is relatively small. The decision as to which child is most appropriately referred to these centers must proceed on the basis of a careful and rapid evaluation of the child's status on initial presentation. The two most common problems encountered are incomplete assessment that overlooks potential additional organ system injury, and inadequate stabilization prior to discharge. The Pediatric Trauma Score (PTS) provides a reliable and simple means of carefully assessing the injured child in order to come up with a rapid numeric quantitation of the degree of injury and potential prognostic outcome. This has been shown to be an effective means of determination of triage to the appropriate trauma center; it is applicable both in the field as well as in emergency centers functioning as referral sources in a regional trauma care network.

Having thus identified potential pitfalls attendant to initial assessment of the child, it is obvious that this process must be one of a protocolized organized approach that begins with airway assessment and proceeds to a progressively more involved and exhaustive organ system survey.

INITIAL ASSESSMENT

The small size of the patient, the diminished caliber and size of the vascular system, and the unique anatomic characteristics of the airway frequently cause the standard procedures used in basic life support to be extremely challenging and technically difficult in the pediatric patient (Table 6-1).

Obviously, the immediate availability of appropriate-sized equipment is essential for successful initial management of the injured child. An attempt at placement of an overly large intravenous cannula or inappropriate-sized endotracheal tube may result in more injury than benefit for the patient. A pediatric trauma resuscitation area must therefore have im-

Table 6-1 Problems and Potential Adverse Outcomes in the Initial Assessment of the Child who has Sustained Multiple Trauma

Problem	Potential Result
Airway	
Failure to protect cervical spine	Completion of cord injury
Supplemental O_2 not immediately provided	Worsening hypoxia to injured brain
Lack of understanding of sniffing position	Poor visualization of larynx, passive airway obstruction
Inadequate preoxygenation prior to airway manipulation	Severe hypoxia with only minimal airway control measures
Use of inappropriately sized endotracheal tubes	Direct damage to larynx/trachea
Breathing	
Failure to assess breath sounds repeatedly	Development of tension pneumothorax with improved tidal volume
Incomplete examination overlooking	
Chest dynamics	Missed flail chest, pneumothorax
External bruises	Harbinger of significant internal injury
Dorsal surfaces	Missed penetrating injury
Circulation	
Nonuse of Trendelenburg position	Insufficient filling of central circulation
Underassessment of shed blood volume	Inadequate initial resuscitation
Inadequate or late transfusion of RBCs	Minimal oxygen-carrying capacity
Overresuscitation of crystalloid in absence of hypotension	Needless increase for potential cerebral edema
False security from normal or near-normal initial vital signs	Missed hypotension progressing to shock
Disability	
Failure to document mechanics of cervical spine injury	Improper immobilization
Loss of consciousness overlooked in history	Missed potential for delayed injury effect (edema, bleed)
Inadequate baseline assessment	Inability to document change, especially fundoscopy
Incomplete examination	Missed "minor" injury with major disability
Laboratory	
Overuse of tests	Unnecessary blood drawn, unnecessary expense, and laboratory overwork
Incomplete assessment	Missed metabolic derangement
Triage	
Inappropriate transfer decision	Inadequate total care, overuse of expensive limited resource
Incomplete assessment of injury in transport	Progression of missed injury in transport
Inadequate stabilization	Deterioration during transport

Table 6-2. Equipment Necessary For Initial Care of the Injured Child

	Premature (3 kg)	Newborn (3.5 kg)	6 months (7 kg)	1–2 yr (10–12 kg)	5 yr (16–18 kg)	8–10 yr (24–30 kg)
C-collars	—	—	Small	Small	Small	Medium
Chest tubes	10–14 Fr	12–18 Fr	14–20 Fr	14–24 Fr	20–32 Fr	28–38 Fr
Nasogastric tubes	5 Fr Feeding	5–8 Fr Feeding	8 Fr	10 Fr	10–12 Fr	14–18 Fr
Foley catheters	5 Fr Feeding	5–8 Fr Feeding	8 Fr	10 Fr	10–12 Fr	12 Fr
IV equipment	22–24 angio 25 scalp	22–24 angio 23–25 scalp	22–24 angio 23–25 scalp	20–22 angio 23 scalp	20–22 angio 19 scalp	20–22 angio 19 scalp
Arm bands	6 in.	6 in.	6–8 in.	8 in.	8–15 in.	15 in.
Blood pressure cuffs	Newborn	Newborn/ infant	Infant/child	Child	Child	Child/ Adult
Bronchovesicular markings	Infant	Infant	Pediatric	Pediatric	Pediatric	Pediatric/ Adult
Laryngoscopes	0 Straight	1 Straight	1 Straight	1 Straight	2 Straight and curved	2–3 Straight and curved
Endotracheal	2.5–3.0 Uncuffed	3.0–3.5 Uncuffed	3.5–4.5 Uncuffed	4.0–4.5 Uncuffed	5.0–5.5 Uncuffed	5.5–6.5 Cuffed
Suction/stylets	6–8F/6Fr	8Fr/6Fr	8–10Fr/6Fr	10Fr/6Fr	14Fr/14Fr	14Fr/14Fr
Oral airways	Infant	Infant/Small	Small	Small	Medium	Medium/ large
O_2 masks	Premie/ Newborn	Newborn	Pediatric	Pediatric	Pediatric	Adult

(Luten R: Pediatric resuscitation chart and equipment shelf: Aids to mastery of age-related problems. J Emerg Med 4:9, 1986.)

mediately available laryngoscope blades, endotracheal tubes, nasogastric tubes, Foley catheters, chest tubes, blood pressure cuffs, oxygen masks, and other associated equipment of appropriate size. Table 6-2 indicates our recommended sizes of standard rescue equipment required for children of different size and age groups.[5] These guidelines should be posted and readily available both for emergency field rescue units as well as for emergency rooms and resuscitation areas.

Airway

The primary goal of initial assessment and triage of the injured child is the restoration of adequate tissue oxygenation as effectively and completely as possible. Oxygenation and circulation are as essential to the injured child as to the adult. In this regard, the standard A-B-C, principles of airway control, breathing, and circulation are no differently addressed in the injured child than in the injured adult. As always, the first priority of assessment is the child's airway. The smaller the child, the greater the disproportion between the size of the cranium and midface, and the greater the propensity of the posterior pharyngeal area to buckle as the relatively larger occiput forces passive flexion of the cervical spine. As a result, the child's airway is best protected by a slightly superior–anterior position of the mid-face known as the sniffing position. Careful attention to mainte-

nance of this position is especially important in the obtunded child whose level of consciousness is waxing and waning.

The larynx of the child is smaller than the adult, has a slightly more anterocaudad angle, and is frequently more difficult to visualize for direct cannulation. Despite this, the most reliable means of ventilation in the child with airway compromise remains direct endotracheal intubation. Nasotracheal intubation requires blind passage around the relatively acute posterior nasopharyngeal angle and may cause inadvertent penetration of the cranial vault. In the absence of appropriate intubation equipment, bag-valve-mask ventilation with 100 percent oxygen is an acceptable alternative. In the child whose craniofacial injuries cause upper airway obstruction, a needle cricothyroidostomy through which high-flow (15 L/min) oxygen can be delivered in 45/15 second on–off cycles, may provide enough support until definitive management can be provided.

Breathing

As in the adult, endotracheal intubation must include careful attention to the cervical spine. The head must be maintained in a neutral position by an assistant while careful laryngoscopy and insertion of an endotracheal tube are performed. In infants and toddlers, an uncuffed endotracheal tube should always be used, the size of which can be readily assessed by evaluating the diameter of the child's fifth finger. A slight amount of cricoid pressure will frequently bring the anterior structures of the child's larynx into better view and will passively obstruct the esophagus thereby insuring accurate cannulation of the trachea. A frequent error made in children intubated under emergency circumstances is an overly aggressive insertion of the endotracheal tube causing right mainstem bronchial intubation. Accordingly, careful auscultation of the chest must be performed as soon as the endotracheal tube is placed. In addition to confirming the presence of the endotracheal tube above the carina, this will confirm adequate breath sounds bilaterally and rule out the possibility that a tension pneumothorax or other primary pulmonary injury has occurred. A child with a compromised airway and a primary pulmonary parenchymal injury who is successfully intubated may be in greater jeopardy for the development of a tension pneumothorax as a result of a more efficient deliv. y of tidal volume to the tracheobronchial tree. It is absolutely essential that careful auscultation be a reflex maneuver immediately and periodically after insertion of the endotracheal tube to document both the presence of breath sounds, and the overall status of ventilatory function.

Circulation

Establishment of intravenous access in a child can be succinctly summarized by the adage that injured children need a good intravenous line, not necessarily a central one. The survival rate from immediate exsanguinating

injury is low in the pediatric population. Fortunately, most injured children usually present with some degree of adequacy to their circulating volume. In fact, 2,665 of 3,045 NPTR records reported an initial systolic blood pressure above 90 mmHg.

If initial assessment suggests severe hypotension or shock, the most likely cause is blood loss through either a major external wound (readily observable), an intrathoracic wound (identifiable by diminished ventilatory mechanics and auscultatory findings), or loss of blood through a major abdominal visceral injury. Since blood is a nondistensible medium, the latter will frequently produce abdominal distention and increasing abdominal girth. Consequently, any attempt at repletion of a child with severe hypotension or frank shock must deliver adequate fluid volume to the right atrium. The most appropriate site for initial venous access is thus above the diaphragm and should first address the antecubital fossa. If the antecubital veins cannot be cannulated, either percutaneous cannulation or direct cannulation of the external or internal jugular veins should be considered. Placement of a subclavian catheter in an injured child should be performed only under the most controlled of circumstances. The area beneath the clavicle is extremely small and has in close proximity most of the major neurovascular components of the upper extremity. A misguided catheter may not only be unsuccessful in attaining venous access but actually may worsen the child's overall status by causing either a pneumothorax or brachial plexus injury.

Multiple sites beneath the level of the diaphragm are much more easily approachable for direct surgical cannulation. A saphenous venous cutdown at the level of the ankle or direct cannulation of the femoral vein at the groin are both appropriate and adequate resuscitative measures, however they should be considered after an attempt is made above the diaphragm.[6] Obviously, in the severely hypovolemic child multiple IV sites are required and use of the subdiaphragmatic areas are to be encouraged as supplemental.

As in the adult, lactated Ringer's or other balanced electrolyte solutions are the best initial resuscitating fluid for the hypovolemic child. Since the intravascular phase of crystalloid is so short, a 3:1 ratio for crystalloid resuscitation must be followed. As in the adult, signs of significant hypotension develop with the loss of approximately 25 percent of the circulating volume. Thus, an initial resuscitative bolus should reflect approximately 25 percent of the standard circulating volume in the pediatric patient, which calculates to be approximately 20 ml/kg. Considering the 1:3 ratio, it becomes obvious that a bolus of 40 to 60 ml/kg is actually what is required to achieve adequate and rapid initial repletion of significant volume loss.

Injured children usually present in one of three ways; normotensive, hypovolemic shock, or evolving shock. The greater majority demonstrate little evidence of blood loss and hypotension. With the high incidence of potential head injury, it becomes obvious that volume repletion of these children must consider not only the restoration of adequate circulating volumes but also the possibility of overresuscitation with potentially detrimen-

tal effect on evolving cerebral edema. Children who present in hypovolemic shock may need both large amounts (50 to 60 ml/kg in 20 ml/kg boluses) of crystalloid resuscitation as well as fresh RBCs as soon as possible. This group, fortunately very small, consists of the children in whom a high potential for preventable deaths occur. As a result of expeditious crystalloid bolus, these patients frequently regain adequate cardiac output of a circulating volume that has minimal oxygen-carrying capacity. Tissue oxygenation plummets because of the diminished circulating RBC mass as well as the diminished oxygen-carrying capacity of senescent RBCs usually transfused during initial resuscitation. The child who presents in shock must therefore be aggressively resuscitated with at least 60 ml/kg as an initial fluid bolus and must be transfused with RBCs that are as fresh and warm as soon as possible. Only under these circumstances will these unfortunate children have even the slightest chance of successful management of their hemorrhagic injury.

A major consideration in the assessment of the pediatric trauma victim is the concept of evolving shock. By nature of their increased physiologic reserve, children will frequently sustain hemorrhagic injury and present to the emergency room with only slightly abnormal vital signs. Initial tachycardia may be the result not only of hypovolemia, but of the effect of psychological stress, pain, and fear. Moreover, a small child may have a systolic blood pressure that may be considered alarmingly low for an adult, but that reflects normal homeostasis for a young, healthy child. Accordingly, all injured children must be monitored continuously with close-order observation of blood pressure, heart rate, respiratory rate, and overall CNS status. Adequate circulating volume can be maintained in the child with a hemorrhagic injury by increasing peripheral vascular resistance to maintain mean arterial pressure. If initial resuscitation is inadequate, circulating volume will eventually diminish to a point below which increased peripheral resistance can maintain arterial pressure. Tragically, this may occur hours after the injury, when the child is either being inadequately observed or undergoing secondary assessment at a location remote from adequate resuscitative facilities. The concept of evolving silent shock must therefore constantly be considered; the ability to monitor injured children closely and resuscitate them appropriately must be provided until it is absolutely confirmed that no evidence of significant hemorrhagic injury exists.

Disability

Once initial assessment, airway control, and fluid resuscitation have been accomplished, attention can be addressed to a thorough examination of the entire child. The overall guidelines for this evaluation must be a high index of suspicion that every major organ system is injured until proved otherwise. Examination must first assess neurologic status. While the child's level of consciousness remains a reliable prognostic indicator of se-

verity of CNS injury, this assessment must be expanded to include pupillary and ocular muscle function, mid-brain function, motor function, orientation, and sensory perception.[7] The Glascow Coma Scale is an excellent guideline for assessment of neurologic status and should be repeated frequently throughout the evaluation process of the child to determine progression or regression of any CNS findings.

Completion of the child's physical examination follows a standard head to toe protocol. Intrathoracic organs must be evaluated with special consideration of potential cardiac or pulmonary contusions, both of which may be worsened by overly aggressive fluid resuscitation. Since the increased resiliency of the child's ribs may have permitted transmission of significantly greater energy to underlying thoracic organs, there may be an extensive direct pulmonary contusion. The child's pulmonary status could only be worsened by aspiration should the level of consciousness diminish. Examination of the abdomen must focus on distention, tenderness, discoloration, or presence of a mass. A careful rectal examination must be performed addressing the status of the rectal sphincter, the presence of blood in the rectal ampulla, and the overall status of the intrapelvic structures. Careful palpation of the iliac crests will provide evidence of an unstable pelvic fracture and will increase suspicion for possible retroperitoneal or urologic injury. A nasogastric tube should be passed and the aspirated contents carefully assessed for the presence of blood. In the unstable child, a Foley catheter should be passed into the bladder after exclusion of a pelvic, perineal, or direct urethral injury. When auscultating the injured child's abdomen, the presence of bowel sounds is highly suggestive of normal peristalsis with lessened potential for significant injury. The absence of bowel sounds may be the effect of aerophagia, ileus, stress, or a primary intraperitoneal process. Bowel sounds thus play an extremely minor role in the overall assessment of the child's abdomen, although their status should be assessed and documented for future comparison.

Each extremity must be palpated to rule out deformity, diminished vascular supply, or neurologic deficit. The child's incompletely calcified skeleton with its multiple growth centers increases the possibility of epiphyseal disruption. Accordingly, any area of edema, pain, tenderness, or diminished range of motion should be carefully evaluated and then radiographed.[8] A missed orthopedic injury may have little effect on mortality but a major long-term effect on deformity and disability.

Once anoxia and hypoprofusion have been either excluded or treated, the next major threat to the injured child is sepsis secondary to disruption of a hollow viscus. Because of this, the most efficacious means of assessing the injured child is the combination of repeated thorough physical examinations and appropriate imaging techniques determined on the basis of initial findings.

As the number of reports documenting the successful healing of splenic, hepatic, and renal injuries without operative intervention has increased, a consensus has developed among pediatric surgeons that the presence of

blood within the abdominal cavity does not necessarily mandate operative intervention.[9–11] Because of this, peritoneal lavage is not a particularly specific tool for the assessment of an injured child's abdomen. While analysis of lavage return for increased leukocyte count or the presence of bile, amylase, or feces may be helpful, in reality most types of injuries suggested by these findings can be easily diagnosed by thorough physical examination complemented by computed tomography (CT) when indicated.

Because head trauma is frequently the most severe and life-threatening injury, the probability of an injured child requiring immediate CT scan for assessment of the CNS is quite high. Accordingly, in children with findings of associated abdominal trauma who are hemodynamically stable, it is appropriate to complement the CT scan of the head with a scan of the abdomen and pelvis. When indicated, the installation of IV contrast will identify both renal units as well as provide an indication of their functional capacity. The installation of contrast within the stomach via swallowing or direct nasogastric tube will also highlight the status of the upper gastrointestinal (GI) tract and further emphasize any abnormalities in architecture of the spleen, liver, or retroperitoneum. Obviously, prior to instillation of contrast by any route, the potential for allergic response or detrimental hyperosmotic effect must be considered.

In the absence of positive findings on initial physical examination, the most effective means of further evaluation of the child is continued careful abdominal physical examination. The development of increasing tenderness, abdominal guarding, or distention are all signs suggestive of evolving peritonitis, which mandates further evaluation and, if necessary, prompt operative intervention. Automatic abdominal CT scan obtained in the absence of objective historical or physical findings suggestive of abdominal injury is rarely useful. A review of 98 NPTR records identified a 54 percent rate of positive findings in children with objective evidence of abdominal injury and no positive findings in those without objective evidence of abdominal injury.

Laboratory Findings

Upon arrival in the emergency department, all pediatric trauma patients must have blood drawn for complete blood count, and transfusion crossmatch. Other than the hematocrit, there is very little laboratory support required for the accurate initial assessment and triage of an injured child. Serum electrolytes, coagulation profile, platelet count, arterial blood gases, and the myriad of laboratory tests that attend routine medical care are frequently noncontributory in the first phase of assessment of the injured child. For all children, however, it is essential to obtain a white blood count, hematocrit, and urinalysis, so that subsequent assessments can assist documentation of either evolving inflammatory process (i.e., peritonitis) or primary urologic dysfunction (i.e., renal contusion). The remainder of the standard laboratory battery including serum electrolytes, amylase, co gula-

tion parameters, and platelet count, are frequently superfluous studies in all but the most seriously injured child. In a recent review of the contribution of emergency room laboratory studies to the initial assessment and triage of the injured child, we identified that $2 of every $3 spent for laboratory assessment is wasted on laboratory tests that frequently return too late to be considered in initial assessment of the child or with results that are completely within normal limits and noncontributory to clinical decision making.[12] It is obvious that in all but the most seriously injured child, most of these laboratory values reflect the normal baseline homeostasis that existed at the time of injury.

Triage

The tragedy of preventable traumatic death has been well documented in multiple studies reported over the past three decades. In regard to the pediatric population, Ramenofsky et al.[13] identified an incidence of 53 of 100 pediatric trauma deaths that could be classified as preventable. These statistics have been among the prime motivations for development of regional trauma referral systems intended to provide continuous high-quality sophisticated care at a few centers specifically designated for this function.

Since the level of care and commitment is extremely expensive, the functional efficiency of such systems depends on accurate and judicious triage of appropriately injured patients to appropriately designated centers. This problem becomes even more acute for pediatric trauma patients, since the few hospitals designated as pediatric trauma centers frequently serve relatively larger population areas. Intelligent triage of an injured child thus becomes a major factor not only in the determination of initial patient management but in the overall function and impact of the regional trauma system as a whole as well.

The PTS was developed specifically as a triage tool for dealing with pediatric trauma victims. It consists of a grading system wherein six components of pediatric injury are each graded and added to produce a score predictive of injury severity and mortality potential (Table 6-3). Size is

Table 6-3. Pediatric Trauma Score

PTS Component	Category		
	+2	+1	−1
Size	≥20 kg	10–20 kg	<10 kg
Airway	Normal	Maintainable	Unmaintainable
Systolic BP	≥90 mmHg	90–50 mmHg	<50 mmHg
Central nervous system	Awake	Obtunded/ L.O.C.	Coma/decerebrate
Open wound	None	Minor	Major/penetrating
Skeletal	None	Closed/fracture	Open/multiple fractures

Sum: _____ (PTS)

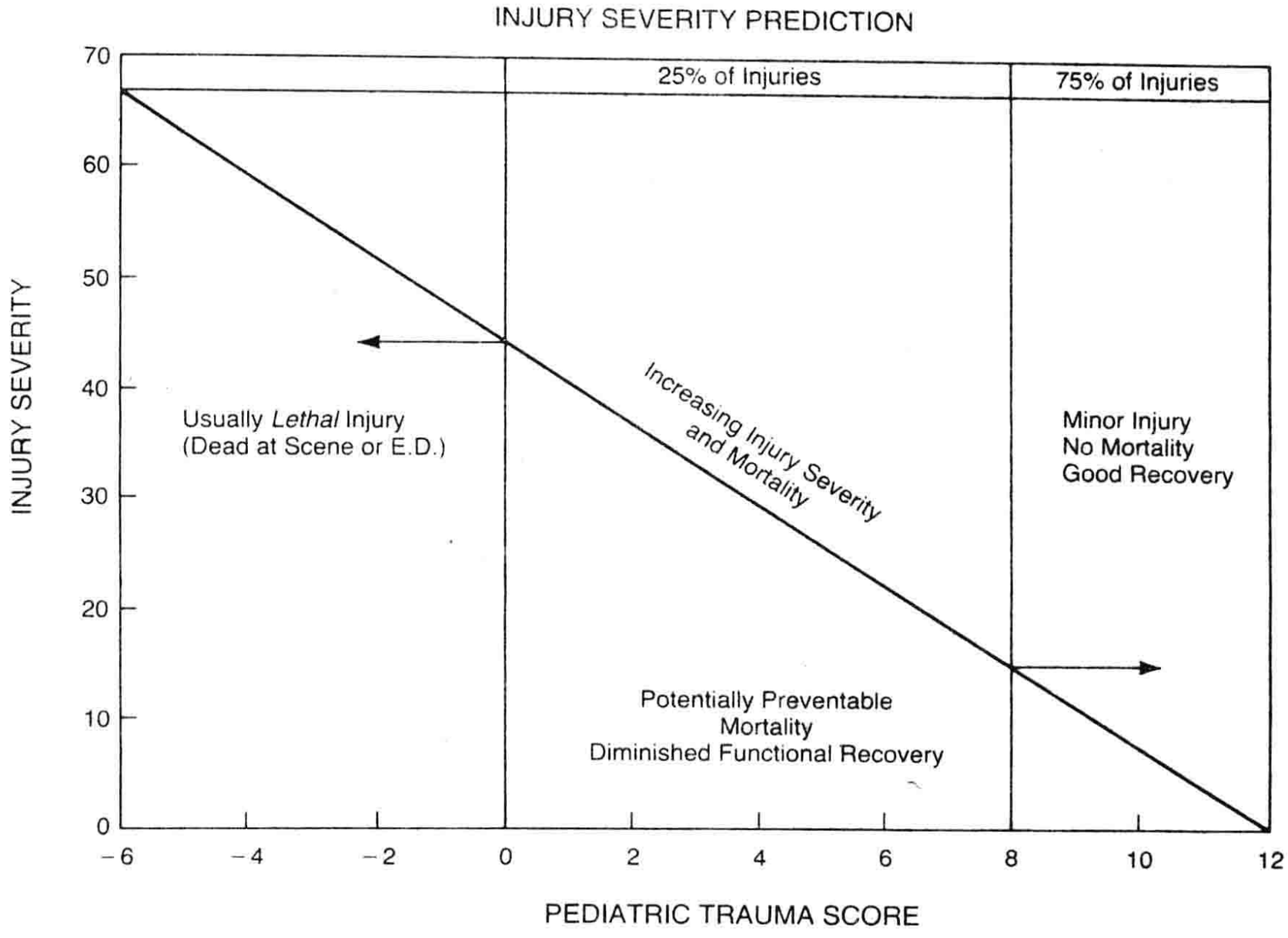


Fig. 6-1 Predicting the severity of injury.

considered first because it is readily obvious and is a major consideration in the infant–toddler group, in which mortality from injury is highest. Airway is assessed not just as a functional status but as a descriptor of what care is required to provide adequate management. Any child who requires endotrachial intubation for management is clearly in a life-threatened status and is graded as AL minus one (−1). Systolic blood pressure assessment is arranged primarily to identify those children in whom evolving preventable shock may occur—50 to 90 mmHg systolic pressure (+1). Regardless of size, a child whose systolic pressure is below 50 mmHg (−1) is in obvious jeopardy. The child whose systolic pressure exceeds 90 mmHg (+2) probably falls into a better outcome category than does a child with even a slight degree of hypotension.

The most important factor in initial assessment of the CNS is level of consciousness. Because children frequently sustain transient loss of consciousness during injury, the obtunded (+1) grade is applied to any child with loss of consciousness, no matter how fleeting.[14] Thus, the patient is identified as able to develop potentially fatal and frequently treatable intracranial sequelae secondary to head injury. Because of the high incidence of skeletal injury in the pediatric population and its potential contribution to mortality, it is also a consideration in determining PTS.[15] Finally, cutaneous injury both as an adjunct to common pediatric injury patterns and as an injury category that includes penetrating wounds is considered in computing the PTS.

By nature of its simple design, the PTS serves as a simple checklist ensuring that all components critical to initial assessment of the injured child have been considered. It can be employed by paramedics in the field as well as by physicians in facilities other than pediatric trauma units. As a predictor of injury, the PTS has a statistically significant inverse linear relationship with both the injury severity score (ISS) (Fig. 6-1) and mortality.[16] Analysis of this relationship has identified a threshold PTS of 8, below which all injured children should be triaged to an appropriate pediatric trauma center. These are the children in whom the potential for preventable mortality and morbidity is highest. According to NPTR statistics, they represent approximately 25 percent of all pediatric trauma victims and are clearly the ones who require the most aggressive monitoring and observation.

DEFINITIVE ASSESSMENT

Head Injury

Head injury continues to be the most common cause of death in the pediatric trauma patient.[7,17] While the most severe injuries are generally treatable only by prevention, many initial resuscitative measures may at least ameliorate the initial effect of the child's CNS injury. Again, the premise of adequate oxygenation, ventilation, and circulation must be addressed and, in the head injured child, hyperventilation with adequate oxygenation should be considered (Table 6-4).[7] From the moment of injury, the reflex

Table 6-4. Definitive Assessment of the Child who has Sustained Multiple Trauma

Problem	Result
Head and Neck	
Failure to palpate skull for depressions	Missed fracture with potential bleed
Failure to note soft tissue, edema, discoloration, tenderness	Missed basal fracture
Failure to document absence of blood in auditory canal, nares, mouth	Confusion regarding injury versus result of intervention
Inadequate assessment of cervical spine	Progression of cord injury
Chest	
Incomplete auscultation of breath sounds, heart sounds, abnormal sounds	Missed life-threatening injury
Missed pulmonary contusions worsened by overhydration	Progression of respiratory failure
Inadequate assessment in absence of external trauma	Missed internal derangement
Abdomen/Pelvis	
Failure to decompress stomach	Ventillatory dysfunction
Underassessment of abdominal tenderness	Missed intestinal pancreatic injury
Inappropriate peritoneal lavage	False data/security
Inadequate documentation of baseline findings	Inadequate subsequent evaluation
Failure to complete rectal examination	Missed injury, evaluation of sphincter tone
Incomplete examination of genitalia	Missed disruption or injury
Extremities	
Failure to document limitation of range of motion, digital and vascular status	Missed fracture or dislocation
Failure to diagnose greenstick torus fractures	Missed potential disability
Disposition	
Inappropriate delays	Increasing potential for morbidity/mortality
Missed subtle child abuse	Recurrence of injury

responses occurring within the closed cranial vault are partially related to perfusion pressure, tissue oxygenation, and evolving edema. Thus, any child who sustains head trauma in which there has been a continued diminution in level of consciousness should be considered for hyperventilation as soon as initial therapy is begun. Tissue perfusion can be improved considerably by simply hyperventilating the patient to a PCO_2 level of 25 to 28 mmHg. This is easily achieved in the obtunded or comatose child by initial airway control and ventilation with either mask or endotracheal tube with 100 percent oxygen. Since animal studies have suggested that cerebral ischemia

may develop at PCO_2 levels below 20 mmHg, careful blood gas monitoring is essential for effective hyperventilation therapy.[18]

Initial assessment of the level of consciousness is a rapid and reliably prognostic exercise. It must be considered that any child, regardless of the adequacy of neurologic status on initial assessment, may be susceptible to the secondary changes post-CNS injury as the result of even the most minor trauma. Loss of consciousness is an excellent criterion that indicates which children should be more aggressively monitored and carefully observed. Any child who sustains even a transient loss of consciousness must be assumed to have sustained adequate degree of mechanical trauma to the brain stem and reticular activating system to permit at least transient depolarization and dysfunction. If the level of consciousness returns to normal, careful observation should be considered to rule out the possibility of evolving secondary dysfunction as a result of cerebral edema or space-occupying lesion in the form of subdural or epidural hematoma. A careful Glasgow Coma Scale assessment and follow-up examination is therefore paramount to successful monitoring and management of these children.

Children whose Glasgow Coma Score is ≤ 7 or who have sustained severe intracerebral injury require direct monitoring of the intracerebral pressure. While this modality is somewhat imperfect in its reflection of the status of the neuronal metabolism, it is still a relatively good predictor of the overall status of the circulation and perfusion pressures within the cranial vault. The application of other nonsurgical methods of coma management includes initiation of hypothermia, barbiturate coma, and chronic mannitol or glycerol drip therapy. All these modalities require extremely aggressive and careful monitoring and must be performed under the direct observation and control of a physician in a pediatric critical care unit. Since intracerebral pressure is just one facet of a complex pathophysiologic process, concomitant monitoring of central venous pressure, cardiac output, and arterial oxygenation must also be a consideration in these children. Recent experimental work relevant to the physicochemical response of the CNS to anoxia suggests that superoxide radical formation may play a major role both in immediate injury response and in outcome. Multiple investigations have evaluated a variety of drugs that may influence this process, but to date, nothing has been identified that has a direct documented effect on minimizing initial cerebral response to anoxic injury.

Thoracic Injuries

The extremely resilient rib cage of the child with its incomplete calcification provides a means wherein energy transferred through the thoracic cage to intrathoracic organs is frequently more devastating than in the adult population. As a result, the child may have sustained a significant parenchymal injury, disruption of vascular anatomy, or simple contusion without manifestation of even the slightest degree of abnormality on external examination. A high index of suspicion is key to diagnosis of these

injuries. Every child who has sustained injury to the chest and torso should be carefully monitored, anteroposterior and lateral chest radiographs in an upright position, and have cardiopulmonary and ventilatory function monitored. It is not uncommon to identify radiologic evidence of pulmonary contusion in a child who, other than having a history of blunt torso trauma, is completely asymptomatic. Cognizance of this potential mandates continuous careful monitoring of fluid status.

Finally, the possibility of cardiac contusion in children sustaining blunt thoracic trauma must be considered. Any child who has sustained a high impact blunt thoracic injury should at least have a comprehensive 12-lead electrocardiogram.

Abdominal Injuries

Because of the increased size of the torso relative to the extremities in the pediatric population, abdominal injuries continue to be an extremely common problem. The presence of blood within the peritoneal cavity does not necessarily mandate immediate laparotomy.[19]

Since the initial description of overwhelming postsplenectomy sepsis in asplenic patients by King and Schumaker[20] (1952), there has been a gradually increasing ground swell among the pediatric surgical community to minimize splenectomy whenever possible. Multiple studies have confirmed successful nonoperative management of splenic injuries, and have most recently documented similar success with hepatic and renal injuries. Obviously, common sense and good clinical judgment must be applied in the management of the child who has sustained blunt abdominal trauma. A child who is unstable on presentation and remains so or a child who requires large volumes of blood transfusion to maintain adequate cardiovascular stability must be explored. Figure 6-2 demonstrates a commonly used protocol initially developed at the Hospital for Sick Children in Toronto that recommends transfusion to almost 50 to 75 percent normal circulating volume prior to the consideration for operative intervention.[9] Repeat careful clinical examination of the abdomen is absolutely mandatory. The presence of abdominal distention, tenderness to palpation, and evolving rebound or guarding are all signs suggestive that either a hemoperitonium or rupture of a hollow viscus may be present. In unstable or suspicious cases, it is clearly better to explore the abdomen than to treat a potentially fatal injury with uncertainty.[21]

Although complex pancreatic or duodenal injuries seem to be somewhat infrequent in the pediatric age group, duodenal perforation can and does occur. A simple radiograph of the abdominal cavity with careful attention to the presence of gas in the retroduodenal area on lateral projection may indicate the possibility of a subtle duodenal injury. The incidence of isolated mid-body pancreatic injuries secondary to falls and blunt trauma is quite significant. The actual traumatic incident causing these types of injuries

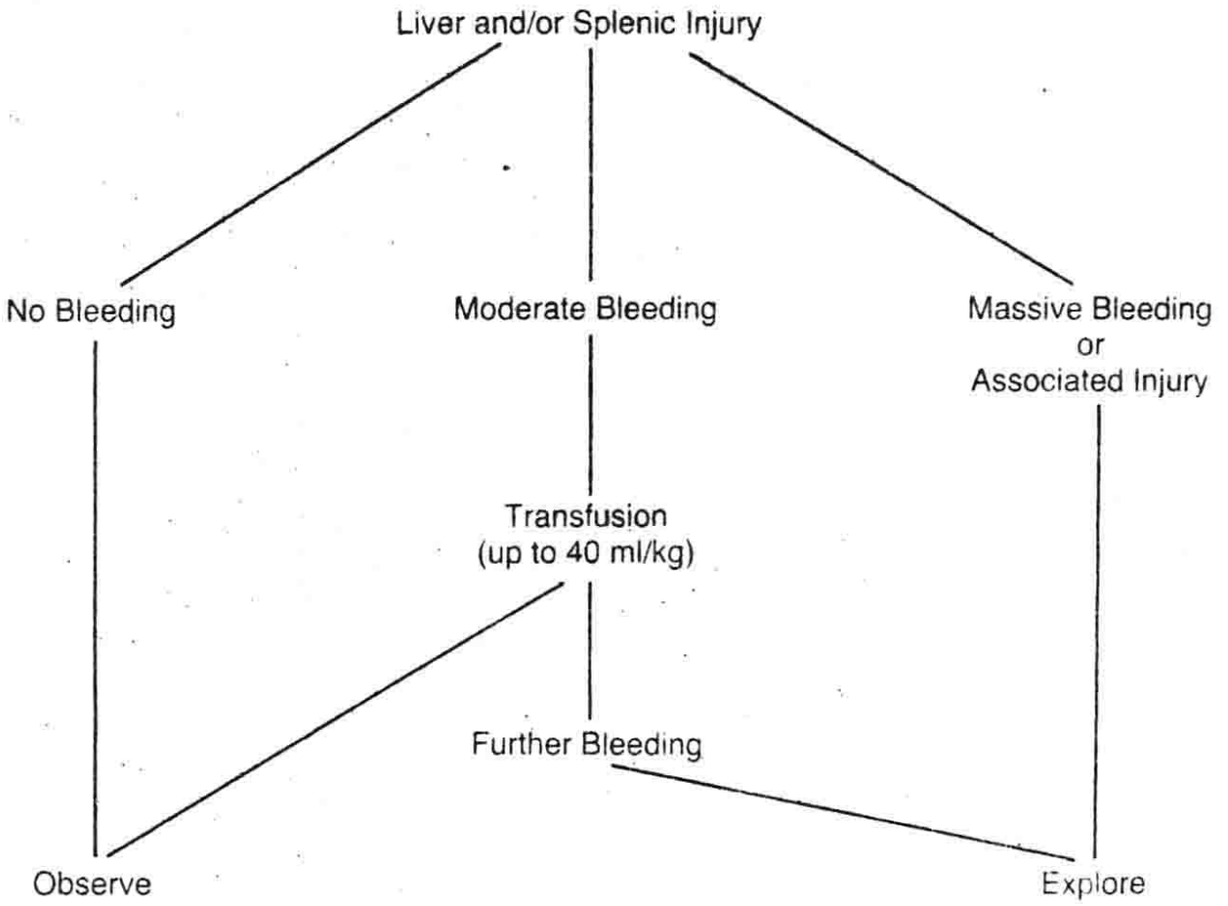


Fig. 6-2 Protocol for management of liver and spleen injuries.

may actually be minor, so a high index of suspicion is essential for successful identification and treatment of these injuries. The abdominal CT scan is an extremely sensitive tool for assessment of the status of the pancreas and can be effective in identifying these injuries. Adequate therapy of these lesions requires operative intervention and in most cases resection of the segment of pancreas distal to the area of disruption. Since this is usually the mid-portion of the body of the pancreas as it overlies the vertebral column, a distral subtotal pancreatectomy without splenectomy is frequently the procedure of choice in treatment of this injury.

The development of peritonitis secondary to perforation of a hollow viscus is both an immediate and long-term possibility. Children who are injured wearing seatbelts in an incorrect manner frequently sustain an avulsion injury, in which part of the distal small bowel is torn from its underlying mesentery. This, in combination with a direct contusive injury to the abdomen, may cause immediate blowout of the involved segment of gut with purulent fecal peritonitis developing shortly thereafter. It may, however, cause a simple area of devascularization and ischemia that will progress to necrosis and perforation within 48 to 72 hours postinjury. Therefore, any child sustaining significant blunt abdominal trauma must be carefully observed for this possibility and explored immediately should signs or symptoms of acute abdominal distress suddenly develop.

Extremity Trauma

In comparison to the adult, the child's skeleton is incompletely calcified and consists of a high degree of cartilagenous tissue and metabolically active growth plates. The collateral ligamentous structure binding this skeleton is frequently stronger and better able to withstand mechanical disruption than are the bony elements to which it is attached. As a result, children sustaining skeletal trauma frequently sustain major deforming forces prior to development of fractures or disruptions of their bony skeleton. Primary joint disruption from other than penetrating injury is uncommon in comparison with disruption of the diaphyseal or epiphyseal segments of bone. Fractures involving the growth plate must be carefully identified and treated in a manner that will ensure not only adequate healing but prevention of subsequent displacement or deformity.

The association of vascular injuries with orthopedic injuries must always be excluded in the pediatric population. Besides the obvious injury to the brachial artery secondary to suprachondylar fracture of the humerus, the possibility of minor vascular disruptions causing growth plate ischemia or major vascular disruption causing low-flow or hypoprofusion states must be considered. Since the pediatric population does not usually suffer from advanced atherosclerosis, mechanical forces will temporarily deform rather than disrupt major vessels. The adventitia and media of the child's arterial system can well tolerate this transient deformity and immediately return to a normal anatomic state. The intima of these vessels, however, will frequently fracture as a result of this stretching deformity and, upon return to the normal anatomic position, develop an intimal flap that will impede vascular flow. Rather than gross disruption of the vessel with hypovolemia and hemorrhagic shock, a child sustaining blunt vascular injury either primarily or in association with an orthopedic injury may sustain diminution of flow as a result of a silent intimal flap. Here again, a high index of suspicion and careful assessment of the distal vascular runoff of the injured child must be routinely considered. When there is the slightest suggestion of decrease in perfusion, arteriography should be considered. While the natural history of these lesions remains unclear, there is no question that a major disruption of blood flow as the result of an intimal flap within the aorta may well warrant at least careful observation, if not immediate operative correction.

Long-term Follow-up

According to Gallagher and co-workers,[22] for every child who dies of traumatic injury, 45 others require admission and intensive hospital care and 1,300 additional children require at least assessment and therapy in the emergency room. It becomes obvious that the effect of traumatic injury on our pediatric population is extensive. Unfortunately, long-term follow-up of injured children has focused primarily on that small segment with severe

injury who have identifiable CNS dysfunction requiring prolonged rehabilitation. In reality, a variety of other injuries, some of which are survivable, are being poorly followed and yet are providing ongoing morbidity and health care problems for these children. Careful follow-up must be provided for all injured children specifically in regard to developmental anomalies and propensity for repeat injuries. Centers providing care for the injured child and persons committed to this end should ensure that all children to whom care is provided are given careful long-term scrutiny, so that the effects of injury can be analyzed, with the hope that their detrimental results can be minimized. This type of program should focus on subtle residual deficits in CNS function as well as psychological residual and the identification of accident-prone children.

In conclusion, initial and definitive care of the injured child requires careful application of the standard trauma life-support principles to the unique characteristics of this patient population. An understanding of the types of injuries that frequently affect children, the mechanical forces at play when children are injured, and the unique characteristics of the child as a patient will most likely lead to a level of care that will be expeditious, effective, comprehensive, and successful. Achievement of this goal will provide not only a tremendous sense of personal satisfaction but will also be a major step forward in diminishing the most common cause of death and disability in our pediatric population.

REFERENCES

1. National Safety Council: Accident Facts—1984 Edition. National Safety Council, Chicago
2. Cales RH, Trunky DD: Preventable trauma deaths. JAMA 245:1059, 1985
3. Rimel R, Geordani B, Barth J, et al: Disability caused by minor head injury. J Neurosurg 9:221, 1981
4. Plant M, Gifford R: Trivial head trauma and its consequences in a perspective of regional health care. Milit Med 141:244, 1976
5. Luten R: Pediatric resuscitation chart and equipment shelf: Aids to mastery of age-related problems. J Emerg Med 4:9, 1986
6. Fleisher G, Templeton J, Delgado-Paredes C: Fluid resuscitation following liver laceration: A comparison of fluid delivery above and below the diaphragm in a pediatric animal model. Ann Emerg Med 16:147, 1987
7. Walker ML, Starrs B, Mayer TA: Head Injuries. p. 272. In Mayer, T (ed): Emergency Management of Pediatric Trauma. WB Saunders, Philadelphia, 1985
8. Rivara FP, Parish RA, Mueller BA: Extremity injuries in children: Predictive value of clinical findings. Pediatrics 78:803, 1986
9. Wesson DE, Filler RM, Ein SH, et al: Ruptured spleen—When to operate. J Pediatr Surg 16:324, 1981
10. Karp MP, Cooney DR, Pros GA, et al: The nonoperative management of pediatric hepatic trauma. J Pediatr Surg 18:512, 1983
11. Polley TZ, Coran AC: Special problems in management of pediatric trauma. Crit Care Clin 2:775, 1986

12. Bryant MS, Tepas JJ, Mollitt DL, Talbert JL: Impact of Emergency Room laboratory studies on the ultimate triage and disposition of the injured child. South Med J In press
13. Ramenofsky MD, Luterman A, Quindlen E, et al: Maximum survival in pediatric trauma: The ideal system. J Trauma 24:818, 1984
14. Stablein DM, Miller JD, Choi SC, et al: Statistical methods for determining prognosis in severe head injury. Neurosurgery 6:243, 1980
15. Champion HR, Sacco WJ: Measurement of injury severity and its practical application. Trauma 1:25, 1984
16. Tepas JJ, Ramenofsky ML, Mollitt DL, et al: The pediatric trauma score as a predictor of injury severity: An objective assessment. J Pediatr Surg 22(1):14, 1987
17. Kraus JF, Fife D, Cox P, et al: Incidence, severity and external causes of pediatric brain injury. Am J 140:687, 1986
18. Overgaard J, Tweed WA: Cerebral circulation after head injury. J Neurosurg 41:531, 1974
19. Douglas GJ, Simpson JS: The conservative management of splenic trauma. J Pediatr Surg 6:545, 1971
20. King H, Schumaker HB: Susceptibility to infection after splenectomy performed in injury. Ann Surg 136:239, 1952
21. Feleciano DV, Mattox KL, Jordan GL, et al: Management of 1000 consecutive cases of hepatic trauma (1979–1984). Ann Surg 204:438, 1986
22. Gallagher SS, Finison K, Guyer B, Goodenough S: Incidence of injuries among 87,000 Massachusetts children and adolescents. Am J Public Health 74:1340, 1984

7 Problems in Minor Trauma Care

Robert Schafermeyer

Any experienced parent or emergency physician who cares for children of all ages knows of the great variability in each encounter. The encounter can be terrifying for the child and frustrating for parents and health care providers. Some children are braver than others and tolerate the minor injury, pain, and any approach used. Others are calm and inquisitive. Still others demonstrate fear—of pain, punishment, or the unknown. This is the child one must approach in a special way to help overcome fears and anxiety.

The emergency physician should not rush in and try to look at the injury as the first order of business. It is important to be calm and unhurried, regardless of whether treatment is for an abrasion, laceration, burn, or other minor traumatic event. Just a few minutes are necessary at the start to gain the child's confidence and obtain essential history about the child from the parents. Once the child's confidence has been gained, the injured part can be examined, slowly and carefully.

Checking the neuromuscular function of an injured or lacerated part can be a major undertaking or a test of ingenuity. Using a toy, light, paper clip, or cotton is not as threatening as a pin or needle. Games that imitate motions or that involve forming letters with the fingers are useful in assessing the child's motor function. How a child plays with a toy, pen, or penlight will provide more information than will commands or forced manipulations.

The emergency physician should honestly explain the procedure to the child, whether it involves debridement, suturing, or manipulation. Lights,

soap, drapes, and needles can be frightening to the child. Explanations should continue as the desired procedures are carried out. An aide or parent can help distract the child by discussing an area of the child's special interest with the child. The child should be allowed to cry or scream when it hurts but to try not to move the injured part.

Age alone does not predict which child will cooperate or which one is in such a panic that the child cannot be quieted long enough to hear what the physician has to say, let alone cooperate in the examination process; some 2-year-olds cooperate better than 14-year-olds. Some form of physical restraint is usually needed for children under age 2 for the repair process. Papoose boards or wrapping the child with a sheet can be used to make it easier for an assistant or parent to hold the head or other injured part still; children aged 2 to 5 years should be given a chance to cooperate without physical restraints. The use of hypnosis may be desirable in this age range, provided one is skilled in its methods.[1] The last resort should be chemical sedation. Most soft tissue injuries do not require chemical sedation, since topical agents can be used for relief of pain; furthermore, the risk of overdosage and other adverse reactions could convert a simple injury into a tragic event. However, there are times when an unusually frightened or very uncooperative child needs sedation. Choices include Nitronox (50 percent O_2–50 percent N_2O mixture), meperidine and hydroxyzine, meperidine and secobarbital, or chloral hydrate (Table 7-1). Maximal doses are used for effect, and naloxone and emergency airway equipment must be available at all times. The child must be observed in the emergency department for 4 to 6 hours, until the sedation has worn off and the risk of respiratory arrest has passed. Complex injuries, even in cooperative patients, may be handled in a safer and more humane fashion in the operating room with general anesthesia. If this route is chosen, it is well to remember the maxim: A child's stomach is always full.[2]

One should not forget to check beyond the local wound or injury. Associated injuries should always be considered. Just as one considers a dislocated hip or vertebral body fracture with a calcaneal fracture, there are combination injuries to be considered in the child. For a lacerated chin, it is important to check for dental occlusion and injury to the temporomandibular joints. If the primary dentition has been injured, the child should see a dentist to check the secondary dentition. When a facial or scalp laceration is noted, it is crucial to look at and palpate the wound for fractures. All lacera-

Table 7-1. Sedation Dosages

Agent	Dosage
Meperidine	1 mg/kg IM
Hydroxyzine	1 mg/kg IM
Meperadine	1 mg/kg IM
Secobarbital	3 mg/kg IM
Chloral hydrate	50 mg/kg (1 g max) PO or rectal

tions should be checked visually for retained foreign bodies and debris. If a wound was caused by a piece of glass, the neuromuscular function must be evaluated carefully; occasionally the wound must be enlarged so that it can be properly explored. In many cases, a more serious tendon or nerve injury lies below an innocent half-centimeter surface wound. If a tooth is missing, the wound should be checked first, and a determination made as to the need for a chest-abdomen radiograph to check for aspiration of the tooth. Consideration should also be given to the possibility of significant internal injury or intracranial injury, if the history suggests that significant force or energy was involved in the accident. For example, a 3-cm scalp laceration may have to be repaired in a child who tripped and hit a coffee table or in one who fell off the top of a slide in the playground. In the latter case there is a greater chance of intracranial injury, for which the child should be thorough examined. Clearly, the approach to the child should be calm, gentle, and thorough.

Children heal faster than adults, since atherosclerotic disease, hypertension, and diabetes are almost nonexistent. Oxygenation of their tissues is excellent and, given proper care, injured parts heal rapidly. However, there are children with chronic disease states, hematologic, and immunologic problems mandating meticulous cleansing and care of their injuries and close observation. For those who are susceptible to wound and skin infections, the wounds might be debrided and cleansed more thoroughly and short-term prophylactic antibiotics considered. The same holds true for children on subacute bacterial endocarditis (SBE) prophylaxis. The hemophiliac patient may require Factor 8 or 9 and close attention to hemostasis in skin wounds and compartment syndrome in contusions of even a mild nature.

One other consideration in the traumatized child is whether the problem might be due to child abuse or neglect. Improper supervision and abnormal delays in seeking medical care may be signs of neglect. Physical abuse may be indicated by a contradictory history or a history inconsistent with the physical findings. Physical findings of multiple soft tissue bruises over the soft parts of the body at various stages of healing, bruises that resemble objects found at home (i.e., belt marks, electric cord loops, burns that take on a pattern of a hot object), and multiple fractures especially of ribs, scapula, and vertebrae should raise suspicion of abuse[3] (Table 7-2). In addition, some injuries are unlikely to occur at younger ages (i.e., in a 6-month-old with burns only on the hands and feet or perineum). All states have mandatory reporting laws, and it is up to the physician to consider abuse when the history or physical findings support such a suspicion.[3]

SPECIFIC MINOR TRAUMAS

The following discussion addresses specific clinical conditions commonly presented in the emergency department. Table 7-3 delineates the potential problems inherent in these conditions, as well as the likely result if they are improperly handled.

Table 7-2. Soft Tissue Injuries Caused by Abuse

- Integument injuries
  - Pattern marks (buckle imprint)
    - Linear whip marks
    - Loop marks (electric cord)
    - Hand prints
    - Punctures (fork marks)
    - Binding of wrist or ankles
  - Multiple bruises
  - Bruises in various stages of healing
  - Unusual location
    - Central distribution
    - Protected areas (neck)
  - Human bite
  - Hair loss
- Burns
  - Pattern burn (cigarette, iron)
  - Immersion burn
    - Stocking or glove distribution
    - Buttocks and feet pattern
  - Splash burn
- Injury to sensory organs
  - Ear
    - Boxed ear
    - Hemotympanium
    - Cauliflower ear
  - Eye
    - Periorbital ecchymosis
    - Conjunctival hemorrhage
    - Corneal abrasion
    - Hyphema
    - Dislocated lens
    - Vitreous hemorrhage
    - Retinal detachment
    - Retinal hemorrhage
  - Nose
    - Epistaxis
    - Fracture
  - Mouth
    - Lip trauma
    - Frenulum tear, "bottle jamming"
    - Posterior pharyngeal trauma
    - Airway obstruction
- Timing of healing—Bruises
  - Red/blue: first 24 hr
  - Dark purple: 1–4 days
  - Green: 5–7 days
  - Yellow: 7–10 days
  - Normal tint: 1–3 weeks

(Ludwig S: Pediatric Emergencies. Churchill Livingstone, New York, 1985.)

Table 7-3. Problems in Minor Trauma Care

Problem	Potential Result
Abrasions	
Failure to cleanse adequately	Infection
Use of adhering dressings	Pain with resultant lack of compliance with instructions
Bites	
Failure to differentiate types of bites	Inappropriate treatment and complications of same
Failure to address tetanus status	Risk of tetanus
Failure to address rabies potential	Risk of rabies
Puncture Wounds	
Underestimation of significance	Severe infections
Attempts of foreign-body retrieval	Pain; unnecessary extension of wound size; excessive morbidity
Lacerations	
Misuse of anesthesia	Complications of anesthesia
Failure to address allergies	Potential of anaphylaxis
Failure of good infiltrative technique	Infection, pain
Incorrect dosage of lidocaine	Potential with small children and large doses for cause of toxicity
Misuse of anesthesia with epinephrine	Ischemic necrosis
Improper suture selection	Infection and other foreign-body complications
Failure to consider special areas	Missed injuries
Closing of oral mucosa for puncture wounds	Infection
Failure to consider special goals of finger injuries	Loss of function; sensitivity; painful scar
Minor burns	
Considering burns as minor	Loss of rapport with patient and parents, with resultant lack of cooperation and compliance
Failure to consider CO poisoning	CO complications
Failure to consider special burns	Sequelae of inadequate treatment causing poor function or cosmesis
Inadequate pain control	Pain, lack of cooperation, potential additional latrogenic injury
Misuse of antibiotics	Lack of infection control
Dental injuries	
Failure to consider age	Loss of viable permanent tooth
Failure of adequate follow-up	Incomplete treatment
Permanent teeth	
Failure to replace avulsed tooth	Loss of tooth; malalignment
Failure to search for "missing" tooth	Potential loss of viable tooth; aspiration of foreign body
Failure to refer fracture involving pulp	Loss of tooth; infection
Poor technique in replacing tooth	Loss of viable tooth

Abrasions

Improper handling of abrasions can lead to infection or tatooing. Because children heal rapidly, it is generally sufficient to cleanse abrasions adequately and use a nonadhering dressing that can be changed everyday. The wound should be gently cleansed daily. Deeper abrasions may be treated as a skin graft donor site with cleansing and a fine-mesh gauze dressing. Bits of gravel, rock, dirt, or tar must be removed to avoid infection or tatooing of the wound. For large areas of debridement, general anesthesia will permit proper debridement. For smaller areas, a topical anesthesia agent may be useful, such as TAC solution [topical adrenaline (epinephrine) and cocaine] or gauze soaked in 2 percent lidocaine (Xylocaine). A stiff brush, scalpel, scissors, pick ups, and a pulsating jet lavage unit should suffice for equipment needs.

Bites

Animal bites may be closed with special considerations. Puncture wounds should not be closed, as they cannot be adequately debrided. Other lacerations should be anesthetized, irrigated, sharply debrided, and closed in standard fashion. The use of antibiotics for prophylactic coverage is controversial. Some authorities believe that no antibiotics are needed, while others suggest the use of penicillin or a cephalosporin for 2 to 3 days. Bites to the lower extremities tend to heal more slowly and are more susceptible to infection.[2] Rabies prophylaxis should be considered on a case-by-case basis based on Centers for Disease Control (CDC) and local guidelines[4] (Table 7-4). Human bites should not be closed but rather debrided thoroughly, irrigated, and given local wound care. Most physicians use short-term penicillin or cephalosporin coverage during the healing process.

Puncture Wounds

Many people consider a puncture wound to be a minor but painful experience. In reality it has potential to escalate into a major problem. Cellulitis, closed-space infections (especially palmar and plantar spaces), abscesses, retained foreign bodies, and osteomyelitis can occur. The first premise for puncture wound care should be to do no harm. The second premise should be adequate local debridement and wound care with careful follow-up. Consider whether joint spaces or bone penetration may have occurred. Radiographic examination may reveal radiopaque foreign bodies. Most glass is radiopaque. The radiograph may also demonstrate air in a joint space.

Osteomyeletis has been reported with puncture wounds especially through tennis shoes. Some puncture wounds must be opened and debrided and antibiotics given for 3 to 5 days. Retrieving the foreign body is important to prevent infection and further injury. However, many a physician has palpated a needle edge and confidently attempted to get a "needle in the

Table 7-4. CDC Rabies Postexposure Prophylaxis Guide—July 1984[a]

Animal Species	Condition of Animal at Time of Attack	Treatment of Exposed Person[b]
Domestic		
Dog and cat	Healthy and available for 10 days of observation	None, unless animal develops rabies[c]
	Rabid or suspected rabid	RIG[d] and HDCV
	Unknown (escaped)	Consult public health officials. If treatment is indicated, give RIG[d] and HDCV
Wild		
Skunk, bat, fox, coyote, raccoon, bobcat, and other carnivores	Regard as rabid unless proved negative by laboratory tests[e]	RIG[d] and HDCV
Other		
Livestock, rodents, and lagomorphs (rabbits and hares)	Consider individually. Local and state public health officials should be consulted on questions about the need for rabies prophylaxis. Bites inflicted by squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, mice, other rodents, rabbits, and hares almost never call for antirabies prophylaxis.	

[a] These recommendations are only a guide. In applying them, the animal species involved, the circumstances of the bite or other exposure, the vaccination status of the animal, and presence of rabies in the region should be taken into account. Local or state public health officials should be consulted if questions arise about the need for rabies prophylaxis.

[b] *All bites and wounds should immediately be thoroughly cleansed with soap and water.* If antirabies treatment is indicated, both rabies immune globulin (RIG) and human diploid cell rabies vaccine (HDCV) should be given as soon as possible, *regardless* of the interval from exposure. Local reactions to vaccines are common and do not contraindicate continuing treatment. The vaccine should be discontinued if fluorescent-antibody tests of the animal are negative.

[c] During the usual holding period of 10 days, treatment is begun with RIG and HDCV at first sign of rabies in a dog or cat that has bitten someone. The symptomatic animal should be killed immediately and tested.

[d] If RIG is not available, antirabies serum, equine (ARS) should be used, in not more than the recommended dosage.

[e] The animal should be killed and tested as soon as possible. Holding for observation is not recommended.

(Rabies Prevention—United States, 1984. MMWR 33:393, 1984.)

haystack" and been unable to readily locate the needle. It may be better to refer the deep extremity foreign bodies to an orthopedist or general surgeon. (Markers or 25-gauge marking needles inserted under fluoroscopy at 90-degree angle to each other will improve your odds of retrieving the foreign body.) Retained foreign bodies may cause infection, nodular granulomas, chronic granulomatous lesions, and pilonidal sinuses and may even migrate from the point of entry.[5] Tetanus prophylaxis is a must for puncture wound since they favor the growth of anaerobic organisms in closed tissue spaces[6] (Table 7-5).

Table 7-5. Tetanus Prophylaxis

History of Tetanus Immunization (Dose)	Clean Minor Wounds		All Other Wounds	
	Td[b]	TIG[c]	Td[b]	TIG[c]
Uncertain	Yes	No	Yes	Yes
0–1	Yes	No	Yes	Yes
2	Yes	No	Yes	No[d]
≥3	No[e]	No	No[f]	No

[a] Refer to text on specific vaccines or toxoids for contraindications, precautions, dosages, side effects and adverse reactions, and special considerations. Important details are in the text and ACIP recommendation (MMWR 1981;30:392–407).

[b] The combined preparations Td, containing both tetanus and diphtheria toxoids, is preferred to tetanus toxoid alone.

[c] Tetanus immune globulin.

[d] Yes, if wound more than 24 hours old.

[e] Yes, if more than 10 years since last dose.

[f] Yes, if more than 5 years since last dose (more frequent boosters are not needed and can accentuate side effects.)

(Adult Immunization Supplement MMWR 33(15): 685, 1984.)

Lacerations

Lacerations provide the bulk of soft tissue injuries next to contusions in the emergency department setting. Lacerations are more than skin deep. Occult injuries do occur. The face, scalp, and hands seem to be the most common sites of injury. All lacerations must be properly examined, cleansed, thoroughly explored for deeper injuries, closed, and have proper follow-up before one can be satisfied with the repair. Transections of nerves or tendons may be apparent only under proper visualization. Thus, approach to the child must be handled carefully as outlined previously. Use of one's ingenuity to test motor function plus direct exploration of the wound should prevent missing injury to deeper structures. Occasionally, general anesthesia will be needed for proper examination and repair. The risks of general anesthesia must be weighed against the benefits of the examination and repair.

TREATMENT

The approach is crucial if one is to secure the confidence of the child. Children are not small adults; they may have fears out of proportion to the injury. These fears can be exacerbated by a hurried approach, brusk manner, bright lights shining in their eyes, or drapes covering their eyes, nose,

and mouth. One should approach the child slowly and confidently and talk to him first. Once the child's confidence has been won, explain what must be done to get him repaired and on his way home. To check neurovascular function, one just needs to use one's ingenuity by using some of the methods cited previously.

Anesthesia

Next, one must consider what type of anesthesia to maintain the child's cooperation. Does one use a topical anesthetic, local infiltration, Nitronox, or general anesthesia? What does one do for the "Novacaine" allergic patient? If the child is suggestible and not in a panic state, you may wish to use hypnosis if skilled in its application. Others may opt to use a topical agent to numb the skin edges while preparing the suture tray. One can take a 2 × 2 and saturate it with 5 ml of 2 percent Xylocaine and have the parent hold the gauze pad on the wound for 5 to 10 minutes. Supplemental infiltration may be needed.

TAC is useful for small lacerations, but is it safe? One could use TAC solution (a mixture of tetracaine 0.5 percent, epinephrine 1 : 2000, and cocaine 11.8 percent in unit doses of 5 ml). White et al.[7] noted that TAC solutions were useful on facial lacerations with minimal to no complications. Schaffer[7a] also noted that TAC solution was more effective than tetracaine alone and erythema of wound margins in 7 percent of cases without progression to frank infection as the only complication. Pryor et al.[8] noted that there was no difference in TAC versus lidocaine infiltration with efficacy and total wound complications. He noted a shorter time needed for repair. Nichols et al.[9] evaluated success of anesthesia, length of wound, and complications prospectively in 125 cases and found that wounds anywhere on the body other than pinna, nasal alae, digits, or of limited circulation were successfully anesthesized in approximately 75 percent of patients if 3 cm or less in length. There was only 2 percent induration and 0 percent infection incident. However, Barker et al.[10] noted increased infection in guinea pigs and rabbits. Wehner, et al.[11] noted that even minimal usage of 10 ml or less of TAC solution in burn patients led to active seizures and respiratory arrest. Thus, one would not want to use TAC in wounds that would permit rapid absorption of drug into the circulation such as burns or large abrasions.

If local infiltrative anesthesia is elected, which is still the gold standard, the wound should be infiltrated slowly through the open wound edges (unless highly contaminated) with a 1-inch 25- or 27-gauge needle.[2] Rapid instillation may shorten the painful experience at the cost of markedly increasing the pain and burning sensation associated with lidocaine. In small children and in very large lacerations, one must pay attention to total dose delivered over time as several children had had adverse CNS stimulation and or seizures from excess dosage. An occasional child has had a respiratory arrest (Table 7-6). Local anesthetics with epinephrine can be

Table 7-6. Local Anesthetics

Factor	Procaine	Lidocaine	Tetracaine[c]	Bupivacaine[c]	Mepivacaine
Concentration for infiltration[a]	1%	0.5–1%	1%	0.25%	1%
Onset of action[b]	5–15 min	5–10 min	10–20 min	10–20 min	5–10 min
Duration of action[b]	45–60 min	30–60 min	1.5/2–3 hr	2–4 hr	1–3 hr
Dosage	7 mg/kg	5 mg/kg	1.5 mg/kg	2 mg/kg	7 mg/kg
Group	Ester	Amide	PABA	Amide	Amide

[a] Recommended concentrations for children.
[b] Onset and duration varies from source to source.
[c] Not recommended for children under 12 years of age.

used to prolong the anesthetic effect. However, in small infants, one may see systemic effects. In addition, one would avoid its use where end digital arteries supply the tissues such as fingers, toes, tip of the nose, and pinnal of the ears.[3]

What should be done if the parents say the child is allergic to Novacaine or lidocaine? There are two major groups of local anesthetics, and it is rare to have cross-reactions to these agents. Procaine is an ester type anesthetic while lidocaine is an amide-type agent. Bupivacaine is an amide type, as is prilocaine and tetracaine is a *p*-aminobenzoic acid agent.

One might consider the use of Nitronox for anesthesia by self-administration. Nitronox will provide enough analgesia to repair lacerations or debride large abrasions. However, one must be skilled in its use and in airway management. The person administering the agent must be able to intervene and protect the airway from aspiration. Nitronox is a 50 : 50 mixture and relatively safe as compared with wall or tank sources of N_2O and O_2 which must be adjusted individually.[12,13]

Cleansing

Once the wound is adequately anesthetized, thorough cleansing and debridement of devitalized tissue may be completed. The wound should be explored to its depths searching for foreign bodies and injury to the deeper tissues. Crushed and jagged edges should be trimmed.

Sutures

Selection of suture material and needle are determined by wound depth and location. Absorbable sutures such as polyglycolic acid suture is used for closure of subcutaneous tissues and ligation of small blood vessels. Nonabsorbable monofilament sutures such as nylon are useful for skin closure. A

reverse cutting needle is a good choice for skin closure, since it has a lesser tendency to cut out of the tissues, is a stronger needle yet penetrates the tissue with less trauma.

The suture itself should not be stronger than the tissue it will hold together. 4-0 suture is frequently used in the subcutaneous tissues and for the skin in general; 5-0 nylon suture is useful for the hand and 6-0 nylon suture for the face.[14] The depth of the suture should equal the distance between the sutures, with the wound edges approximated or everted slightly. If the edges invert, one may need to undermine the tissues or use a vertical mattress suture in selected places.

OTHER INJURIES

Areas of special consideration in the pediatric patient include oral lacerations and fingertips. Children frequently fall with foreign objects in or near their mouths and subsequently have puncture wounds to the oral mucosa, tongue, and soft palate. The wounds must be properly and thoroughly examined for associated injuries and, unless bleeding is persistent or there is a significantly placed lesion, the wound does not need to be closed. If there may be injury to the nasopharynx, retropharynx, or Stenson's duct, the wounds should be evaluated under general anesthesia. If a child falls with the end of a spoon or pointed object in his mouth, consider pharyngeal performation and its possible sequelae if the retropharyngeal space was violated. If there is palpable crepitus, drooling, or dysphagia, endoscopy is indicated.[2]

If there is a through-and-through laceration of the lip, the wound should be irrigated and debrided thoroughly, the muscle layer closed, and then the skin layer (loosely), and the mucosal side left open, if small. If the mucosal wound is large or bleeding briskly, two or three sutures may be used to decrease the size of the defect and stop the bleeding. Penicillin is frequently given for several days due to the high bacterial content of the oropharynx. If the vermillion border is crossed by the laceration, it must be aligned exactly since a 1-mm mismatch is visible. The border area should be the first stitch.[13]

Injuries to the fingers of children are very common and the basic goals of therapy are to preserve length and function, have fingertip sensitivity, and a pain-free scar.[15] The age of the child and condition of the nail bed often affect decisions as to type of repair. There may be simple lacerations, flap lacerations and complete amputations of tissue. Simple lacerations are cleaned and repaired with 5-0 nylon after appropriate examination. If the nail is involved one must check the nail bed for laceration and accurately approximate the tissues for good results. Often that necessitates removal of part or all of the nail to close the nail bed properly. The nail can then be used as a protective splint.[16]

Tip lacerations that create a flap may be closed after proper cleansing

as if they were a graft. 5-0 nylon approximates the edges and a firm occlusive dressing should be left in place for 7 days. A second dressing can be left for 7 more days before sutures are removed. Tip amputations, involving less than 1 cm tissue, will heal by secondary intention. The wound should be cleaned and frequent dressing changes should occur down to a fine-mesh gauze layer.[15,17]

With avulsions or amputations of more tha 1 cm tissue, one may need to consult with a hand surgeon. Some hand surgeons would allow an infant or toddler to heal by secondary intention down to a level with bone tuft exposed. Others would use some other method of closure. Once the amputation level moves below the mid-shaft of the distal phalanx, special procedures will be necessary.

If one is having difficulty evaluating a wound on the fingers due to excessive bleeding, rather than blindly clamping, one should take a ¼- to ½-inch diameter penrose drain and apply it around the base of the finger. This will provide a dry field for completion of the evaluation. The tourniquet may be left in place for 5 to 10 minutes.

Crush injuries involve tremendous forces to an area of the body and result in celular and vascular disruption in both the superficial and deep tissues. Swelling worsens with time and necrosis of tissues is common but frequently not apparent for several days. After proper evaluation of neurovascular status, crush injuries are best treated with loose dressings and elevation if only one to two fingers are involved. If the hand or extremity is involved, the child should be referred immediately to a hand or orthopedic surgeon since there is great risk of developing a compartment syndrome or necrosis of the deep muscles.

Glass injury to the fingers and hands always appears innocent and requires a very careful and complete neurovascular assessment and includes wound exploration. If a tendon laceration is uncovered, prompt consultation with a hand surgeon or orthopedist is important.

Proper evaluation, cleansing, and repair would not be complete or guarantee excellent results unless follow-up care is ensured. The parents must be properly instructed verbally and have a written copy of the guidelines for a reference. The parents are frequently distraught and may not remember what was said once they get home. They should keep the wounds clean and dry. If no dressing is applied, the parents should be instructed in gentle cleansing with hydrogen peroxide or betadine solution. Suture removal varies literally from head to toe with facial sutures left in place for 3 to 5 days and possibly supported with benzoin and steri-strips for 1 week and lower leg and foot sutures left in place for 8 to 14 days (Table 7-7).

MINOR BURNS

To a child, no burn is minor. Most burns can be prevented by a few

Table 7-7. Suture and Suture Removal

Site	Needle	Days
Face	6-0 nylon P-3	3–5
Scalp	4-0 nylon FS2	7
Trunk	4-0 nylon FS2	7–10
Extremity	4-0 nylon FS2	8–14
Hand and foot	5-0 nylon P3	8–14

simple measures at home. The child's play area should be supervised and matches locked up. Water heater temperature should be reduced to 120°F instead of 140°F. Children should be kept away from kerosene and wood stoves and out of the kitchen while someone is cooking. Most burns are scald burns. At 140°F, it only takes 3 to 4 seconds to get a second-degree burn.

When evaluating a child with burns, the ABCs should be checked first and any necessary airway, ventilation, or circulatory resuscitative maneuvers performed. One must then consider any possible associated injuries such as those due to blast, smoke inhalation, or a fall. One should also consider whether carbon monoxide poisoning has occured.

Burn patients can then be divided into two groups—major and minor. Minor burns include superficial, less than 10 percent dermal, and less than 1 percent subdermal. Burns of the face, neck, perineum, hands, and feet are considered major burns for cosmetic or functional reasons. If touch sensation is absent, the burn most likely is subdermal.

Basic care of the minor burn includes analgesia, debridement, dressings, and fluids. Acetomenophen, soothing lotion, and cool compresses may be all that are needed for first-degree burns while a shot of meperidine and hydroxyzine may be needed for deep dermal burns. All loose skin should be gently trimmed away, and gentle saline cleansing of the burned area completes the debridement. Small burns of the first-degree type and small burns about the face are usually treated without dressings. Silvadene or another antiobiotic cream may be applied three times daily with warm water cleansing between applications. Deeper burns less than 9 percent dermal may be treated with a fine-mesh gauze such as Zeroform and a light Kerlex gauze wrap over it. The Kerlex gauze is changed daily. The wound should be checked every 2 to 3 days for any signs of infection. Antibiotics are not recommended in minor wounds unless signs of infection are present or if one needs to provide SBE prophylaxis to a patient.

Fluids become more important the smaller the child and the larger and deeper the burn area. A child with a deep dermal burn of 8 percent can lose a

Table 7-8. Maintenance Fluids

100 ml/kg for the first 10 kg
50 ml/kg for the next 10 kg
20 ml/kg for kg over 20

moderate amount of fluid. The parent should see that the child drinks the normal amount plus an additional amount of fluid to maintain good urine output. An infant or toddler who goes more than 8 hours without wetting the diaper should be rechecked. The amount of fluid can be calculated as follows:

Maintenance plus 4 ml × weight in kg × percent burn
given over 24 hours

DENTAL INJURIES

Injuries to the teeth are common in childhood and cause anxiety for the patients and parents. Although the physician is frequently the initial provider, most dental injuries ultimately will be resolved by the dentist. Delay in seeking dental follow-up may adversely affect the ultimate prognosis. Injuries can be divided into two major categories: primary teeth, with peak periods between ages 2 and 5 years; and permanent teeth, with peak periods between ages 8 and 12 years. Injuries are subdivided into coronal and root fractures, displacement injuries, and avulsion. The goals of treatment are preservation of the teeth and the dental arch, proper occlusion, and proper development of the maxilla and mandible.[18]

Displacement is the most common injury in the primary dentition; it usually occurs in the maxillary incisors and less frequently in the mandibular incisors. Displacement may cause disruption of the neurovascular supply, with subsequent pulp necrosis and apical abscess formation. More important, it may damage the unerupted permanent teeth. Minimally displaced primary teeth should be repositioned after local anesthesia and splinted if necessary, unless the tooth is near exfoliation or pathologically involved. Dental consultation and follow-up are indicated for grossly displaced or intruded primary teeth, which are often extracted.

Primary coronal fractures involving only the enamel are smoothed and kept under observation. If the fracture involves dentin or the pulp, or both, the patient should be referred to the dentist for pulpal therapy and/or crown restoration. Root fractures may necessitate extraction, with follow-up dental treatment.

Avulsions are more likely to occur as physiologic root resorption occurs. Avulsed primary teeth should not be reimplanted.[18] Although rare, if bleeding occurs following avulsion or extraction, a rinse should be used initially, followed by direct application of pressure for 20 minutes.

It is important to see the injured tooth. If unable to do so, a radiograph should be obtained to rule out intrusion, retained fragments, or soft tissue impaction. A chest radiograph may be indicated as well to survey for aspiration of a tooth.[19]

Injuries to Permanent Teeth

Protusive maxillary incisors ("buck teeth") are the most commonly injured permanent teeth; this injury occurs most often in boys aged 8 to 12 years. Crown fracture is the most common injury. Because sports are a factor in dental injuries, parents and coaches should insist on the use of protective appliances. Because malocclusion predisposes the patient to dental injuries, early orthodontic treatment and preventive measure will reduce the incidence of tooth injuries.

Coronal fractures may involve enamel only; enamel and dentin; or enamel, dentin, and the pulp. Treatment of coronal fractures involving enamel only consists of smoothing the rough edges, radiographs to check for root fractures, and watchful waiting by the dentist to determine pulpal vitality. If loss of contact with the adjacent teeth occurs, dental restoration is important. When enamel and dentin are involved, a slightly off-white area and temperature sensitivity usually are present. It is important to protect the exposed dentin. Referral to the dentist for adequate pulpal protection and restoration of the tooth is essential. As a temporary measure to reduce thermal sensitivity, a layer of zinc oxide paste may be placed after drying the tooth.[7]

Coronal fractures of permanent teeth involving the pulp must be referred for immediate dental treatment. Pulpal exposure with subsequent pulpal necrosis presents difficult problems in younger children. Root formation is not completed until 2 to 4 years following eruption of the permanent tooth. Although root canal treatment can be performed, the long-term prognosis is diminished because of the shorter root. Therefore, tooth fractures involving the pulp should be seen by a dentist as soon as possible.

Displacement of permanent teeth is common. When seeing the patient, the physician should reduce the tooth digitally, if possible, and stop the bleeding with direct pressure. Dental wax can be used as a temporary splint until the patient sees a dentist. The wax is molded into a U shape and applied over the teeth. Dentists use direct bonding of acrylic resins to teeth for splinting. Splinting times are 1 to 2 weeks for tooth injuries and up to 6 to 8 weeks for bony fractures. Root canal therapy is required in approximately one-half of displaced teeth; therefore, routine dental follow-up to check for pulpal necrosis is essential. Intruded teeth must have root canal treatment to eliminate inflammatory root resorption.

Avulsion of permanent teeth is a true emergency as the successful reimplantation is inversely proportional to the length of extraoral time. Ideally, all avulsed teeth should be immediately reimplanted at the sight of the injury. If dirty, the tooth should be rinsed gently with water or saline to remove debris avoiding injury to the periodontal membrane remnants; the tooth should then be reimplanted gently in the socket. If clotting has occurred in the socket, the clot is removed by gentle saline irrigation. The tooth is repositioned as well as possible. Dental wax can be used as a tempo-

rary splint, until dental care can be obtained. If unable to reimplant the tooth, it should be placed in the space between the gum and cheeks. If the patient is too young and swallowing or aspiration are a danger, the tooth should be placed in a glass of milk, physiologic saline, or water to keep the tooth moist. Immediate dental care should be sought.[20]

Teeth reimplanted within less than 30 minutes after avulsion have a good prognosis for long-term retention. All reimplanted teeth undergo some root resorption. Reimplantation may be achieved even after several days; however, more extensive root resorption and ankylosis will occur if the extraoral time exceeds 2 hours. Retention of the permanent teeth is preferable to even the best prosthesis. All avulsed teeth that have fully formed roots must have the pulp removed during the first week following replantation to avoid inflammatory root resorption. Therefore, dental referral is mandatory following avulsion. It is important to institute tetanus prophylaxis.

Root fractures are best treated by reduction of the fractured segment and placement of a splint. Dental splinting with acid etch resin techniques are required to immobilize the coronal segment for 6 to 12 weeks. Union of the segments may occur if reduction is achieved before blood clotting occurs and the fracture is intrabony following reduction. Root fractures that communicate with the oral cavity following reduction will not heal, and the coronal segment must be extracted. If the root is long enough, root extrusion may be achieved orthodontically and the tooth restored. Dental consultation is mandatory.

CONCLUSION

Minor trauma can be a major event for any child. Compassion and honesty are crucial in the approach to the child. Ingenuity in examination and evaluating function are important to identify subtle neuromuscular injuries. Checking for retained foreign bodies and evaluation for associated injuries is essential. Attention to details of anesthesia, cleansing, and repair should result in a good result and a happy child.

REFERENCES

1. Olness K: Hypnosis in pediatric practice. Monograph Current Problems in Pediatrics Vol 12 No 5, 1981
2. Seashore JH: Soft tissue injuries. p. 219. In Touloukian RM (ed): Pediatric Trauma. John Wiley & Sons, New York, 1978
3. Ludwig S: Pediatric Emergencies. Churchill Livingstone, New York, 1985
4. Rabies Prevention—United States, 1984. MMWR 33(28):393, 1984
5. Scott MJ Jr, Scott MJ III: Baffling skin reactions to foreign body fragments. Consultant Vol 26 No 11, 1986

6. Adult immunizations. MMWR 33(15):15, 1984
7. White WB, Iserson KV, Criss E: Topical anesthesia for laceration repair: Tetracaine versus TAC. Am J Emerg Med 4(4):319, 1986
7a. Schaffer DJ: Clinical comparison of TAC anesthetic solutions with and without cocaine. Ann Emerg Med 14(11):1077, 1985
8. Pryor GJ, Kilpatrick WR, Opp DR: Local anesthesia in minor lacerations. Topical TAC vs lidocaine infiltration. Ann Emerg Med 9(11):568, 1980
9. Nichols FC III, Mucha P Jr, Fornell MB: TAC topical anesthetic and minor skin lacerations. Resident Staff Physician 33(2):59, 1987
10. Barker W, Rodeheaver GT, Edgerton MT, et al: Damage to tissue defenses by a topical anesthetic agent. Ann Emerg Med 11:307, 1982
11. Wehner D, Hamilton GC: Seizures following topical application of local anesthetics to burn patients. Ann Emerg Med 13(6)456, 1984
12. Crone RK: Nitrons oxide, oxygen sedation for minor surgery. Petiatr Alert 6:57, 1981
13. Templeton JJ Jr, Ziegler MM: Minor trauma and minor lesions. p. 800. In Fleisher G, Ludwig S (eds): Pediatric Emergency Medicine. Williams & Wilkins, Baltimore, 1983
14. Markovchick VJ, Cantrill SV: Soft tissue injuries. p. 397. In Barkin RM, Rosen P: Emergency Pediatrics. CV Mosby, St Louis, 1984
15. Finseth, F: The injured hand. p. 549. In Touloukian RM (ed): Pediatric Trauma. John Wiley & Sons, New York, 1978
16. The American Society for the Surgery of the Hand: The Hand Examination and Diagnosis. Society, 1983
17. Lindsay WK: Hand injuries. p. 59. In Care for the Injured Child. Williams & Wilkins, Baltimore, 1975
19. Andreasen JO: Traumatic Injuries of the Teeth, 2nd Ed. WB Saunders, Philadelphia, 1981
20. Fountain SB, Camp JH: Traumatic injuries. p. 493. In Cohen S, Burns R (eds): Pathways of the Pulp. 3rd Ed. CV Mosby, St. Louis, 1984

8 Problems in Managing Cervical Spine Injuries

Fred Tecklenburg

A spinal cord lesion in a pediatric patient is a devastating injury for the child, family, and society. The child suffers a neuromuscular disability with multiple attendant medical, rehabilitative, and psychological problems. The family is often shattered emotionally and economically as the lifelong struggle to support the unfortunate child unfolds. Our society also bears the economic cost of spinal cord injuries; approximately $4 billion is expended annually on this problem.[1]

It is understandable that prehospital EMS personnel and emergency physicians have adopted "airway with cervical spine control" as the standard first priority in trauma resuscitation.[2] A significant percentage of spinal cord injuries may be incomplete at the moment of trauma and, therefore, have potential for neurologic recovery with appropriate early management.[3] Cervical spine immobilization and radiographic evaluation have been liberally applied in the trauma management of adult and pediatric patients, often without an appreciation for the multiple differences between the two age groups.

This chapter explores relevant data on pediatric cervical spine injuries, anatomic and biomechanical properties unique to the child's spine, and common pitfalls in the assessment and management of suspected cervical trauma in children.

EPIDEMIOLOGY OF CERVICAL SPINE INJURIES

The annual incidence of traumatic spinal cord injury in this country has been estimated to be 30 to 50 per million population or, in absolute numbers, approximately 11,000 new cases each year.[4–7] Children under 16 years of age account for roughly 6 to 9 percent of these debilitating cord lesions.[4,8,9] Spinal cord injury is primarily a disease of adolescence and young adulthood. The National Spinal Cord Injury Data Research Center (NSCIDRC) reported the data on 5,912 spinal cord injury patients referred to Regional SCI Systems throughout the United States.[10] This group represented approximately 10 percent of the total spinal cord injury patients in the United States during the years the data were collected. Nearly one-half the spinal cord injuries occurred in the 15- to 24-year-old age group. The mode, or most common age, was 19 years, while the median, or 50th percentile, was 24 years old. Only 3.8 percent of the referred patients in this group were under 15 years of age.

These incidence figures and statistics do not include either deaths due to spinal injuries at the scene or on arrival to emergency departments or the tens of thousands of spinal column injuries not associated with cord lesions. Although these latter statistics are not available, several studies suggest some generalizations. First, spinal cord lesions have a high mortality and may be underestimated as a cause of acute traumatic death. One of the first thorough epidemiologic studies on spinal cord injury documented 619 cases over 2 years (1970 to 1971) in northern California via elaborate protocol autopsies and medical record searches.[6] There were 299 deaths, yielding a case mortality rate of 48 percent. Death at the scene or on emergency department arrival accounted for 79 percent of the mortality, with most autopsy reports incriminating the spinal cord lesion as the primary cause of death. Forty-four patients of the 619 cases (7 percent) were under 15 years of age and, as an age group, they suffered a 66 percent mortality, compared with a 37 percent mortality in the 15- to 24-year-old group. Other postmortem studies of consecutive traffic accident victims have demonstrated cervical injuries in 21 to 24 percent by stress radiographic view or autopsy dissection.[11,12] The ages of the victims were not revealed, but two facts have implications for pediatric patients: (1) auto–pedestrian victims were disproportionately represented in the cervical lesion groups, and (2) atlanto-occipital and C1-C2 injuries accounted for the vast majority of lesions. Young children actually die more frequently as pedestrians than as motor vehicle occupants[13]; they have a predisposition to upper cervical lesions. How these facts interrelate is left to conjecture without further study, but suffice it to say that cervical spine injuries are probably underreported in acute traffic accident deaths in children.

Another generalization regarding the incidence of pediatric cervical spine injuries is that most cervical column (ligamentous-osseous) injuries do not lead to neurologic deficits. An analysis of several series of pediatric cervical spine injuries illustrates this point.[8,14–17] (Table 8-1). Approximately one-third of radiographically positive cervical spine lesions will in-

Table 8-1. Cervical Spine Injuries in Pediatric Series

Investigators	Age (years)	Cervical Spine Osseous/Ligamentous	
		Neurologic Deficit[a]	Without deficit
Hill et al.[8]	2–18	27 (36)	47
Anderson and Schultz[14]	0–14	26 (42)	61
Apple et al.[15]	1–16	11 (38)	18
Henrys et al.[16]	0–14	7 (38)	11
Hubbard[17]	1–17	3 (25)	9
		74 (34)	146

[a] Numbers in parentheses represent percentage.

volve a neurologic deficit. These studies emanated from referral institutions and are probably biased toward more severe injuries. Although acute cervical spine injuries are not common in children, an accurate estimate of all significant cervical spine injuries in children or adults has not been reported. A crude extrapolation of the foregoing would be that 6 percent of 11,000 spinal cord injuries are in pediatric patients, and these lesions would represent roughly one-third of the total pediatric cervical spine injuries demonstrable by radiography—or approximately 2,000 cases occur annually.

The etiology of cervical spine injury in children does not differ substantially from adult series. The major categories in both age groups remain motor vehicle accidents, falls, diving accidents and, in urban areas, penetrating injuries. The young child seems more susceptible to auto–pedestrian accidents and falls than the teenager or young adult. In adolescence, motor vehicle accidents, athletic injuries (especially diving injuries), and gunshot wounds play a larger role. The United States is unique among Western countries in the proportion of spinal cord injuries due to penetrating wounds (typically gunshot wounds). NSCIDRC data[10] reveal penetrating injury as the second most common cause of spinal cord injury in the 0 to 14-year-old group (Table 8-2). Several other mechanisms of injury in children and adults warrant mention because of their disproportionate responsibility for

Table 8-2. Etiology and Age

	Age			
	0–14		15–29	
	N	%	N	%
MVA/Pedestrian	91	40	1,938	52
Penetrating	54	23	508	14
Sports—diving	32	14	467	12
Falls	27	12	394	11
Sports—other	19	8	240	6
Other	7	3	204	5

spinal cord injuries. Motorcycles accounted for nearly 15 percent of spinal cord lesions due to motorized vehicles.[10] One of every 14 occupants ejected from their cars suffers a neck injury.[18] Football and trampoline injuries lead the list of athletic related injuries, although the trampoline has steadily decreased as a cause of cervical trauma as its use has fallen into disfavor.[19]

An uncommon mechanism of injury unique to pediatrics is spinal cord injury associated with parturition. Typically this injury occurs in the neonate with breech presentation and with intrauterine hyperextension of the neck—"the star-gazing neonate."[20] Shulman et al.[21] demonstrated 25 cases of cervical spinal trauma or vertebral artery disruption in 192 stillborn and neonatal deaths.

Finally, another unusual etiology of spinal injuries specific for children is violent shaking by abusive parents.[22-24] There may be minimal outward signs of abuse on examination, but frequently retinal hemorrhages and intracranial pathology are evident on further examination or radiologic workup.

ANATOMIC AND BIOMECHANICAL CONSIDERATION

The anatomic and biomechanical properties of the child's cervical spine will yield radiographic appearances and injury patterns different from those of the adult. The pediatric spine exhibits hypermobility, unique vertebral configurations, epiphyseal growth plates, synchondroses, incomplete ossification, and numerous normal variations and congenital anomalies. This developing anatomy will dictate the radiologic appearance and influence the pathophysiology of injuries.[25-28]

Although the details of the developmental anatomy are beyond the scope of this chapter, several facts regarding ossification are necessary for recognizing the normal appearance of the atlanto-axial segment at different ages (Fig. 8-1 and 8-2). The atlas usually has three ossification centers: two centers are present at birth, with each forming a lateral mass and neural arch, which then extend posteriorly to unite the other arch at 3 to 4 years of age; the third ossification center appears at 1 year of age and forms the anterior arch, which fuses with the lateral masses by 8 years.

The axis has five primary ossification centers. The odontoid process, which is phylogenetically the body of the atlas, has two centers, which fuse in fetal life and form the base of the dens. An apical secondary growth center (ossiculum terminale) appears at 2 to 3 years and fuses to the odontoid by 12 years. The other primary centers form the body and neural arches of the axis and are present at birth. An epiphyseal growth plate separates the odontoid from the axis and appears as a lucent horizontal line for the first 4 or 5 years. Closure of this epiphysis is variable with delayed closure up to 12 years of life and vestigial remnants lasting for life in some adults. Fractures of the odontoid in childhood generally occur at this growth plate with anterior odontoid displacement.

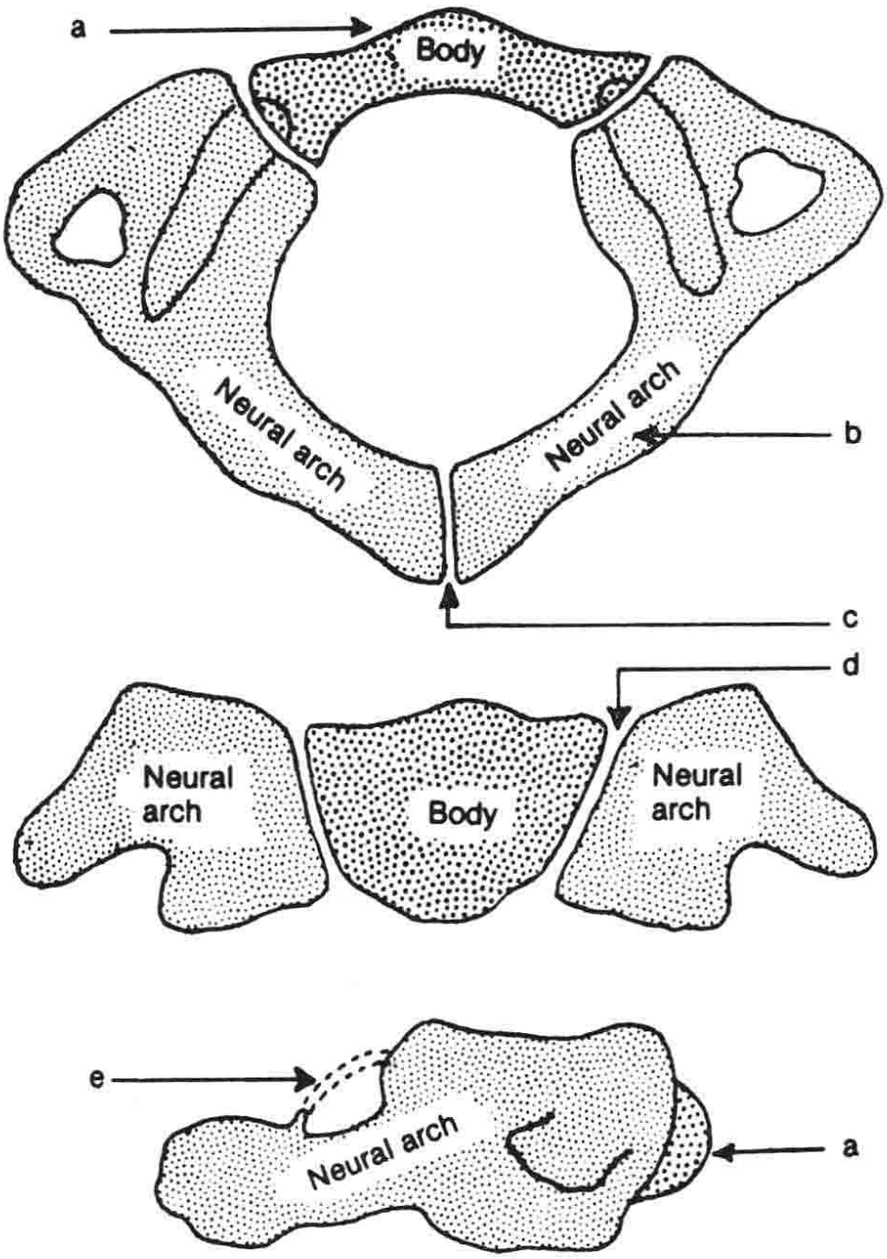


Fig. 8-1 Diagram of first cervical vertebra (atlas). a, Body. Not ossified at birth center (occasionally two centers) appears during first year after birth; body may fail to develop and forward extension of neural arches may take its place. b, Neural arches. Appear bilaterally about seventh fetal week; most anterior portion of superior articulating surface is usually formed by the body. c, Synchondrosis of spinous processes. Unite by third year. Union may rarely be preceded by the appearance of a secondary center within the synchondrosis. d, Neurocentral synchondrosis. Fuses about the seventh year. e, Ligament surrounding the superior vertebral notch. May ossify, especially in later life. (Bailey DK: The normal cervical spine in infants and children. Radiology 59:712, 1952.)

Ligamentous laxity and increased joint mobility are hallmarks of the musculoskeletal system in the neonate and young child. This hypermobility allows dissipation of traumatic forces and generally serves the child well during childbirth and the later adventurous and physically active behavior of childhood. Any casual observer in a busy pediatric emergency department has noted the paucity of true sprains or dislocations in the very young

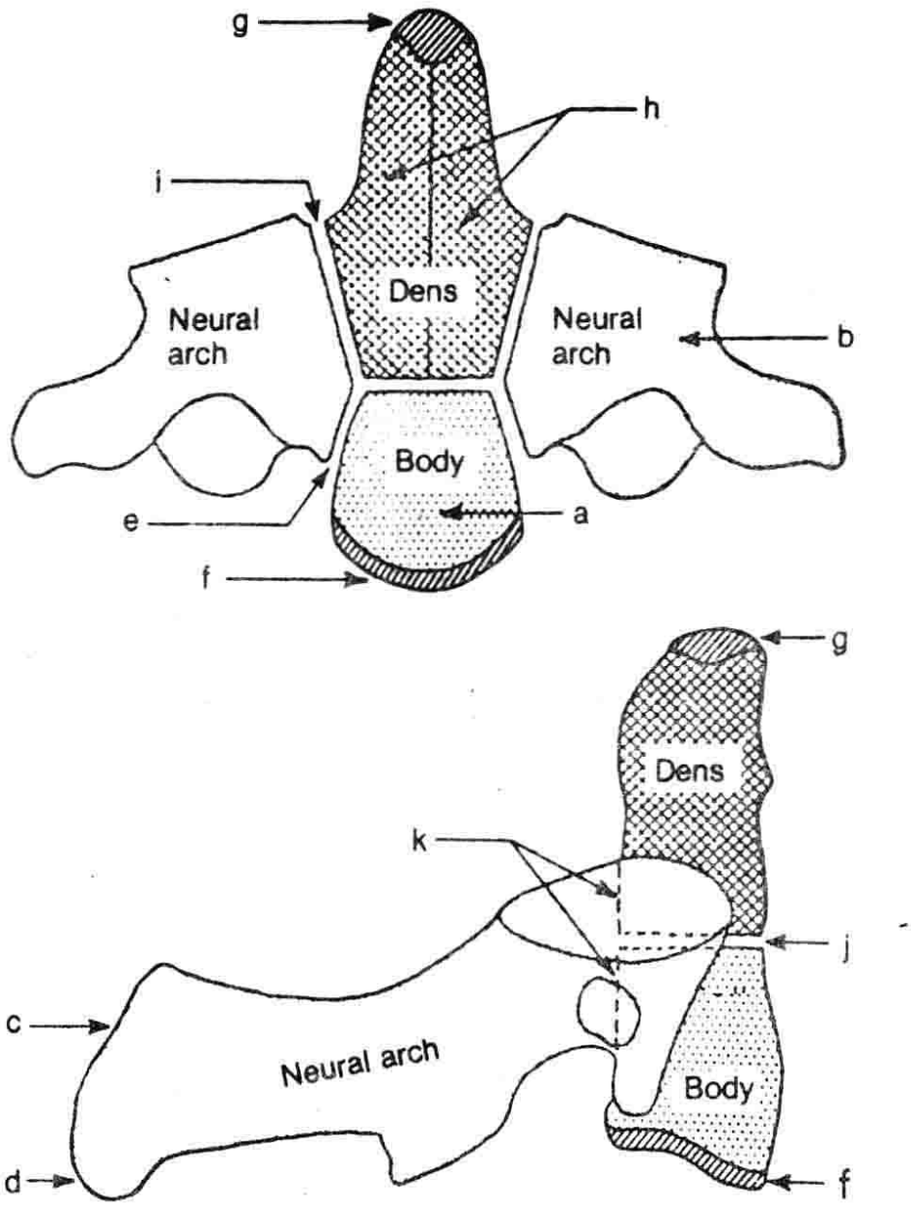


Fig. 8-2 Diagram of second cervical vertebra (axis or epistropheus). a, Body. One center (occasionally two) appears by the fifth fetal month. b, Neural arches. Appear bilaterally by the seventh fetal month. c, Neural arches fuse posteriorly by second or third years. d, Bifid tip of spinous process (occasionally a secondary center is present in each tip). e, Neurocentral synchondrosis. Fuses at 3 to 6 years. f, Inferior epiphyseal ring. Appears at puberty and fuses at about 25 years. g, Summit ossification center for odontoid. Appears at 3 to 6 years and fuses with odontoid by 12 years. h, Odontoid (dens). Two separate centers appear by the fifth fetal month and fuse with each other by seventh fetal month. i, Synchondrosis between odontoid and neural arch. Fuses at 3 to 6 years. j, Synchondrosis between odontoid and body. Fuses at 3 to 6 years. k, Posterior surface of body and odontoid. (Bailey DK: The normal cervical spine in infants and children. Radiology 59:712, 1952.)

child's joints. The child's cervical spine is no exception to this general rule and, in fact, might be considered the epitome of the flexible pediatric joint.

The pediatric atlanto-axial joint has relatively lax ligaments allowing major anterior-posterior excursion of the atlas during flexion and extension. Further laxity of the transverse ligament is occasionally found in patients

with Down's syndrome, Morquio's disease, and rheumatoid arthritis leading to clinically significant atlanto-axial instability.[27] This fact is the rationale for the American Academy of Pediatric recommendation that all children with Down's syndrome participating in Special Olympic activities be screened with dynamic cervical radiography.[29] Even in normal children, minimal trauma or the local inflammation of a pharyngitis causing increased atlanto-axial ligamentous laxity may lead to rotatory subluxation of C1 on C2.[30-32] Rotation is the predominant normal motion between the atlas and axis, with the alar ligaments preventing excessive rotation. It has been demonstrated that in the setting of a 5-mm anterior displacement of the atlas on the axis, unilateral C1 to C2 dislocation can occur at 45-degree rotation.[30] As the child matures, the transverse ligament, the primary stabilizer of the atlanto-axial joint, will prevent excessive anterior shift of the atlas on the axis.

The other cervical levels in children also have lax ligaments and elastic joint capsules, but several other anatomic factors contribute to the pediatric spinal column's flexibility. In the upper cervical spine (C2 to C4), the anterior portion of the vertebral bodies are wedge shaped and the facet joints are in a relatively horizontal plane. The child's vertebrae have no joints of Luschka in the first decade. These physical properties allow some degree of physiologic subluxation in C2 to C4 on flexion in 20 percent of children,[28] and they also give the young child a higher fulcrum of anterior-posterior motion. Pennecot et al.[33] demonstrated that children 3- to 8-years old have greater anterior-posterior mobility at C2 to C3 to C4 than at C5 to C6 to C7, although with increasing age the upper cervical spine mobility decreases. The most mobile joint space, or greatest angular displacement, is at C3 to C4 for 3- to 8-year-olds, C4 to C5 for 9- to 11-year-olds, and C5 to C6 for 12- to 15-year-olds.

Hypermobility, however, does not always protect the skeleton or spinal cord. In fact, most traumatic pathology in pediatric cervical injuries occurs at the atlanto-axial level and upper cervical levels, the areas of greatest flexibility.[16,17,34] The combination of a relatively large and heavy head, weak neck musculature, and hypermobile upper cervical spine may predispose the child's spine to injuries from accelerative and torsional stresses.[35] The occipito-atlanto-axial segment seems especially vulnerable to torsional stresses. For example, in the unfortunate newborn with difficult breech presentation or forceps extraction, transection of the cord, atlanto-occipital and atlanto-axial dislocations, and odontoid fractures have been reported. The rotational acceleration and resultant shear stress in the shaken baby syndrome have also produced similar injuries.

Most cervical lesions in children under 8 years of age have been odontoid fractures, atlanto-axial dislocation or subluxations, and hyperextension fractures of the axis (the "hangman's" fracture). The mechanisms of injury are similar to adults, but the traumatic lesions remain centered in the atlanto-axial region. Adolescents and adults tend to have pancervical lesions and a greater percentage of neurologic deficits. The large diameter of

the spinal canal at C1 probably accounts for this discrepancy in neurologic sequelae. Steel's rule of 3's states the spinal cord at the atlas is equally occupied by the spinal cord, odontoid process, and free space.[36] Anterior displacement of the atlas on the axis past a distance equal to the AP diameter of the odontoid places the cord at risk. Since odontoid fractures are the most common osseous injury in young children, it follows that many of these patients may be salvaged without neurologic disability. Table 8-3 separates the available data from the previously cited series into two age groups, demonstrating a lower percentage of neurologic deficits in children under 9 years of age.

It is not surprising that the spinal cord in a hypermobile spine may suffer damage without fractures or dislocations of the spinal column. Some pediatric series have reported a significant percentage of spinal lesions without evidence of osseous or ligamentous pathology.[34,37–39] The young child's long neuronal pathways are poorly myelinated and may be more vulnerable to the effects of shear stress. Pang and Wilberger[38] reported a series of 24 children with spinal cord injury and no radiographic abnormality. These patients represented 67 percent of all nonpenetrating spinal injuries treated at their institution during the study period. Fourteen of the children were under 8 years of age and, as a group, had many more neurologic deficits than the older group. Thirteen of the younger 14 patients had complete transections or severe central cord syndrome compared with only one child of the older group with a severe deficit. Interestingly, 54 percent of all the patients had delayed neurologic deterioration ranging from 30 minutes to 4 days. It was suspected that flexion compression forces were responsible for the more severe lesions in the younger children. The traumatic myelopathy occurred at C1 to C4 in most complete lesions in the younger patients. However, the younger patients also had less severe injuries at C5 to C8—the area in which all but one older children incurred their lesions. Hyperextension, longitudinal distraction, and ischemia also probably played a role in the pathogenesis of some of these lesions. These data are difficult to correlate with the outcome of osseous-ligamentous injuries described in Table 8-3, but a reasonable conclusion is that the upper cervical spine is inherently predisposed to injury in the young pediatric patient and hypermobility places the spinal cord at risk. Further study is need to delineate the biomechanics of injury in the pediatric spine.

Table 8-3. Cervical Spine Osseous-Ligamentous Injuries

	< 9 years old		9–18 years old	
Investigators	Neurodeficit	Without	Neurodeficit	Without
Hill et al.[8]	0	7	27	40
Hubbard[17]	0	4	3	5
Henrys et al.[16]	2	7	5	4
Apple et al.[15]	2	3	9	15
	4 (16%)[a]	21	44 (41%)[a]	64

[a] Percentage of patients with neurologic deficit in specified age group.

PREHOSPITAL CARE

Prehospital care involves the initial assessment and resuscitation, immobilization, extrication, and transport to an emergency facility.[40] The goal is to maintain adequate cardiopulmonary function and, in the case of cervical spine injuries, to avoid any secondary mechanical or vascular disruption of the central nervous system (CNS). There have been multiple reports that 3 to 25 percent of cervical spinal cord injuries occur after the traumatic event during the early emergency care.[3,40–43] The possibility that poor prehospital technique may lead to a lifelong neurologic disability has led to the liberal application of "cervical spine precautions" in the field.

Any traumatic event associated with multiple trauma, altered mental status, or complaints of neck pain mandate cervical spine immobilization of the patient. Many emergency systems also immobilize the cervical spine of the asymptomatic patient when the forces involved in the traumatic event are recognized as potentially life-threatening; the justifiable tendency is to always err on the side of immobilization.

The skilled prehospital provider addresses the airway and cervical spine immobilization simultaneously. Movement from a prone to supine position is performed with a log-rolling maneuver, with one person maintaining head and neck neutrality. Airway patency is ensured and oxygen always supplied in the patient with altered sensorium or serious injuries. (The difficult airway and problems with ventilation are addressed in the emergency department management.) Cervical spine neutrality should be maintained with gentle in-line traction by an assistant as a semirigid collar is applied. Depending on the circumstances of extrication, the patient is eventually placed on a spine board with the rescuer at the head still guarding the cervical neutrality. The patient is then immobilized to the spine board with tape on the forehead and sandbags alongside the head and neck, tape on the collar, and secure binding or strapping of the trunk to the board. Several important points and potential pitfalls in management are noteworthy:

1. Soft cervical collars provide minimal, if any, immobilization.[41,44]
2. A semirigid cervical collar provides only a variable degree of immobilization.[41,44] It is an adjunct to cervical spine immobilization.
3. Taping and sandbags on a spine board are the most reliable technique of immobilization[41] (in studies based on adult patients).
4. It is better to improvise than use ill-fitting collars. Oversized collars do not immobilize and, more dangerously, may block the child's airway. Fitting children less than 5 or 6 years old with standard semirigid collars is difficult, but at least one company is in the process of developing a collar for infants and toddlers.[45] A nice improvisation for an infant is the child's car restraint with rolls of toilet paper at the sides of the head and a towel for a neck roll.
5. The patient should be sufficiently immobilized on the back board so that the entire board may be log-rolled in the event of emesis.

Other questions arise in the field regarding cervical immobilization. Should a crying and fighting child be restrained? I believe, in most cases, no. Generally patients who vigorously resist attempts to immobilize the cervical spine are only going to mobilize the spine more in the fight over the restraints. Children usually acknowledge their musculoskeletal injuries by not moving the affected part; it is unlikely that a child would resist strongly if a cervical spine lesion is present. Young children are not going to be intoxicated with ethanol or other analgesics that make the signs and symptoms of cervical spine injury more elusive in adolescents and adults. On the other hand, every reasonable effort should be made to gently immobilize the crying child who obviously had a stiff neck after trauma. If the head is tilted to one direction and the child resists gentle motion toward neutrality, the spine should be immobilized in the original titled position and transported.

Another common question in the field is should a helmet be removed? There is really no need to remove a helmet if the airway is accessible and there is no extensive scalp bleeding. Aprahamian et al.[46] demonstrated worrisome mobility of a cervical injury in a cadaver model utilizing standard two person removal. Many sports trainers carry bolt cutters and razors to remove the face masks on football helmets.[47] The helmet can be removed in the emergency department after cervical spine assessment with a cast remover cutting in a coronal plane. If the helmet must be removed in the field, a second rescuer should apply mandibular and occipital stabilization and traction as the first rescuer carefully removes the helmet.[48]

In the long run, common sense often dictates the best course to take in the prehospital setting. For instance, a teenager found in a pool should be removed with a spine board and cervical spine precautions. However, the 2-year-old found in the same pool needs to be rapidly scooped out for more effective cardiopulmonary resuscitation. The remainder of the prehospital care for children with possible cervical spine injury is dictated by the standard trauma management guidelines.

EMERGENCY DEPARTMENT

History

As in most patient evaluations, the history is extremely valuable in the assessment of neck trauma and the later formulation of management plans. In the seriously injured patient, a brief and pertinent history is obtained after the assessment and management of life-sustaining functions. The relevant history should detail the mechanism of injury, the delivery of care at the scene, and any prehospital observation, especially changes in neurologic status or cardiorespiratory parameters. Routine questions regarding past medical history, allergies, current medications, and tetanus status should not be neglected in the trauma patient. As time permits, a more detailed description of the force and mechanism of injury should be obtained. The height of a fall, the quality of impact surfaces, the velocity of moving vehi-

cles, and the extent and pattern of vehicular damage will also frequently influence management decisions. In the verbal and oriented child, subjective complaints of head or neck pain, paresthesias, amnesia, or weakness should be elicited. Finally, the history may uncover discrepancies or unlikely circumstances suggestive of child abuse.

Physical Examination

In the critically injured child, the initial assessment takes place with simultaneous resuscitation and should always be prioritized by the acronym:

*A*irway—with cervical spine control
*B*reathing
*C*irculation
*D*isability

The evaluation of the conscious patient with normal vital signs may be more methodic and complete with emphasis on the head, neck, and neurologic examinations. A complete neurologic examination includes evaluation of the mental status, cranial nerves, deep tendon reflexes, muscle tone and strength, sensation, and cerebellar function. It is important to remember that one of the few indications for operative neurosurgical intervention in cervical spine injuries is a deteriorating neurologic exam. It behooves the examiner to do complete exams and to record them serially with time. Motor strength should be scaled numerically, with 0 being no contraction; 1, flicker or trace of contraction; 2, strength that cannot overcome gravity; 3, antigravity strength; 4, active movement against resistance; and 5, normal strength. The sensory examination should also be detailed, including vibratory, positional, and pain stimuli. Testing for pain should be reserved for the end of the examination and should start distal to any suspected complete lesion. Sensory levels should be recorded by anatomic landmarks rather than suspected dermatomes so that serial examinations by different observers will be reliable.

Subtle observations can be diagnostic. Seiman[49] described two children with odontoid fractures who were comfortable in a supine or erect posture but who cried intensely when changing positions unless the head was supported. Another subtle finding may occur in a child with a C5 cord transection who has respiratory efforts with paradoxical chest motion secondary to diaphragmatic motion and no intercostal innervation. The sternocleidomastoid and trapezius are partially innervated by the accessory nerve, and their movement is often mistakenly presumed to be a sign of an intact spinal cord. The child might assume a floppy posture with the hips in a frog-legs or external rotation position. In the same child, a relative bradycardia, mild to moderate hypotension with warm perfused extremities, and priapism would suggest a diagnosis of spinal shock. A complete spinal cord lesion simulates

a sympathectomy, leaving unopposed parasympathetic innervation that produces the signs of spinal shock. Autonomic dysfunction may also lead to a poikilothermic state. A different case presenting with classical physical findings is atlanto-axial rotatory subluxation. Typically, these children present with a painful torticollis after minimal trauma or recent respiratory infection. Their head and neck posture is described as being in a "cock-robin" position: lateral flexion to one side, rotation to the opposite side, and slight flexion.[30–32] In this condition, the elongated sternocleidomastoid is in spasm, as if attempting to correct the deformity, in contrast to spasmodic torticollis (or "wry neck") in which spasm of the shortened sternocleidomastoid is the deforming force. The latter condition is also not as painful, immobilizing, or as recalcitrant to conservative treatment as atlanto-axial rotatary displacement.

Different patterns of neurologic findings should be sought in the exam. For example, an examination that indicates greater motor weakness in the arms than in the legs, loss of bladder function, and varying degrees of sensory deficit is suggestive of a central cord syndrome. The preservation of position and vibratory sensation associated with a complete segmental paralysis and hypesthesia is suggestive of an anterior cord lesion. The importance of spending a couple of extra minutes performing a complete exam cannot be overemphasized. The presence of anal sphincter tone, perianal skin sensation, and bulbocavernosus reflex may be the critically important sign that the acutely paralyzed child has a chance of recovery.

LABORATORY TESTS

Who Requires Radiography?

This is not a question for the child who presents with severe multiple trauma, mental status changes, or complaints of neck pain. It is assumed that this patient has a cervical injury until proved otherwise. The consequences of missing a cervical injury are too catastrophic to act otherwise. However, for many patients who have not suffered such severe injuries, the question of who requires radiographs raises a score of related questions. Does an asymptomatic cervical injury exist in a patient with normal mental status? Can a clinician predict which patients are likely to have a ligamentous/osseous injury? Does every child arriving at the emergency department in cervical spine immobilization require radiographic evaluation? The underlying issue in all these questions is how do we avoid missing a cervical fracture.

The question of whether an occult cervical fracture exists is an important one. Millions of dollars are spent each year searching for one, and thus far the medical literature has not clearly substantiated its existence. On close scrutiny, the frequently cited literature reporting "asymptomatic" cervical lesions do not concern alert patients without neck pain. On the contrary, the reports described patients who complained of neck pain[50,51] or who

had pain demonstrated on physical examination,[51,52] or the report simply referred to patients who were unconscious, inebriated, or suffered multiple injuries.[53] A more recent study[54] concluded painless cervical fractures may exist after reviewing the medical records of 12 patients with cervical fractures who had "no documented complaints of neck pain or stiffness on presentation." Chart review also revealed 10 of these patients had mental status changes secondary to ethanol or trauma, neurologic signs, or major multiple trauma. The remaining two patients had neck pain on readmission to the emergency department, after the injuries were missed by misinterpreted or inadequate radiologic examinations, and the patients were called back. Rather than substantiating the existence of a painless cervical fracture, this study more importantly documented that 10 percent of 67 patients with cervical injury were not initially diagnosed because of difficulties in radiologic interpretation. Supporting the contention that the asymptomatic fracture does not exist, Fischer,[55] in a retrospective study, was unable to document a cervical fracture in 291 pediatric and adult patients without neck pain who were admitted to the hospital for observation of grade 1 head injuries. Five of an additional 42 admitted patients with signs or symptoms of neck injury had cervical spine fractures. Loss of consciousness was reported in 79 percent of the 333 patients, but they were described as alert during the emergency department examination. Intoxicated patients were excluded from this study.

The foregoing seems to discredit the notion that a totally asymptomatic cervical fracture occurs in an alert patient, but what clinical criteria should be used to select the patient for cervical spine radiography? Two recent studies attempted to identify clinical predictors of cervical spine injuries in children. In the first study,[56] 2,133 cervical radiographic studies of pediatric patients over a 6-year period yielded 25 (1.2 percent) cervical fractures. After a chart review of the 25 patients with cervical fractures and 713 randomly selected patients with negative films, it was determined that either a complaint of neck pain or of head injury associated with a motor vehicle accident would have identified all the cases of cervical injury. Using these two criteria would have reduced the number of cervical spine radiographs by 32 percent. Jaffe and colleagues[57] retrospectively reviewed the charts and radiographs of 206 children, 59 of whom had cervical spine injuries. Clinical criteria were developed that would have correctly identified 58 of the 59 children with cervical spine injury and avoided radiographic evaluation of 79 children. The clinical criteria used were abnormal mental status, neck pain, neck tenderness, limitation of neck mobility, abnormalities in reflexes, strength or sensation, and history of direct neck trauma. It was believed that cervical spine immobilization and radiographs should be obtained in any child who has one or more of the clinical criteria. The clinical algorithm had a sensitivity of 98 percent; only one cervical injury was missed, probably due to inadequate information on the medical record. This study had potential biases in the sampling methods, especially considering that 95 percent of children with cervical injuries were referred to the ter-

Table 8-4. Criteria for Cervical Spine Radiography S/P Trauma

One criterion warrants cervical spine radiography
Subjective
Complaint of neck pain
Paresthesias, weakness
Objective
Altered mental status
Tenderness on neck palpation
Decreased range of motion
Abnormal motor or sensory exam
Major multiple trauma
Lower threshold for ordering film
Significant deceleration injuries
Congenital or acquired cervical spine vulnerability
Down's syndrome
Morquio's disease
Klippel-Fiel syndrome
Rheumatoid arthritis
Status post cervical spine operative procedure

tiary institution. Despite the lack of prospective validation, these latter two studies do suggest that the clinician can selectively order cervical films for alert patients after a careful history and physical. In other words, all children who arrive at an emergency department with cervical immobilization in place do not necessarily need cervical radiography. An estimated $140,000,000 is spent each year on cervical spine studies.[58] This enormous expense could be dramatically reduced by a clinical algorithm that has undergone a prospective multicenter trial and maintains a sensitivity of 100 percent. Meantime, it is suggested that when in doubt, radiographs should be ordered. Table 8-4 summarizes the clinical criteria I use. In addition, a low threshold for ordering radiographs must be present in certain high-risk situations, even if the patient is alert and without complaints at that point in time.

Which Views to Order—Or What Views After the Cross-Table Lateral?

There is no question that the cross-table lateral view (CTLV) of the cervical spine is the most reliable view to detect abnormalities. Recent studies in adults, however, have suggested that the CTLV only detects 70 to 80 percent of the cervical skeletal patholgy.[59–61] Blahd et al.[59] found that the CTLV most frequently missed odontoid fractures and C1 to C2 subluxations/fractures because the odontoid, lateral masses, and transverse processes of C1 and C2 were not easily visualized. They also found that the false-negative interpretations of the CTLV were increased in patients older than 35

years, probably due to the degenerative changes, posture, and osteoporosis associated with aging. Another retrospective study of adult patients demonstrated that an anterior-posterior view and open-mouth odontoid view with the CTLV increased the detection of cervical spine abnormalities from 82 percent to 93 percent in 71 patients.[60] Similarly, Shafer and Doris[61] increased the diagnostic yield from 83 percent to 100 percent in 37 adult patients by adding open-mouth odontoid and anterior-posterior views. It is not clear whether these data reflect the pediatric experience. For instance, in their pediatric series, Jaffe et al.[57] noted that the CTLV detected 95 percent of the cervical pathology and 100 percent when it was used in combination with an anterior-posterior view. Again, without a prospective large-scale study for more definitive guidance, the prudent course is to obtain the anterior-posterior and open-mouth odontoid views after the CTLV.

Suspicious findings on the three basic emergency department views—neurologic signs or severe pain and spasm—demand additional views[62] for better definition of cervical anatomy. For example, oblique views will often demonstrate fractures at the pedicles, intervertebral foramina, or lamina and permit examination of facet allignment. Oblique views may be obtained with the patient supine. A pillar view may be helpful in providing visualization of the lower cervical posterior elements. Many authorities advocate thin-section conventional tomography for better definition of suspected or known abnormalities.[62] Computed tomography (CT) can also play a valuable role in identifying abnormalities of the atlanto-axial segment or encroachment of the spinal cord by bone fragments or ruptured discs. Dynamic flexion-extension films are also useful in the diagnosis of ligamentous injury. Pennecot et al.[63] reported eight cases of severe ligamentous injuries in children in whom the diagnosis was delayed for 2 weeks to 4 months. On the first radiologic examinations, there was no cervical lordosis, but otherwise the films were interpreted as normal. When the muscle spasm resolved, but pain continued, the injuries became apparent on active hyperflexion views. Flexion-extension views are also useful in examining the atlanto-axial joint for stability. Finally, in evaluating possible rotatory atlanto-axial subluxation, the diagnosis can be difficult to make on plain film, and lateral fluoroscopy of C1 to C2 during attempted rotation or CT scan may be necessary.[15]

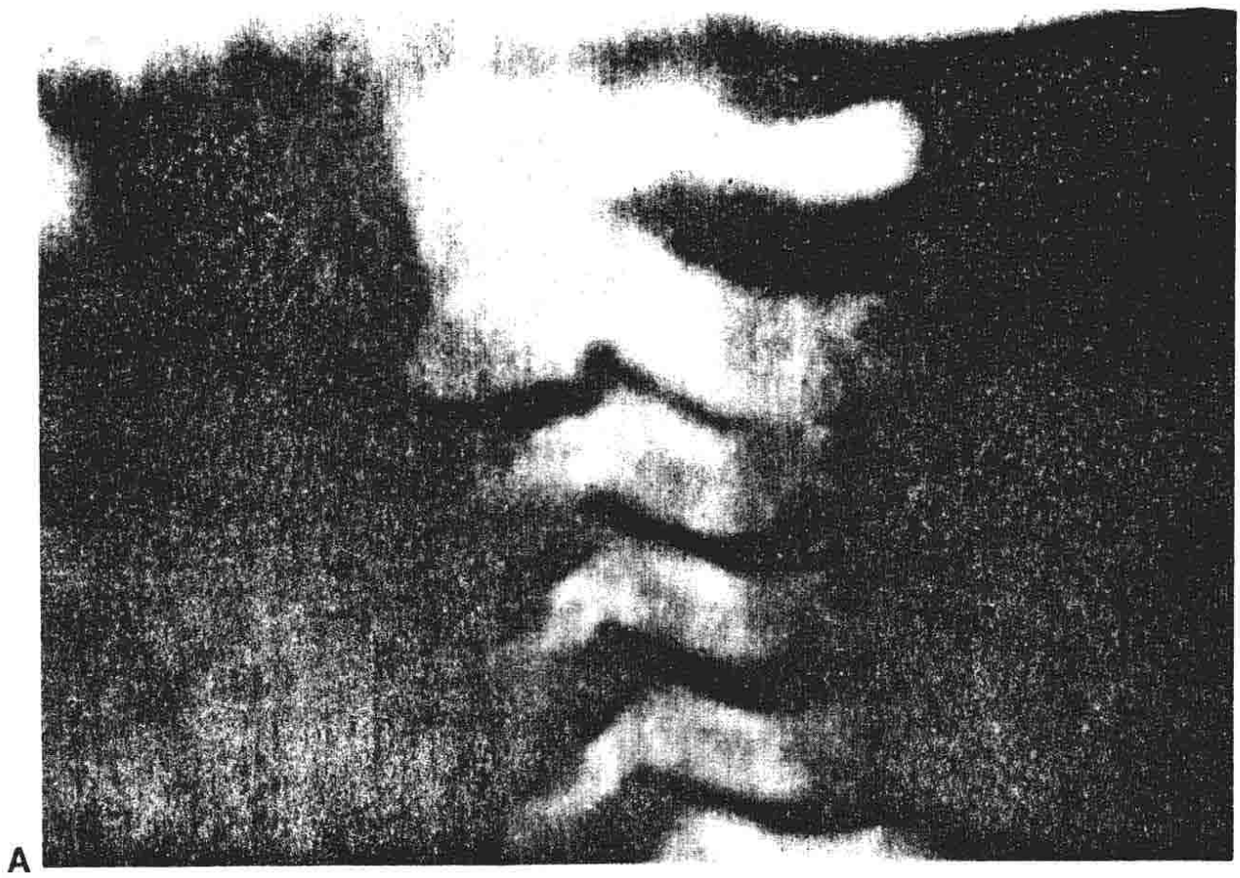
RADIOLOGIC PITFALLS

The pediatric anatomy lends itself to radiographic findings not seen in adults. The emergency physician must be familiar with the more common pediatric findings in order to avoid misinterpretation and overally aggressive treatment and referral. The following description of pediatric findings presumes familiarity with the basic cervical spine film interpretation in adults.[26,64–69]

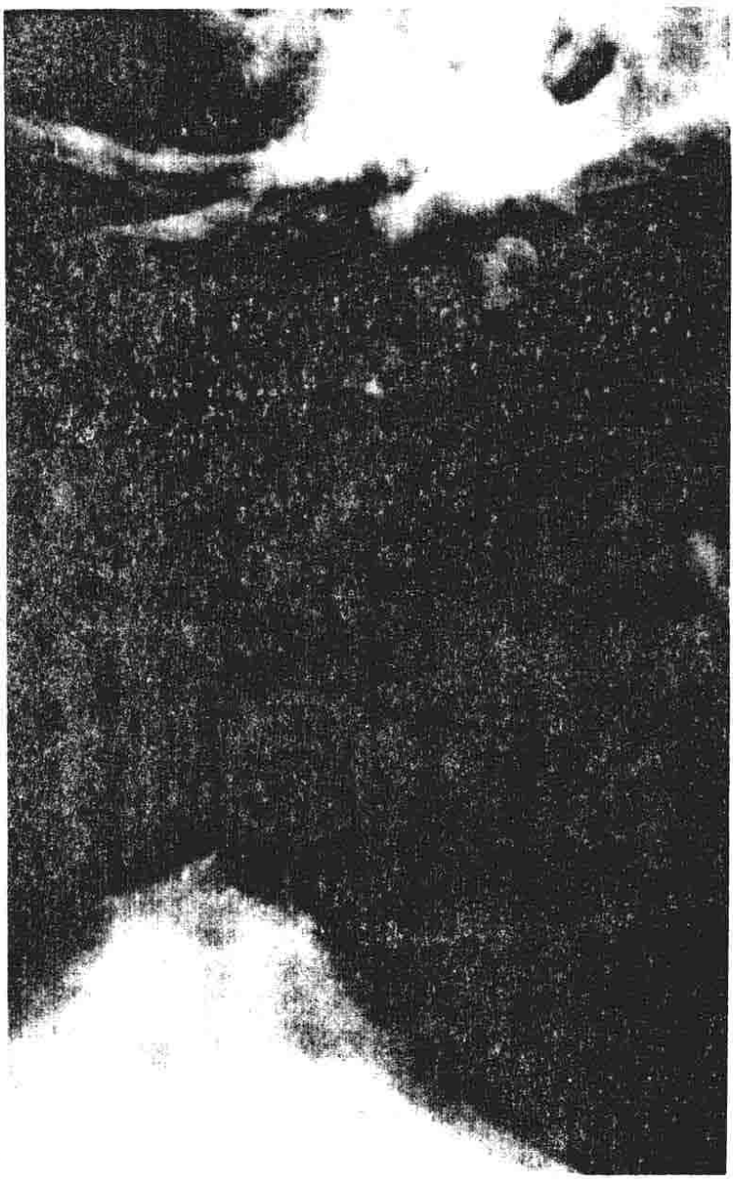
Prevertebral soft tissue space. There is considerable variability in the prevertebral soft tissue space in young children. It often appears increased in flexion or forceful expiration and should not be interpreted as a hematoma due to cervical fractures. Large adenoidal tissue can also cause a pseudothickening of the prevertebral space. It is not possible to give precise measurements of the prevertebral space that is indicative of pathology, but suggested norms have included soft tissue spaces less than 7 mm anterior to C2 or anterior-posterior diameter less than three-fourths the adjacent vertebral body diameter.[15]

Atlanto-dental distance. The predental space (distance between the anterior odontoid surface and posterior cortex of the anterior arch of C1 is increased in childhood. Distances up to 5 mm have been documented in normal children. There is also significant atlanto-axial movement, with flexion and extension manifested by variations in the predental distance up to 3 mm. In extension, the anterior arch may appear to ride up over the odontoid, so that the posterior two-thirds of the anterior arch is lying above the tip of the odontoid process. A truly increased predental space (greater than 5 mm) is suggestive of atlanto-axial instability, a Jefferson fracture, or atlanto-axial rotatory subluxation.

Atlanto-axial-ossification. One of the more notable findings in infant cervical spine films is that the anterior arch does not appear until approximately 1 year of age (Fig. 8-3A) It fuses with the lateral masses of C1 by approximately 8 years of age, while the neutral arches will fuse posteriorly at approximately 4 years of age. A small apical secondary ossification center may appear at the odontoid at 2 years of age and usually fuses by 12 years of age. This small ossification center at the tip of the dens should not be confused with a larger os odontoideum, which is associated with a hypoplastic odontoid. Like many secondary ossification centers the os terminale has variable configurations. The posterior arch of C1 is also a favorite spot for congenital defects, which may be confused for posterior arch fractures. The bony remnants in the congenital defects of the posterior arch often have a triangular shape with tapered ends pointing anteriorally. The synchondrosis between the odontoid and body of C2 is present at birth and through most of childhood. This is the site of most odontoid fractures in children, which typically lead to anterior displacement of the odontoid (Fig. 8-4A,B) Nondisplaced fractures through the synchondrosis can be subtle and the diagnosis may not be made until several weeks later when persistent symptomatology leads to a repeat radiograph. It is hypothesized that hypoplasia of the dens and some os odontoideums may result from such unrecognized fractures. Multiple other synchondroses throughout the cervical spine are best identified with standard radiographic textbooks. It is often on oblique views that these are visualized and they are gener-



A



B

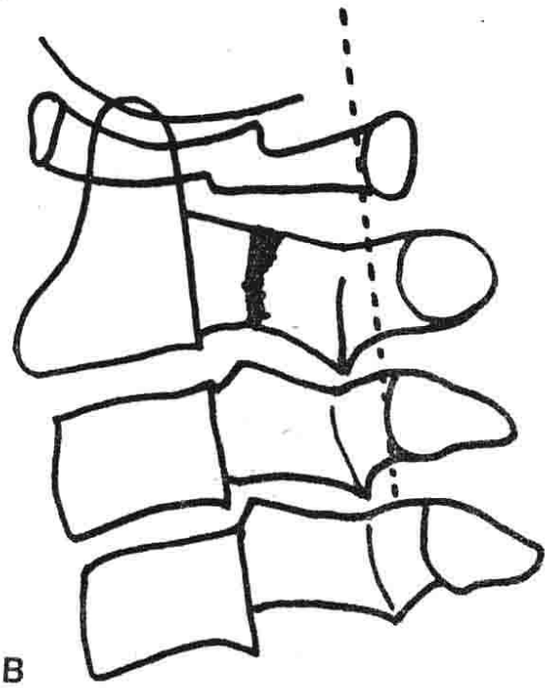
Fig. 8-3 (**A**) Lateral view in an infant shows normal absence of anterior arch ossification and anterior tapering of vertebral bodies. Note the synchondrosis between the odontoid and body of C2. (**B**) Lateral cervical spine of a young child demonstrates normal findings: predental space of 4 mm, pseudosubluxation of C2 on C3; anterior wedged shape of C3.

A

B

Fig. 8-4 (**A**) Odontoid fracture through the epiphyseal growth plate with anterior angulation of the odontoid. (**B**) Occasionally, the synchondrosis may be mistaken for a fracture.

A



B

Fig. 8-5 (**A**) A hangman's fracture may be relatively subtle. The mild C2 to C3 subluxation may have been misdiagnosed as a pseudosubluxation if the fracture site was not visualized. (**B**) Drawing a posterior cervical line would suggest the true diagnosis.

ally very subtle at that. Rarely, a secondary ossification center will be seen at the end of the spinous processes in an older child which may be confused at C7 with a clay shovelers fracture.

Increased C1 to C2 interspinous distance. An increased distance between the spinous processes of C1 and C2 should not be mistaken for posterior ligamentous disruption.

Wedge-shaped C2 to C4 vertebral bodies. The anterior portions of the vertebral bodies C2 to C4 taper anteriorly and may be confused with compression fractures. C3 especially has this tapered appearance in young children (Fig. 8-3B).

Pseudosubluxation. Up to 40 percent of children under 8 years of age have apparent anterior displacement of the upper vertebral bodies C2 to C4 on a physiologic basis. It is especially apparent on flexion radiographs. It may only occur at the C2 to C3 level, which is the fulcrum for flexion in the young child. In evaluating pseudosubluxations, Swischuk[70] described the use of a posterior cervical line that is drawn from the anterior cortices of the C1 and C3 spinous processes. The anterior cortex of the C2 spinous process should lie within 2 mm of

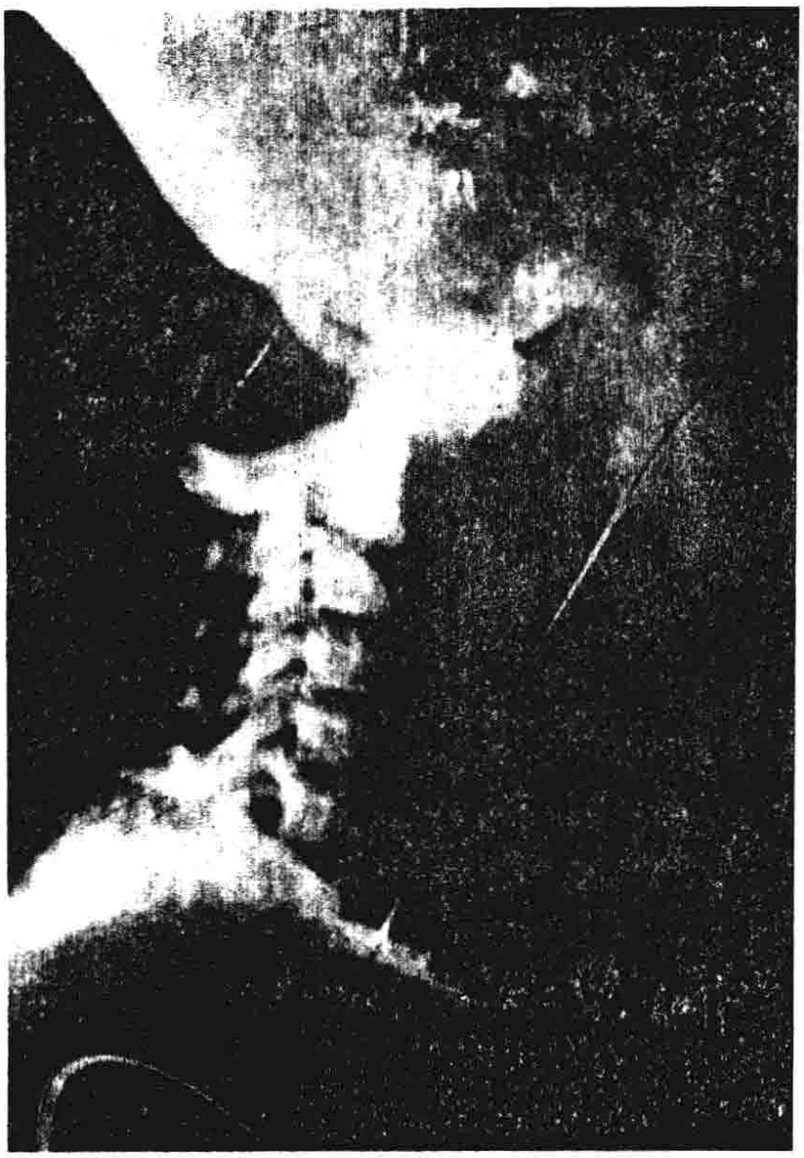
A

Fig. 8-6 Lethal cervical spine injuries in young children typically involve the upper cervical vertebrae. (**A**) Odontoid fracture with major anterior displacement and (*Figure continues.*)

this line. If the C2 spinous process lies more than 2 mm posterior to this line, a fracture through the posterior elements of C2 should be sought in the setting of apparent anterior displacement of C2 on C3 (Fig. 8-5). A fracture through the posterior elements of C2 with resultant anterior displacement of C2 on C3 is the classic hangman's fracture.

Lack of normal cervical lordosis. Older children may lack a normal cervical lordosis that may be mistaken for muscular spasm.

Table 8-5 summarizes these radiographic normals. More detailed descriptions of pediatric cervical radiographic findings are in the medical literature.[26–28,30,31,33,34,70–72] Many of these pathologic lesions resemble the adult radiologic picture (Figs. 8-6 through 8-8).



B

Fig. 8-6 (*Continued*). (**B**) atlanto-axial dislocation were fatal injuries in these cases. Note the significant prevertebral swelling.

Table 8-5. Radiographic Pitfalls

Radiographic Normals	Common Misinterpretations
Spurious prevertebral soft tissue thickening	Prevertebral hematoma
Increased predental distance (<5 mm)	Atlanto-axial instability
C1 anterior arch overiding odontoid (in extension)	Posterior C1 to C2 subluxation
Synchondroses	Fractures, especially at odontoid synchondrosis
Os terminale	Odontoid apical fracture or os odontoideum
C1 posterior arch anomalies	Fractures of posterior arch
Increased C1 to C2 interspinous distance	Posterior ligamentous disruption
C2 to C4 wedge-shaped vertebral bodies	Anterior compression fractures
Pseudosubluxation C2 to C4	Anterior subluxation
Absent cervical lordosis	Spasm with sprain or strain

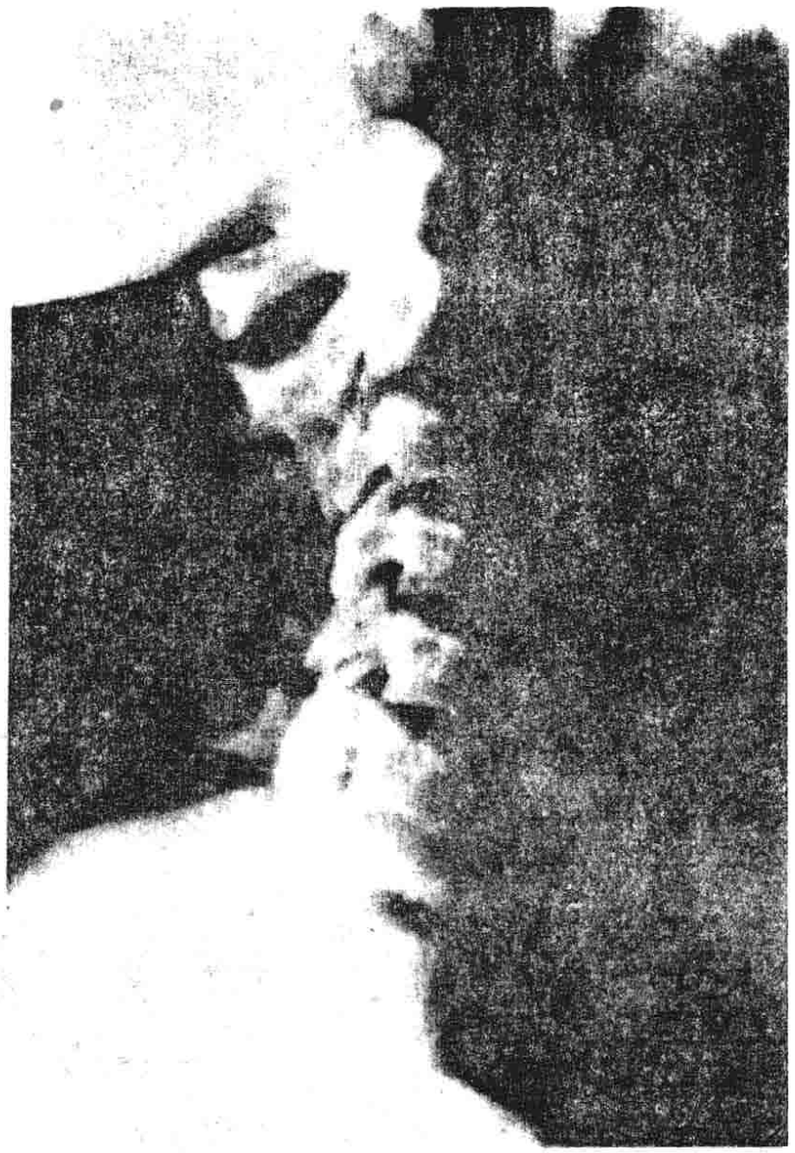


Fig. 8-7 C7 compression fracture in a 16-year-old patient demonstrates the need to visualize C7 to T1 joint space on lateral cervical spine radiograph. Approximately 18 percent of cervical fractures in adolescents and adults involve C7.

MANAGEMENT

Immobilization of the cervical spine with assessment and correction of any cardiopulmonary problems is always the initial concern in management. The immediate diagnostic and therapeutic decisions that follow depend on the overall stability of the patient.

The Alert Child Without Multiple Trauma

The approach to the alert child or adolescent without life-threatening injuries may proceed according to the clinical findings (Fig. 8-9). As a general rule, prehospital and emergency triage personnel should be encouraged to immobilize the spine if there is any question of cervical injury. In my

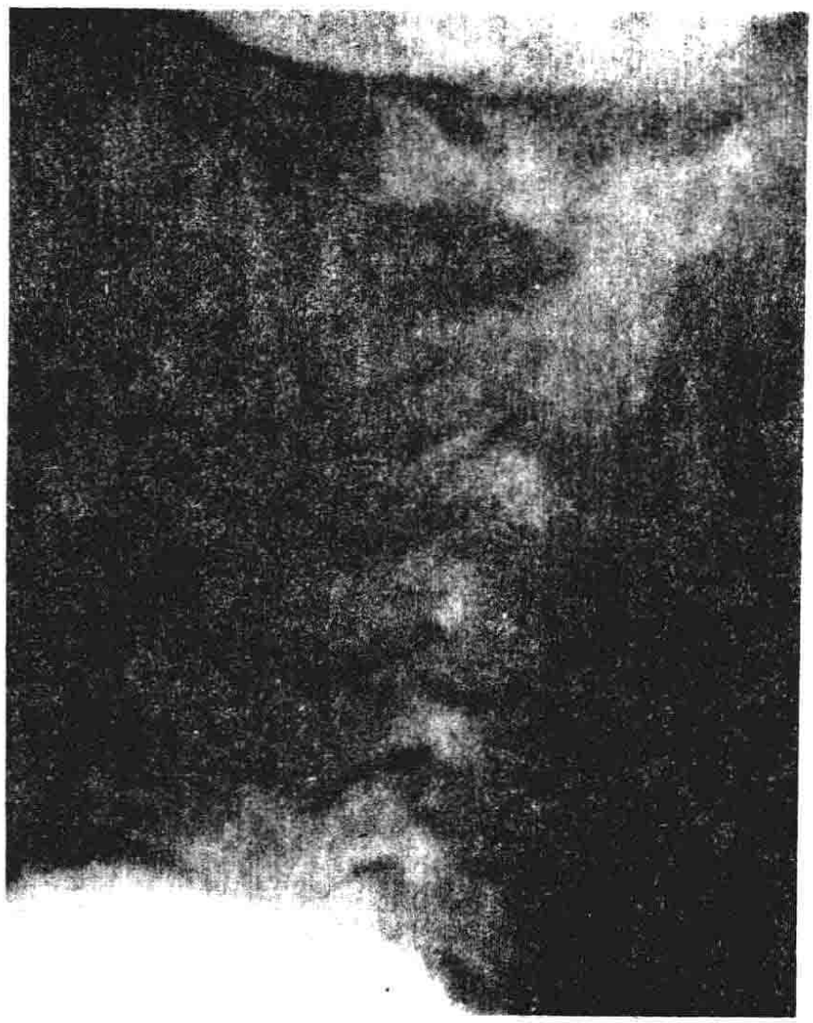


Fig. 8-8 CTLV of 8-year-old demonstrates anterior subluxation of C3 on C4, C4 compression fracture, and increased C3 to C4 interspinous distance.

view, however, immobilization does not obligate the physician to order radiographs. After a careful history and physical examination, when no neurologic deficits or complaints of neck pain are noted and the child is alert and cooperative, the immobilization of the cervical spine may be removed, as an assistant manually safeguards a neutral cervical position. If no tenderness or spasm is evident on physical examination, the child is asked to slowly undergo cervical range of motion, unless pain develops. If there is no pain, radiographs are not needed. In my experience, this algorithm will safely permit more selective radiologic evaluation. Similar approaches have been used successfully in adult patients.[55,62] Another common management decision presents when despite the paramedics' best attempts, the defiant toddler is quite successfully arching out of immobilization with obvious cervical range of motion. If the child is not combative because of hypoxia or other major injuries, I remove the restraints and allow the child to assume the most comfortable position (often sitting up or running to the mother). I do not advocate this approach for physicians who rarely deal with children.

If the decision is made to pursue radiographic evaluation, a portable CTLV should be obtained in the emergency department. After this view is interpreted, the open-mouth odontoid and anterior-posterior views may be

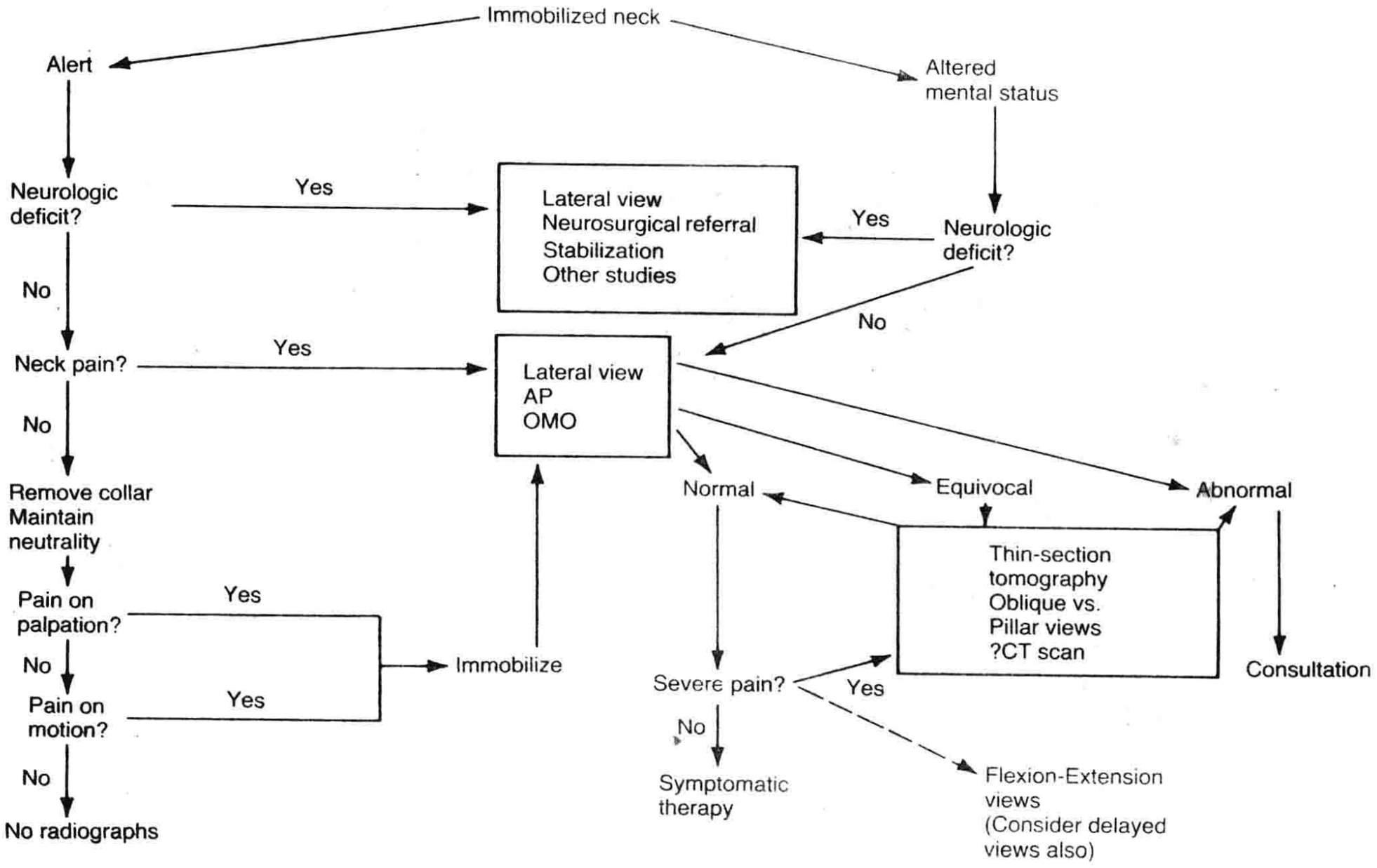

Immobilized neck
Alert
Altered mental status
Neurologic deficit?
Yes
Lateral view
Neurosurgical referral
Stabilization
Other studies
Yes
Neurologic deficit?
No
Neck pain?
Yes
Lateral view
AP
OMO
No
Remove collar
Maintain neutrality
Normal
Equivocal
Abnormal
Thin-section tomography
Oblique vs. Pillar views
?CT scan
Consultation
Pain on palpation?
Yes
No
Pain on motion?
Yes
No
No radiographs
Immobilize
Severe pain?
Yes
No
Symptomatic therapy
Flexion-Extension views
(Consider delayed views also)

Fig. 8-9

filmed in the radiology suite, with the patient remaining in the supine position and immobilized. This approach will identify the vast majority of cervical spine injuries. However, if the patient has significant pain and spasm, additional views or techniques may be indicated.

If additional views are unsuccessful in identifying an abnormality, flexion and extension views should be considered. Depending on the radiologist's and emergency physician's experience and time commitment, it may be reasonable to obtain orthopedic or neurosurgical consultation at this point. Flexion and extension views should only be obtained in alert and cooperative patients with physician supervision. If no abnormality is identified, the patient should be followed as an outpatient with consideration of a repeat dynamic study for persistent symptoms.[63]

Airway and Breathing in the Critically Injured Child

The management of the child with life-threatening injuries presents more challenging therapeutic decisions. In the setting of a possible cervical injury, addressing an unstable airway is the most urgent dilemma because most routine airway techniques produce neck motion. In addition, the stomach is full from accompanying ileus, food, or blood, and facial trauma is common. Study of an adult cadaver model with a surgically induced ligamentous C5 to C6 injury revealed that a chin lift, jaw thrust, and orotracheal intubation produced subluxation, extension, or disc-space widening. The application of a semirigid collar did not limit extension significantly during these procedures. Placement of a nasopharyngeal airway or blind nasotracheal intubation yielded the least cervical motion.[73] It is not known whether these and similar data from adult studies are applicable to the pediatric airway. The pediatric airway does not require much extension for visualization of the glottis; in fact, hyperextension often makes the airway less accessible.

Fortunately, most children can be ventilated via bag-valve-mask with a chin lift elevating the tongue block from the hypopharynx. (The chin life is preferred over the jaw thrust by the Advanced Trauma Life Support course.) A Sellick maneuver (gentle cricoid pressure) reduces gastric insufflation and the risk of regurgitation and aspiration. An assistant should manually provide cervical immobilization and in-line traction with any airway manipulation. The child who is apneic on presentation will require prompt intubation prior to radiologic studies. A common mistake is to jeopardize

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Fig. 8-9 Approach to the trauma patient. AP, anterior-posterior; OMO, open-mouth odontoid; CT, computed tomography. (Adapted from Wales LR, Knapp RK, Morishima MS: Recommendations for evaluation of the acutely injured cervical spine: A clinical radiologic algorithm. Ann Emerg Med 9:422, 1980.)

needed oxygenation and ventilation while awaiting cervical spine clearance.

The ideal method of intubation in the patient with possible cervical injury is via advancement of the endotracheal tube over a flexible fiberoptic bronchoscope.[72–74] This technique is not commonly available, however, and requires experienced hands to perform. Another disadvantage is the size of the pediatric bronchoscope; there is no suction channel, and endotracheal tubes smaller than 4.5 mm will not readily fit over the scope. The guided sytlet for orotracheal intubation is another promising airway technique that avoids movement of the cervical spine,[77,78] but there is little experience with this procedure in pediatric patients. Blind nasotracheal intubation requires a spontaneously breathing patient for best results and is difficult to perform in young children because of their anterior and relatively cephalad airway. When faced with an unstable airway in the pediatric patient with multiple trauma, most emergency physicians will reasonably resort to orotracheal intubation.

A nasogastric tube, or alternatively orogastric with facial fractures, should then always be placed because of uniform gastric atony and ileus in the patient with spinal cord injuries. The actual risks of orotracheal intubation and other airway maneuvers to the pediatric spine require further study. The low incidence of spinal cord injuries in children coupled with the difficulty and risks of performing cricoidthyroidotomies in children probably make the orotracheal route the overall safest approach.

The child with a cervical lesion (below C4) that paralyzes the intercostal musculature but preserves diaphragmatic function will also require intubation relatively early in the hospital course. The physician should not take comfort in seemingly adequate ventilation on initial presentation, because respiratory insufficiency usually supervenes. Vital capacity is only 21 to 24 percent predicted in adult patients with intercostal paralysis.[79] An inability to sigh or cough forcefully adds to the respiratory dysfunction. Prior to intubation, the patient should be preoxygenated; if the patient is alert with a good gag reflex, the stomach may be decompressed by nasogastric suction.[76] Atropine should be used routinely in these patients because of the prominent reflex bradycardia upon laryngeal manipulation. If intubation is facilitated via rapid-sequence induction, succinylcholine may be safely used the first 48 to 72 hours postinjury.[76] After this period, the use of depolarizing agents may induce hyperkalemic cardiac arrest. Other authorities suggest that even fresh spinal cord injury patients are susceptible to this denervated muscle potassium release and therefore advocate nondepolarizing agents or awake intubations.[80] Sedatives should obviously be used in either case.

Neurogenic pulmonary edema is another potential respiratory problem in spinal injuries. Recognition of its existence followed by prompt treatment with oxygen and positive end-expiratory pressure (PEEP) may minimize this complication.

Circulation in the Child with a Spinal Cord Injury

In the setting of multiple trauma and hypotension, the presumption must be that the lowered blood pressure is due to intraabdominal or intrathoracic hemorrhage. Peritoneal lavage or abdominal CT scan should be considered because the patient with a complete cervical cord lesion will not localize pain. Once the assessment of spinal shock is ascertained by physical examination, fluid therapy should be carefully monitored. A Foley catheter should always be inserted in the spinal cord injury patient to assist in monitoring cardiovascular status and avoid bladder distention. Fluid therapy should be guided by urine output, capillary perfusion, and arterial pH rather than the absolute blood pressure. Central monitoring should be strongly considered in the intensive care unit for accurate management of fluid therapy, and any needed vasopressor therapy. Occasionally, hypertension is present immediately after a spinal cord lesion, for up to 30 minutes. This transient phenomenon generally should not be treated.

Referral

Consultation and referral patterns for spinal cord injuries should be pre-established at all emergency departments. Application of Gardner-Wells tongs should only be considered after consultation with the receiving neurosurgery unit. (The soft skull of infants and young children generally precludes tong placement.) Transport of children with spinal cord injury should be as smooth as possible, with special efforts to maintain body temperature and prevent any secondary neural injury due to cardiopulmonary compromise.

REFERENCES

1. Kalsbeek WD, McLaurin RL, Harris BS, et al: The National Head and Trauma Spinal Cord Injury Survey: Major findings. J Neurosurg 53(suppl):19, 1980
2. Committee on Trauma. American College of Surgeons: Advanced Trauma Life Support Manual. American College of Surgeons, Chicago, 1984
3. Babcock JL: Cervical spine injuries. Arch Surg 111:646, 1976
4. Ergas A: Spinal cord injury in the United States: A statistical update. CNS Trauma 2:19, 1985
5. Kraus JF: A comparison of recent studies on the extent of head and spinal cord injury problems in the United States. J Neurosurg 53(suppl):35. 1980
6. Kraus JF, Franti CE, Riggins RS, et al: Incidence of traumatic spinal cord lesions. J Chron Dis 28:471, 1975
7. Bracken MB, Freeman DH, Hallebrand K: Incidence of acute traumatic hospitalized SCI in the US, 1970–1977. Am J Epidemiol 113:615, 1981

8. Hill SA, Miller CA, Kosnik EJ, et al: Pediatric neck injuries: A clinical study. J Neurosurg 60:700, 1894
9. Kewalramani LS, Kraus JF, Sterling HM: Acute spinal cord lesions in a pediatric population: Edipemiological and clinical features. Paraplegia 18:206, 1980
10. Young JS, Burns PE, Bowen AM, McCutchen R: Spinal cord injury statistics: Experience of regional model spinal cord injury systems. Good Samaritan Medical Center, Phoenix, 1982
11. Alker GJ, Young SO, Leslie EV, et al: Postmortem radiology of head and neck injuries in fatal traffic accidents. Radiology 114:611, 1975
12. Bucholz RW, Burkhead WZ, Graham W, et al: Occult cervical spine injuries in fatal traffic accidents. J Trauma 19:768, 1979
13. Rivera FP: Traumatic deaths of children in the United States: Currently available prevention strategies. Pediatrics 75:456, 1985
14. Anderson JM, Schutt AH: Spinal injury in children: A review of 156 cases seen from 1950 through 1978. Mayo Clin Pract 55:499, 1980
15. Apple JS, Kirks DR, Merten DF, et al: Cervical spine fractures and dislocations in children. Pediatr Radiol 17:45, 1987
16. Henrys P, Lyne ED, Lifton C, et al: Clinical review of cervical spine injuries in children. Clin Orthop 129:172, 1977
17. Hubbard DD: Injuries of the spine in children and adolescents. Clin Orthop 100:56, 1974
18. Huelke DF, O'Day J, Mendelsohn RA: Cervical injuries suffered in automobile crashes. J Neurosurg 54:316, 1983
19. Torg JS: Trampoline-induced quadriplegia. Clin Sports Med 6:73, 1987
20. Bresnan MJ, Abrams IF: Neonatal spinal cord transection secondary to intrauterine hyperextension of the neck in breech presentation. J Pediatr 84:734, 1974
21. Shulman ST, Madden JD, Esterly JR, et al: Transection of spinal cord. A rare complication of cephalic delivery. Arch Dis Child 46:291, 1971
22. Coffey J: Whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. Pediatrics 54:396, 1974
23. Dickson RA, Leatherman KD: Spinal injuries in child abuse. Case report. J Trauma 18:811, 1978
24. Swischuk LE: Spine and spinal cord trauma in the battered child syndrome. Radiology 92:733, 1969
25. Bailey DK: The normal cervical spine in infants and children. Radiology 59:712, 1952
26. Shapiro R, Youngberg AS, Rothman SL: The differential diagnosis of traumatic lesions of the occipito-atlanto-axial segment. Radiol Clin North Am 11:505, 1973
27. Bland JH: Disorders of the Cervical Spine: Diagnosis and Medical Management. WB Saunders, Philadelphia, 1987
28. Cattell HS, Filtzer DL: Pseudosubluxation and other normal variations in the cervical spine in children. J Bone Joint Surg 47A:1295, 1965
29. American Academy of Pediatrics Committee on Sports Medicine: Atlantoaxial instability in Down syndrome. Pediatrics 74:152, 1984
30. Fielding JW, Hawkins RJ: Atlanto-axial rotatory fixation. J Bone Joint Surg, 59A:37, 1977
31. Marar BC, Balachandran N: Non-traumatic atlanto-axial dislocation in children. Clin Orthop 92:220, 1973

32. Clark RN: Diagnosis and management of torticallis. Pediatr Ann 5:231, 1976
33. Pennecot GF, Gouraud D, Hardy JR, et al: Roentgenographical study of the stabilization of the cervical spine in children. J Pediatr Orthop 4:346, 1984
34. Sherk HH, Schut L, Lane JM: Fractures and dislocations of the cervical spine in children. Orthop Clin North Am 7:593, 1976
35. Stauffer ES, Mazur JM: Cervical spine injuries in children. Pediatr Ann 11:502, 1982
36. Steel HH: Anatomical and mechanical considerations of the atlanto-axial articulation. J Bone Joint Surg 50A:1481, 1968
37. Burke DC: Traumatic spinal paralysis in children. Paraplegia 11:268, 1974
38. Pang, D, Wilberger JE: Spinal cord injury without radiographic abnormalities in children. J Neurosurg 57:114, 1982
39. Cheshire DJ: The pediatric syndrome of traumatic myelopathy without demonstrable vertebral injury. Paraplegia 15:74, 1977
40. Soderstrom CA, Brumback RJ: Early care of the patient with cervical spine injury. Orthop Clin North Am 17:3, 1986
41. Podolsky S. Baraff LJ, Simon RR, et al: Efficacy of cervical spine immobilization methods. J Trauma 23:461, 1983
42. Cloward, RB: Acute cervical spine injuries. Clin Symp 32:15, 1980
43. Rogers WA: Fractures and dislocations of the cervical spine. J Bone Joint Surg 39A:341, 1957
44. Johnson, RM, Hart DL, Simmons EF, et al: Cervical orthoses—A study comparing their effectiveness in restricting cervical motion in normal subjects. J Bone Joint Surg 59A:332, 1977
45. Garth GC: Personal communication. California Medical Products, Long Beach, California, January 1987
46. Aprahamian C, Thompson BM, Darin JC: Recommended helmet removal techniques in a cervical spine injured patient. J Trauma 9:841, 1984
47. Vegso JJ, Lehman RC: Field evaluation and management of head and neck injuries. Clin Sports Med 6:1, 1987
48. Meyer RD, Daniel WW: The biomechanics of helmets and helmet removal. J Trauma 25:329, 1985
49. Seiman, LP: Fracture of the odontoid process in young children. J Bone Joint Surg 59A:943, 1977
50. Bresler MJ: Occult cervical spine fracture in an ambulatory patient. Ann Emerg Med 11:440, 1982
51. Webb JK, Broughton RB, Park WM: Hidden flexion injury of the cervical spine. J Bone Joint Surg 58B:322, 1976
52. Thambyrajah K: Fractures of the cervical spine with minimal or no symptoms. Med J Malaya 26:244, 1972
53. Maull KI, Sachatello CR: Avoiding a pitfall in resuscitation: The painless cervical fracture. South Med J 70:477, 1977
54. Walter J, Doris PE, Shaffer MA: Clinical presentation of patients with acute cervical spine injury. Ann Emerg Med 13:512, 1984
55. Fischer RP: Cervical radiographic evaluation of alert patients following blunt trauma. Ann Emerg Med 13:905, 1984
56. Rachesky I, Boyce T, Duncan B, et al: Clinical prediction of cervical spine injuries in children. Am J Dis Child 141:199, 1987
57. Jaffe DM, Binns H, Radkowski MA, et al: Developing a clinical algorithm for early management of cervical spine injury in child trauma victims. Ann Emerg Med 16:270, 1987

58. Cadoux CG, White JD: High-yield radiographic considerations for cervical spine injuries. Ann Emerg Med 15:236, 1986
59. Blahd WH, Iserson KV, Bjelland JC: Efficacy of the post-traumatic cross table laterial view of the cervical spine. J Emerg Med 2:243, 1985
60. Streitwieser DR, Knapp R, Wales LR, et al: Accuracy of standard radiographic views in detecting cervical spine fractures. Ann Emerg Med 12:538, 1983
61. Shafer MA, Doris PE: Limitation of the cross table lateral view in detecting cervical spine injuries: A retrospective analysis. Ann Emerg Med 10:508, 1981
62. Wales LR, Knapp RK, Morishima MS: Recommendations for evaluation of the acutely injury cervical spine: A clinical radiologic algorithm. Ann Emerg Med 9:422, 1980
63. Pennecot GF, Leonard P, DesGachons SP, et al: Traumatic ligamentous instability of the cervical spine in children. J Pediatr Orthop 4:339, 1984
64. Williams CF, Bernstein TW, Jelenko C: Essentiality of the lateral cervical spine radiograph. Ann Emerg Med 10:198, 1981
65. Wier DC: Roentgenographic signs of the cervical injury. Clin Othop 109:9, 1975
66. Clark WM, Gehweiler JA, Laib R: Twelve significant signs of cervical spine trauma. Skel Radiol 3:201, 1979
67. Harri JH, Edeiken-Monroe B, Kopaniky DR: A practical classification of acute cervical spine injuries. Orthop Clin North Am 17:15, 1986
68. Whitley JE, Forsyth HF: The classification of cervical spine injuries. AJR 83:633, 1960
69. Bohlman HH: Acute fractures and dislocations of the cervical spine. J Bone Joint Surg 61A:1119, 1979
70. Swischuk LE: Anterior displacement of C2 in children: Physiologic or pathologic? Radiology 122:759, 1977
71. Swischuk LE: The cervical spine in childhood. Curr Prob Diagn Radiol 13:1, 1974
72. Swischuk LE: Emergency Radiology of the Acutely Ill or Injured Child. 2nd Ed. Williams & Wilkins, Baltimore, 1986
73. Aprahamian C, Thompson BM, Finger WA, et al: Experimental cervical spine injury model: Evaluation of airway management and splinting techniques. Ann Emerg Med 13:584, 1984
74. Hemmer D, Lee T, Wright BD: Intubation of a child with a cervical spine injury with the aid of a fiberoptic bronchoscope. Anaesth Intensive Care 10:163, 1982
75. Messeter KH, Pettersson: Endotracheal intubation with fiberoptic bronchoscope. Anaesthesia 35:294, 1980
76. Fraser A, Edmonds-Seal J: Spinal cord injuries: A review of the problems facing the anaesthetist. Anaesthesia 37:1084, 1982
77. Vollmer TP, Stewart RD, Paris PM, et al: Use of a lighted stylet for guided orotracheal intubation in the prehospital setting. Ann Emerg Med 14:324, 1985
78. Ellis DG, Stewart RD, Kaplan RM, et al: Success rates of blind orotracheal intubation using a transillullimination technique with a lighted stylet. Ann Emerg Med 15:138, 1986
79. Ledsome JR, Sharp JM: Pulmonary function in acute cervical cord injury. Am Rev Respir Dis 124:41, 1981
80. Albin MS: Resuscitation of the spinal cord. Crit Care Med 6:270, 1978

9 Problems in Fracture Management

Jorge E. Alonso

Fractures involving the pediatric age group present unique problems of diagnosis and management. There is a false sense of security that children's fractures will always heal and deformities will remodel but, as Mercer Rang stated, "there are bad results." As many as 50,000 children are permanently crippled secondary to trauma each year in the United States. Orthopedic problems are second to neurologic problems in permanent disability. This has driven the pediatrician and emergency physician away from treating pediatric fractures. Most will refer all fractures. This can be improved by a more expedient diagnostic approach and early management of the fracture.

DIFFERENCES BETWEEN PEDIATRIC AND ADULT BONES

The child's skeletal elements are in a more dynamic and constantly changing growth mode, whereas the adult skeleton has ceased growth and apposition is remodeling the established elements in accord with stress responses. The major differences can be grouped in three categories: anatomy, physiology, and biomechanics.[1,2]

Anatomy

Periosteum. The periosteum in the child is thicker, more osteogenic, and more resistant to disruption than the periosteum of the adult. Since periosteum separates more easily from the bone in children when there is a

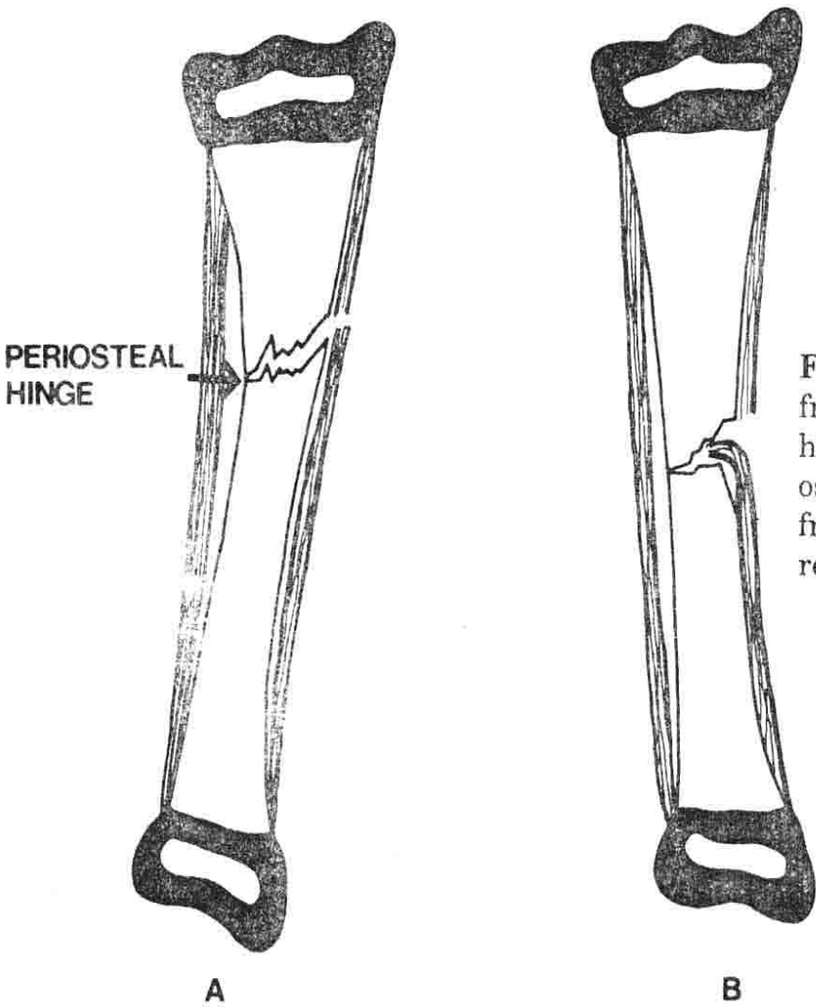


Fig. 9-1 (A) Periosteum as a friend, helping with the hinge for reduction. (B) Periosteum as an enemy in the fracture site, preventing full reduction.

fracture, it is less likely to rupture completely and usually remains intact on the compressive side. This intact side can be used as a hinge to assist in the reduction. At the same time, the periosteum can be our enemy, and the ruptured periosteum on the tension side can be interposed between the fracture fragments and thus hinder reduction (Fig. 9-1). The periosteum permits some degree of continuity as a tube for osteogenic potential. The subperiosteal new bone quickly bridges the fracture leading to a more rapid stability.[1-3]

Growth Plate. Even though ligaments exhibit a greater degree of laxity than they do in adults, they are more resistant to injury than the growth plate. A sprained ligament is the last diagnosis to consider in a child. Growth plate injuries and buckle fractures are the childhood equivalent to an adult sprain.[1-4] Approximately 15 percent of all fractures in children involve the physis. Injury to this area can produce not only angular deformities but growth and overgrowth as well. A careful diagnosis of injuries in this area is therefore mandatory.

Physiology

The child's bone is more porous than an adult's, permitting the bone to bend, buckle, and sustain greenstick fractures. The child's bone is undergoing active, rapid growth and remodeling. Fractures heal rapidly in most

cases, and nonunions are rare. The younger the patient, the faster the fracture heals. However, damage to the capacity of the bone to accomplish these physiologic functions may impair subsequent growth and development in several ways.

Biomechanics

Comminuted fractures are rare in children. The porosity in a cross section of a child's bone is much greater than that of an adult, and this may play a role in stopping fracture propagation. Adult bone usually fails in tension (stiffer bone), whereas a child's bone may fail in either tension or compression.

Joint stiffness is rare in children, but new concepts of cast braces and new philosophy of motion of joints have prepared the orthopedic surgeon not to immobilize a joint for more than 3 weeks regardless of the radiographic appearance.

Remodeling is a tray used by the orthopedic surgeon in treating children's fractures. Remodeling will help if the child has two or more years of growth remaining, has a fracture near the end of a bone, and if the deformity or angulation is the plane of motion of a joint. Remodeling will not help in displaced intraarticular fractures; in mid-shaft fractures that are grossly shortened, angulated, or rotated; and in displaced fractures near the ends of the bones in which the axis of displacement is at right angles to the plane of motion, and in displaced fractures crossing the growth plate at right angles.[2,3]

FAILURE TO CONSIDER THE UNIQUE CLASSIFICATION OF PEDIATRIC FRACTURES

In communicating with other physicians, terminology should accurately locate and morphologically describe any fractures. Anatomic location of fractures in children may have a major impact on appropriate treatment methods and potential long-term problems. The emergency physician must not only understand the classification of children's fractures but must also be able to communicate this information to the consultant.

Diaphyseal Fractures

Diaphyseal fractures involve the central shaft (Fig. 9-2). The basic types are listed below; diagnosis must be based on appropriate roentgenograms of the injury.[2,3]

Longitudinal. The fracture line follows the long axis of the bone. This fracture is more likely to occur in the older child (Fig. 9-2A1).

Transverse. The fracture line is at right angles to the long axis of the

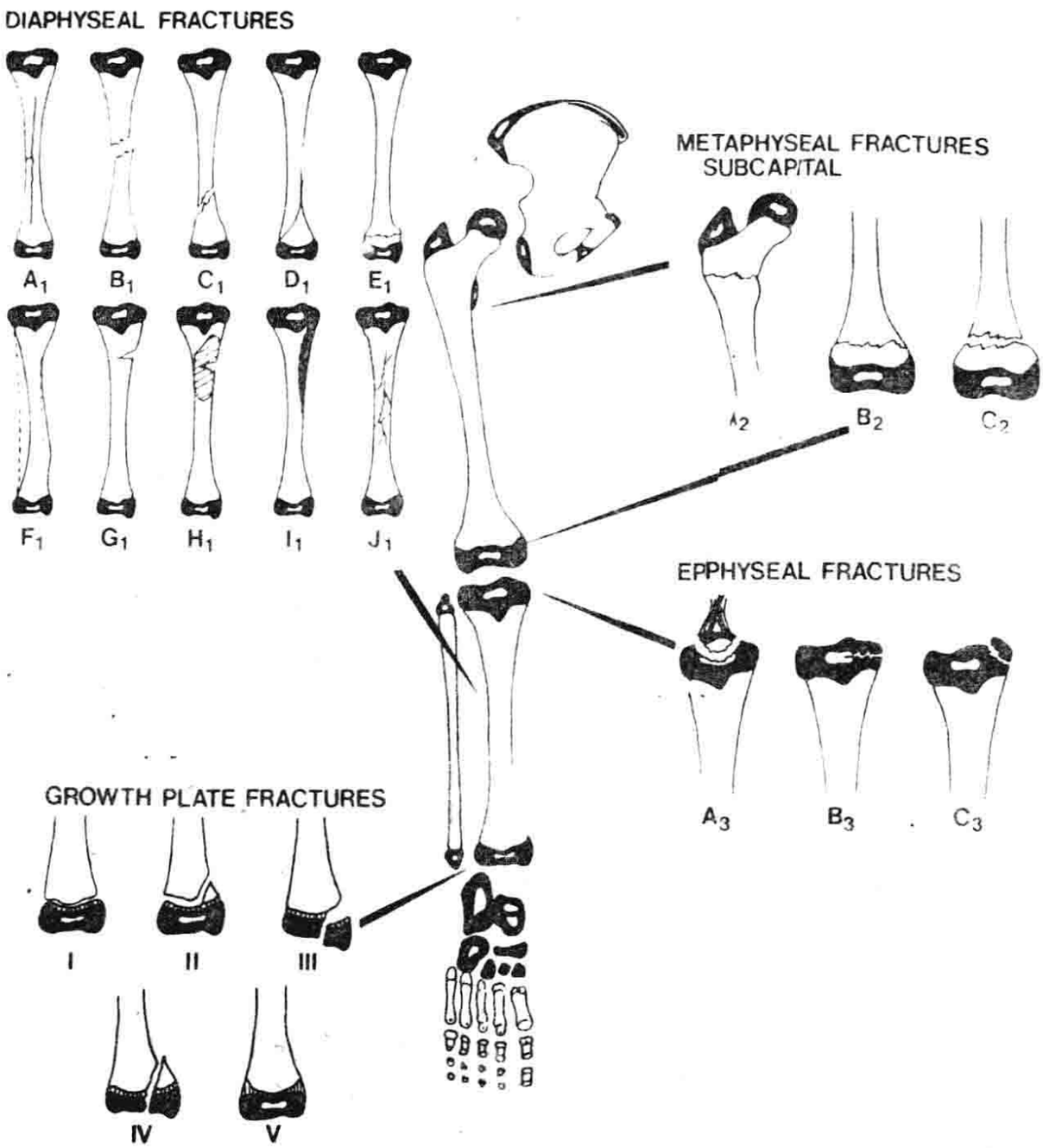


Fig. 9-2 Types of fracture.

bone. It is secondary to direct trauma and is frequently seen in the diaphysis of young children and infants (Fig. 9-2B1).

Oblique. The fracture line is variably angled relative to the long axis of the bone usually 30 to 40 degrees. Its mechanism is usually a direct trauma with some rotational component. This type is common in greenstick injuries (Fig. 9-2C1).

Spiral. The fracture line is not only oblique but encircles a portion of the shaft. It is common in tibial and humral shaft injuries. Its mechanism of injury is usually a twisting injury. When this fracture is seen in the toddler, the possibility of child abuse should be considered (Fig. 9-2D1).

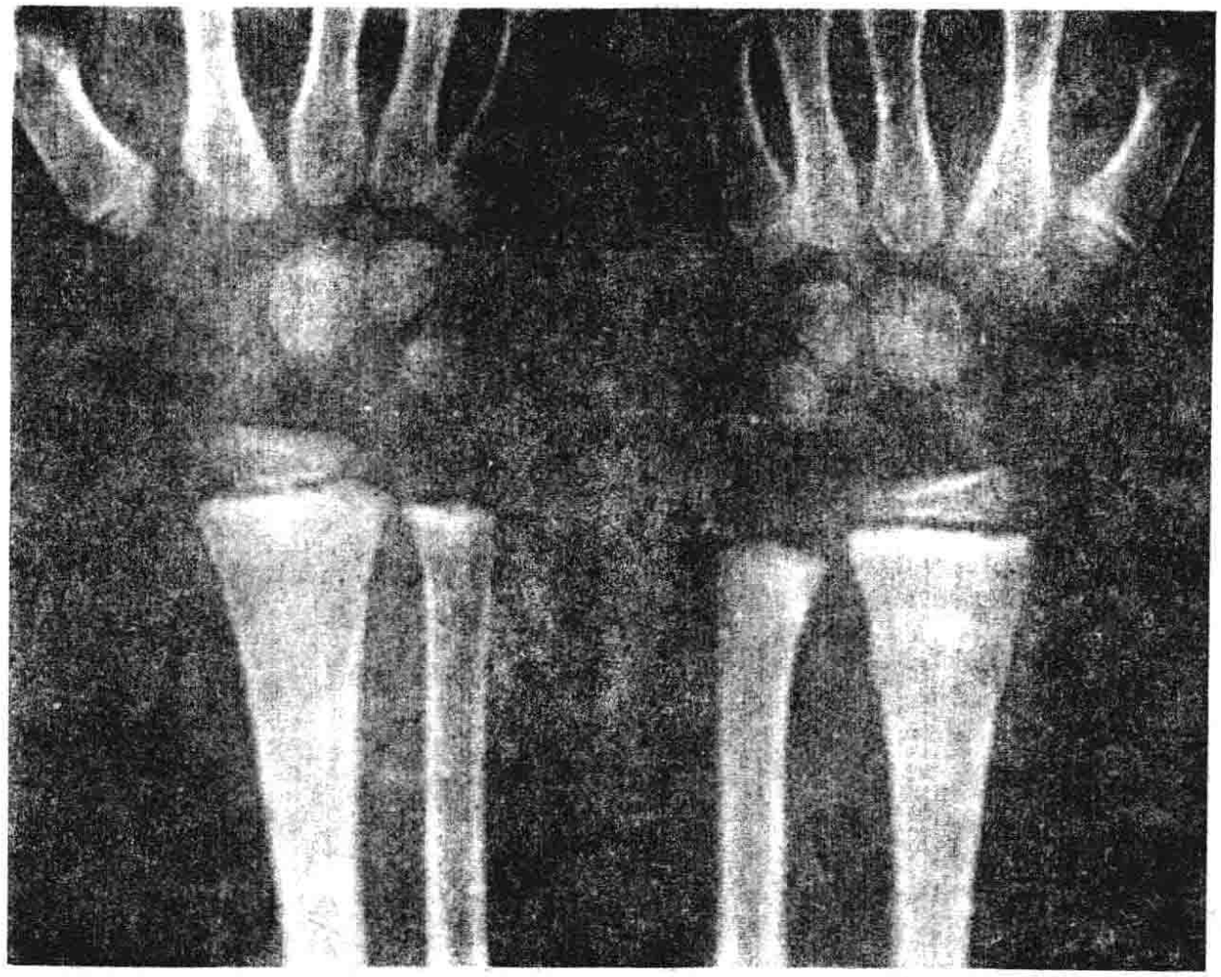


Fig. 9-3 Buckle fracture of distal wrist (arrow) with comparison film of the opposite wrist.

Impacted. The cortical and trabecular bone of each side of the fracture line are crushed together. The mechanism is usually a compression injury, also called torus fractures (Fig. 9-2E1).

Bowing. In this type of fracture, no fracture lines are visible. The bone is deformed beyond full elastic recoil into a phase of permanent plastic deformation. The younger the child, the more likely this type of skeletal injury may occur. It is more commonly seen in the fibula and ulna, both of which may bow, while the paired bone (tibia or radius) tends to fracture (Fig. 9-2F1).

Greenstick. The greenstick fracture is a common injury in children (Fig. 9-3). The bone is incompletely fractured, with a fractured cortex in the tension side and intact cortex in the compression side. Since the intact cortex is deformed and may produce an angular deformity, complete fracture of the intact cortex and reduction are necessary (Fig. 9-2G1).

Pathologic. This is a fracture through abnormal bone. This bone is more susceptible to injury when a tumor, infection, or metabolic hereditary disease alters normal bone formation (Fig. 9-2H1).

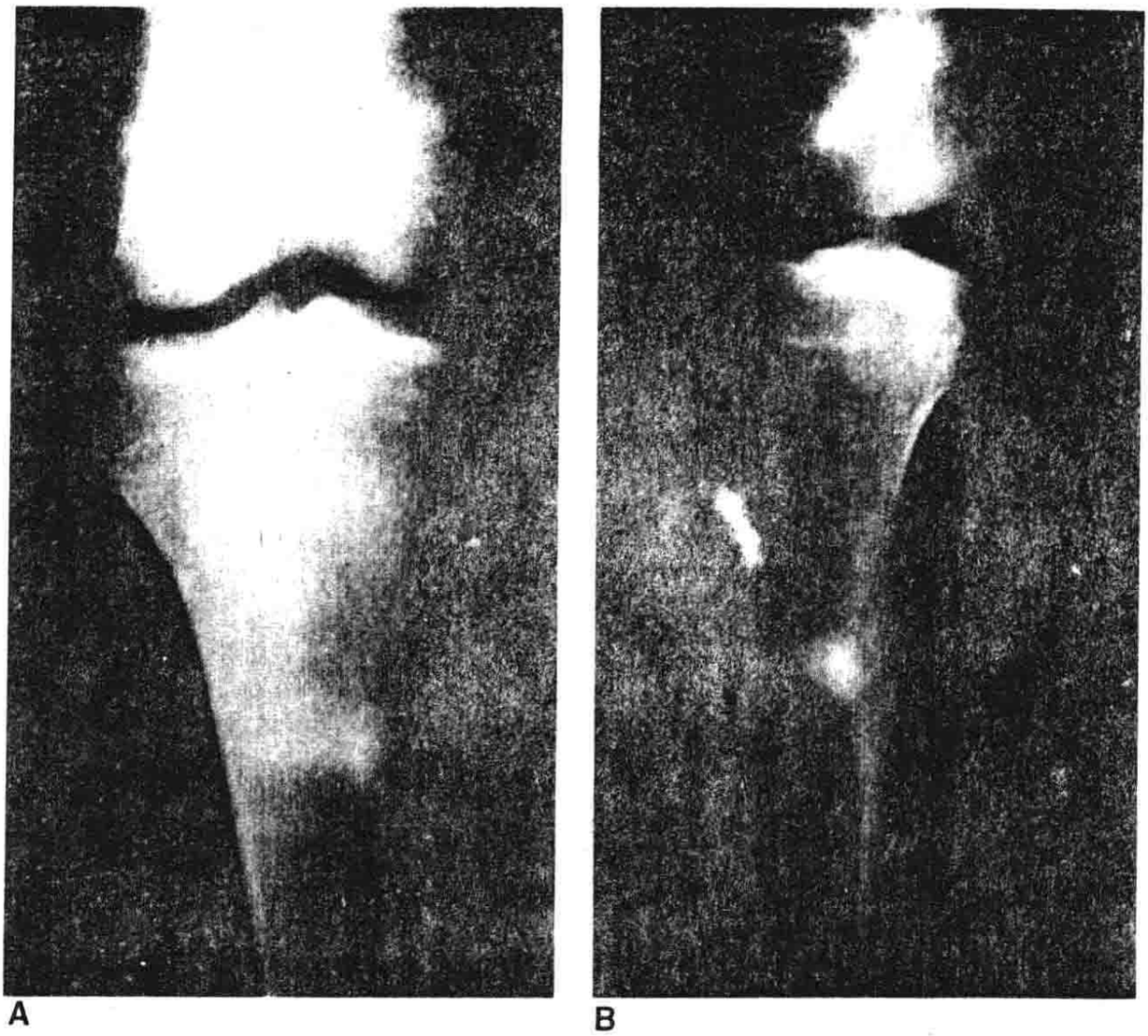


Fig. 9-4 (**A**) anterior-posterior and (**B**) lateral radiographs of stress fracture in a long-distance runner. The proximal tibia is more common in children.

Stress. Cyclic, minor, repetitive trauma on certain bones may lead to cortical fractures. Referred to as stress fractures, in children they are seen more frequently in the proximal tibia, spine, and metatarsals (Figs. 9-2I1 and 9-4).

Comminuted. The fracture lines propagate in several directions. This is an uncommon fracture in infants and young children but becomes more common in adolescents and in high-energy injuries (Figs. 9-2JI and 9-5).

Methaphyseal Fractures

Methaphyseal fractures involve the area between the diaphysis and the epiphysis (Fig. 9-2). They are classified as follows:

Subcapital. Subcapital fractures consist of metaphyseal involvement of certain bones, such as the proximal femur or radius (Fig. 9-2A2).

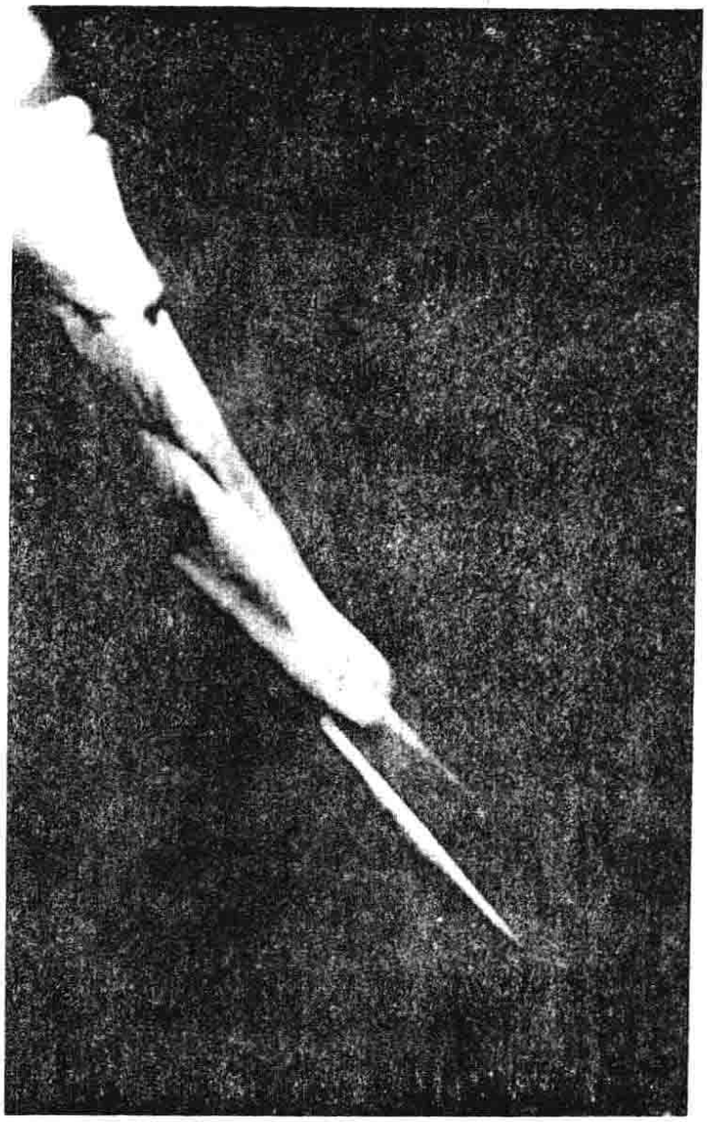


Fig. 9-5 Segmental comminuted fracture of the tibia, most commonly associated with high-energy trauma.

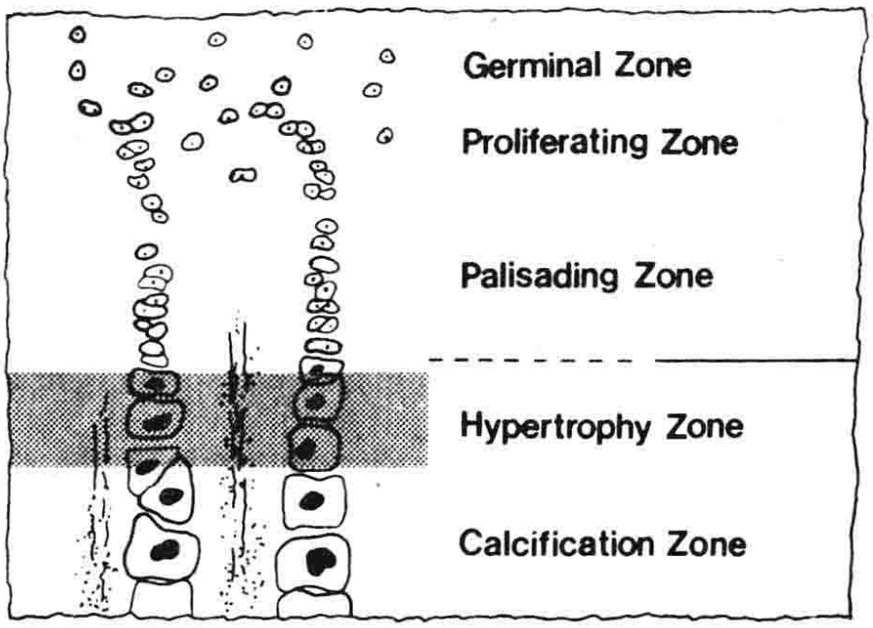


Fig. 9-6 The five zones of the growth plate. Injuries occur through the weakest area the hypertrophy zone.

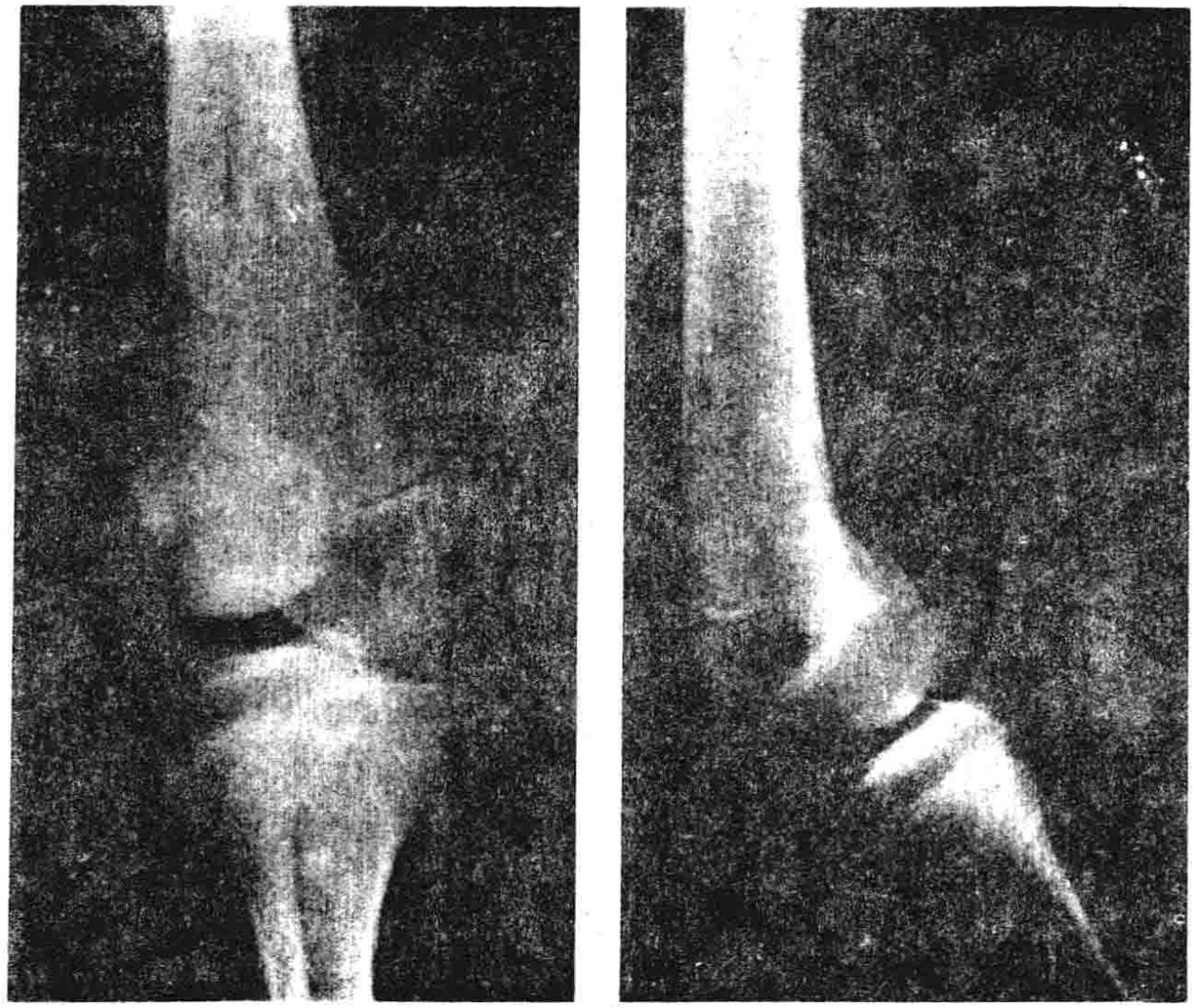


Fig. 9-7 An 11-year-old with injury to the right knee. Anterior-posterior (A) and lateral (B) views are inconclusive (*Figure continues.*)

Supracondylar. Supracondylar fractures are found above the level of the condyles and epicondyles in the femur and humerus respectively (Fig. 9-2B2).

Compression. The torus type of fracture is frequently seen in the metaphyseal area because there is more cancellous bone and the cortical bones is not as thick as in diaphyseal bone.

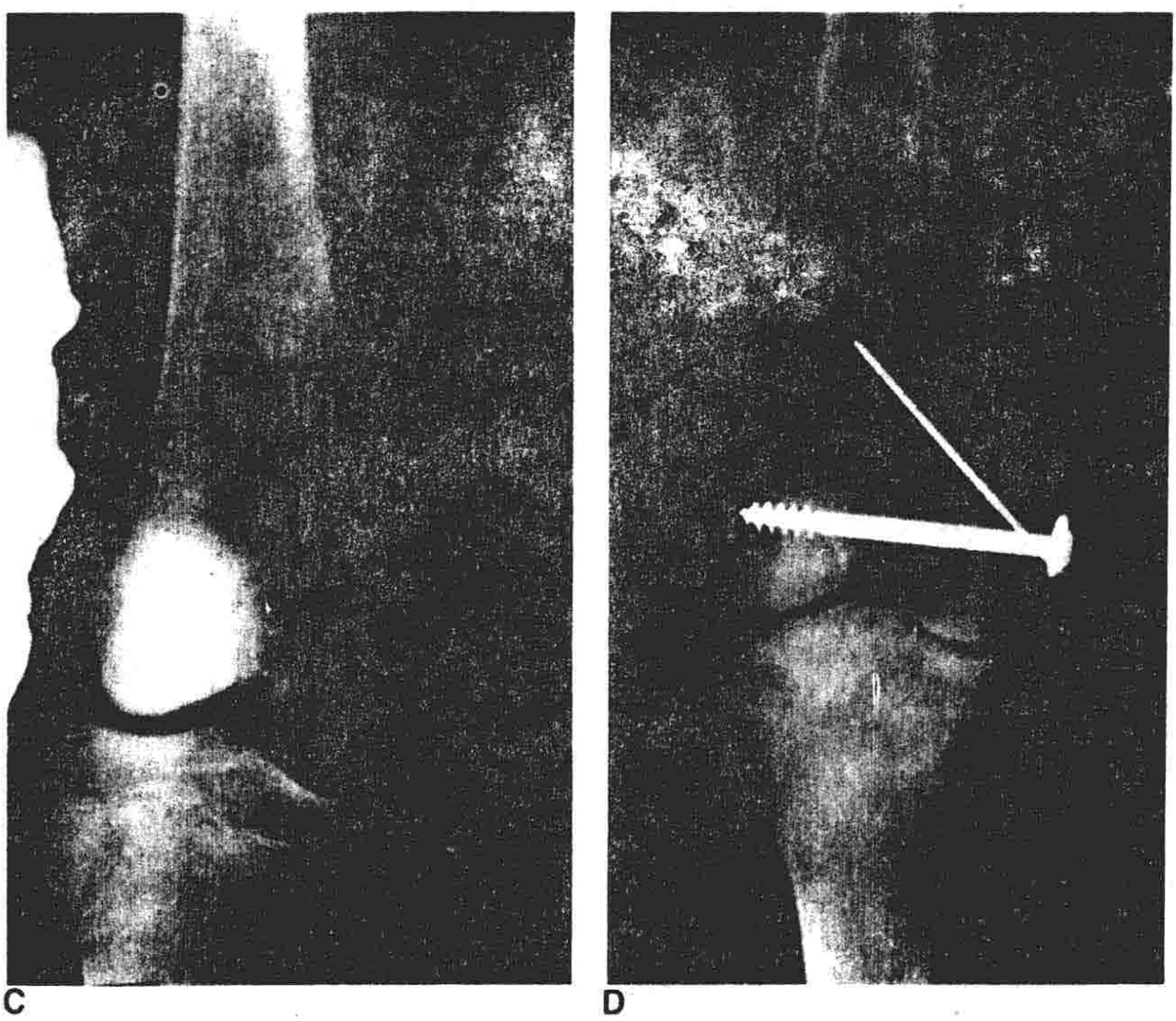
Pathologic. This type of fracture is seen frequently in cystic lesions in the proximal humerus and tibia (e.g., simple bone cyst, aneuryseal bone cyst).

Stress. Stress fractures are seen more commonly in the child in the proximal metaphyseal area of the tibia.

Epiphyseal Fractures

Epiphyseal fractures not involving the growth plate are classified as follows (Fig. 9-2):

Avulsion. Avulsion fractures occur more commonly in the tibial spine,



C D

Fig. 9-7 (*Continued*). Stress film (C) shows an epiphyseal injury. Salter-Harris III anterior-posterior (D) and lateral (*Figure continues.*)

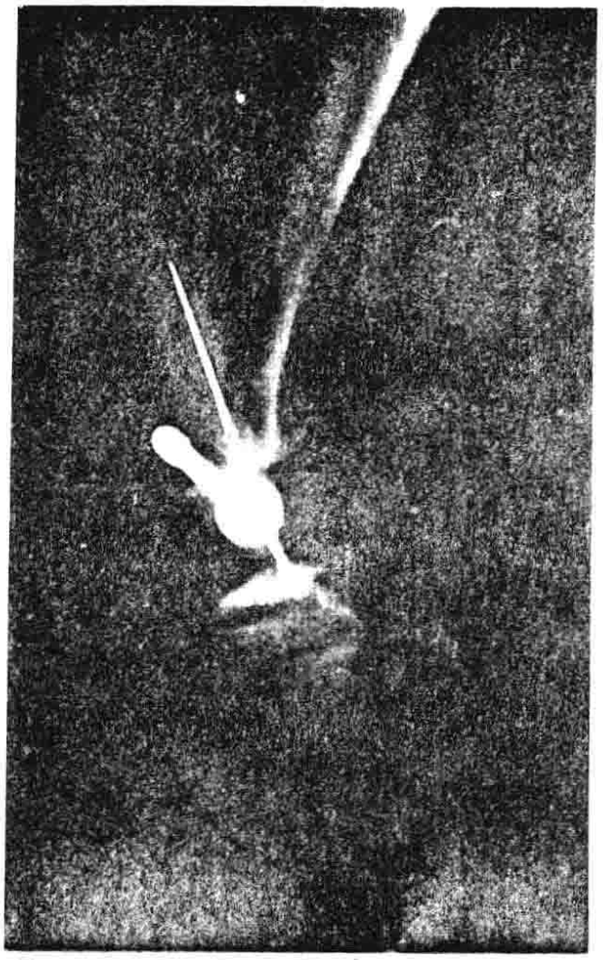
the ulna styloid, and the bases of the phalanges. If the fragment is displaced, nonunion can be seen because the fracture line is bathed in joint fluid and inhibits callus formation. Also, the fragment may block motion or leave a joint unstable (Fig. 2A3).

Osteochondral. Osteochondral fragments are most commonly seen in the distal femur, patella, and head of the radius. If the fragment is small, it should be removed. If it is large and from an important part of a joint, it should be fixed surgically (Fig. 9-2C3).

Compression. Compression fractures of the epiphysis are unusual (Fig. 9-2B3).

Growth Plate Injuries

The growth plate generally repairs well, but occasionally these injuries leave the potential to cause angular deformities, if part of the plate is injured or a bony bridge has formed, progressive shortening of the limb, if the majority of the plate is injured, and joint incongruity if less than optimum alignment has been obtained in intra-articular fractures.[1-3,5]



E

F

G

Fig. 9-7 (*Continued*). (**E**) radiographs of the intraarticular fracture reduced and internally fixed. Follow-up anterior-posterior (**F**) and lateral (**G**) radiographs 1 year after implant removal.

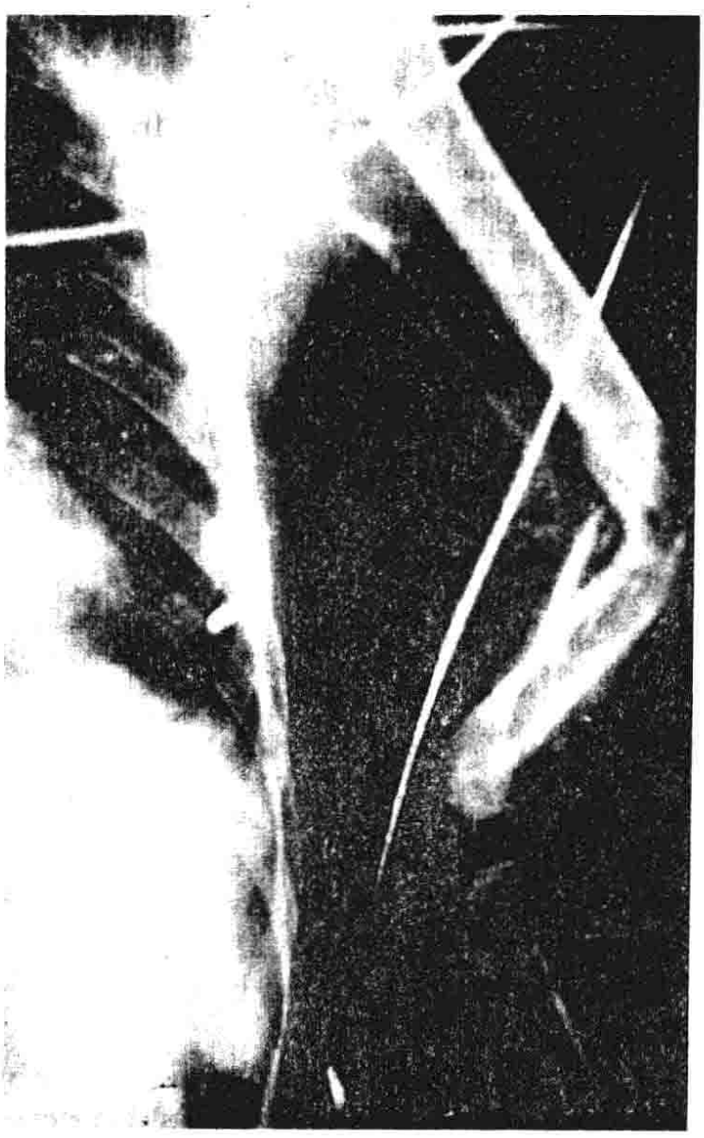


Fig. 9-8 Open grade III humerus fracture. Arteriogram shows the artery to be intact.

The growth plate is a cartilaginous disc situation between the epiphysis and metaphysis. Five layers have been described. The hypertrophic layer is the weaker and accounts for the site of disruption when the plate tears. Because of the wavy configurations of the plate, tears may damage other layers (Fig. 9-6).

The Salter-Harris classification is particular to children. It is an accurate method of describing growth plate injuries (Fig. 9-2):

Type I. Epiphysis separates completely from the metaphysis.

Type II. The line of fracture travels through much of the plate before passing through the metaphysis.

Type III. These fractures are rare. The fracture line passes along with the growth plate for a variable distance before entering the joint through a fracture of the epiphysis (Figs. 9-7 and 9-8).

Type IV. The fracture line passes from the joint surface, across the epiphysis growth plate, and into the metaphysis.

Type V. These are crush injuries and are therefore usually difficult to define. The growth plate is crushed frequently resulting in growth disturbance.

Special attention should be placed to Salter-Harris types III and IV because of their intraarticular component.

DIAGNOSIS

The most important part of fracture care in children is the diagnosis. If the physician fails to make the correct diagnosis, all other steps from here on are set off in the wrong direction. A significant proportion of poor results in children are attributable to this initial error.

Different tools can be used to arrive at the correct diagnosis. A good history by either the parents or the child, or both, can guide us in the right direction. The most important part, however, is a good physical examination; then, after the physician has an idea of the injured area, an appropriate radiologic evaluation should be performed.

Historical Diagnosis

Failure to Appreciate the Mechanism of Injury

The opportunity to make a good start should not be wasted. The injured child and the alarmed parents are impressed—positively or negatively—by the physician's initial behavior. The parents should be asked how the injury happened. The child should be told to point to the painful spot and to move the digits. It is surprising how much information can obtained by a proper history.

Certain fractures can be diagnosed by the mechanism of injury. Avulsion fractures of the anterior intercondylar eminence are seen usually between 8 and 15 years of age and present with a history of a recent accident—commonly a fall from a bicycle. This history should serve as a guide to the right direction, to a good physical examination (painful, swollen knee, with decreased range of motion), and to which radiograph to order. This type of fracture is best appreciated on the lateral radiograph, but both views should always be obtained.

A fall with the elbow extended can produce a fracture of the wrist; if there is more energy, it can produce a greenstick fracture of the olecranon with or without a fracture of the radial neck or avulsion of the medial epicondylar apophysis. A fall against the extended elbow places a varus stress on the forearm. The result is a greenstick fracture of the olecranon with lateral dislocation of the radial head (Monteggia lesion).

Bumper injuries can be determined on the basis of the patient's age. A toddler is usually run over by the car, sustaining head and multiple other injuries. As the patient gets older, hip fractures and pelvis fracture are seen, as in the 6- or 7-year-old group. In the 12- to 14-year-old, fractured femurs are most frequent (Fig. 9-9), and in the adolescent, knee injuries are sustained. Thus, with an adequate history, one can make a good start not only as a guide to the diagnosis but also to impress the child and parents in a positive way.

Clinical Diagnosis

Failure to Consider Adequately All Parameters of the Examination

The worst mistake is to take the history and send the child to the radiology suite without a good clinical examination and then have the child return without a pulse 1 hour later. The physical examination is of paramount importance.

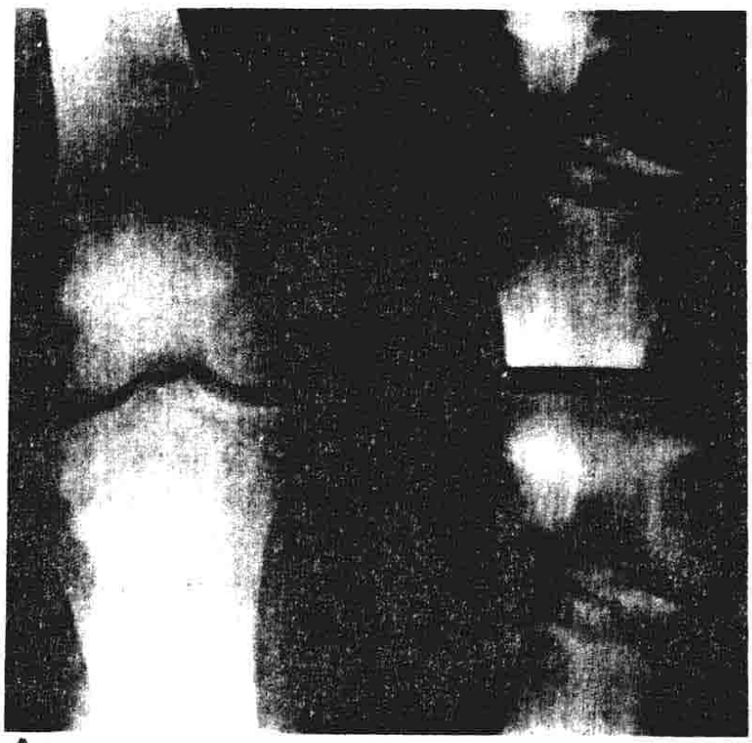
Palpation of the injured limb can give some guidelines as to the type of injury the child has. There are four signs: pain, deformity, swelling, and abnormal motion. The soft tissue should be examined. It is part of the injury. A fracture is defined as the breaking of bone as well as soft tissue. Injuries of the soft parts may be more important than the fractured bone. The physician should check around the limb to make sure there is not an opening, which would convert a closed fracture to an open fracture. The pulses are checked for vascular integrity. The neurologic status of the limb is checked and recorded if there is a change in the first few hours, which would mandate attention oriented toward the soft tissue treatment. Limbs have been lost because physicians have omitted the physical examination, and pulseless extremities have been placed in casts.

The fracture, hematoma, and soft tissue injury cause increased pressure within the limb compartments, ischemia of the musculature, and occlusion of the artery. It must be remembered that a compartment is like an envelope made up of bone, fascia, and interosseous membrane. Compartment syndromes tend to follow relatively minor closed fractures rather than the more severe injuries, in which the soft tissues are disrupted and decompression occurs. Compartment syndromes can be seen in the upper and lower extremities but are more commonly seen in the lower extremities, especially with tibia fractures.

Clinically there is more pain, the pain being out of proportion to the severity of the injury. Sensory deficit is another common finding. When the signs and symptoms are inconclusive, tissue pressures should be obtained. There are many methods of obtaining compartment pressure levels. At our center, we use the Stryker compartment pressure monitor. Fasciotomy is advised when the tissue pressure rises to 10 to 30 mmHg above the patient's diastolic pressure in the presence of any sign or symptom of a compartment syndrome.

Radiologic Evaluation

Radiology is crucial to the evaluation of fractures in children, but fractures in children are not always easy to visualize radiographically. Fractures may be missed if proper views are not requested. It is important to get as many views as necessary to rule out a fracture, and remember that



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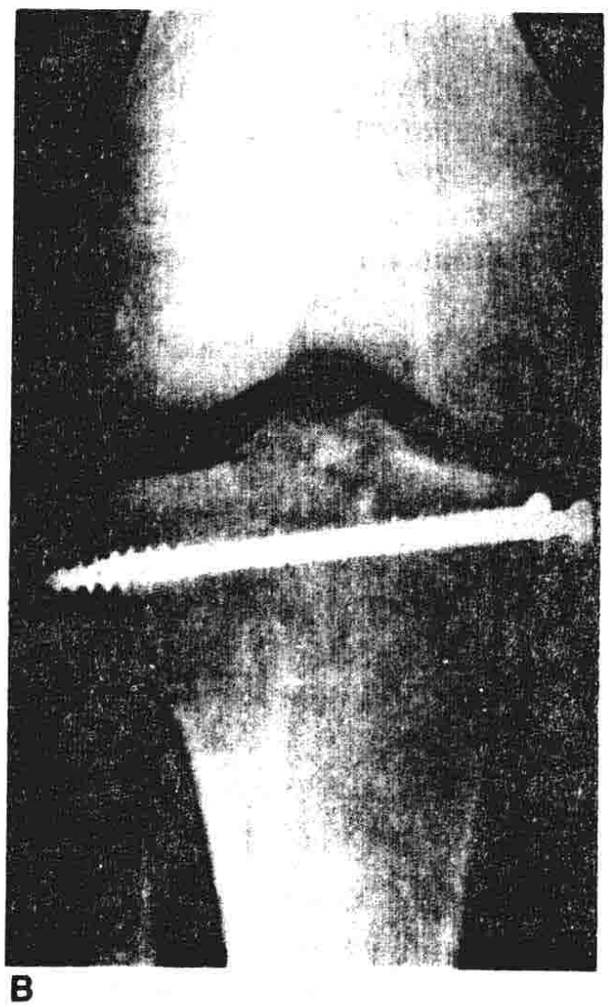

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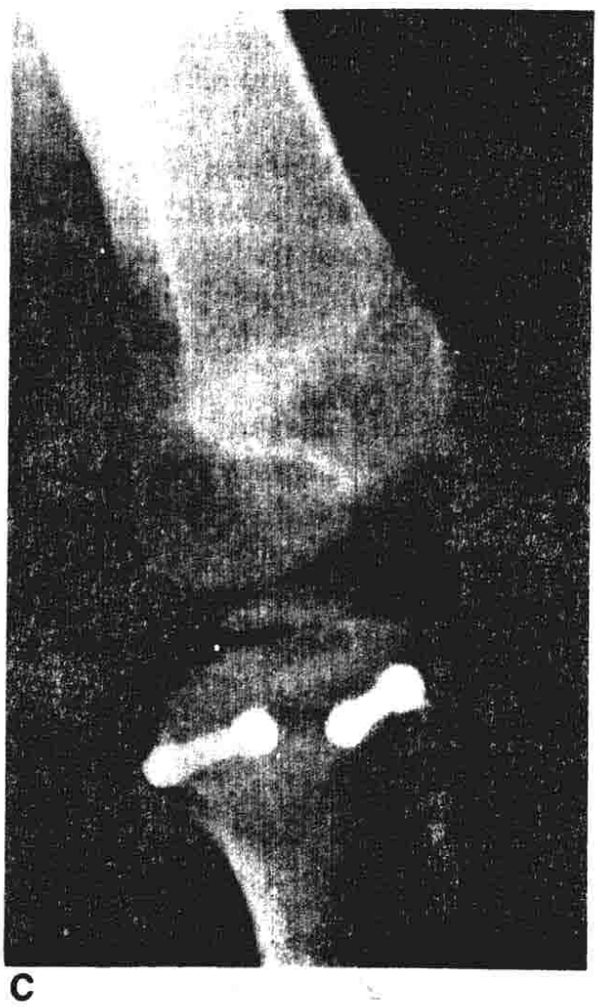


C

Fig. 9-9 (**A**) Anterior-posterior radiographs and tomograms showing the intraarticular fracture of the proximal tibia. (**B,C**) Patient had open reduction-internal fixation. (*Figure continues.*)

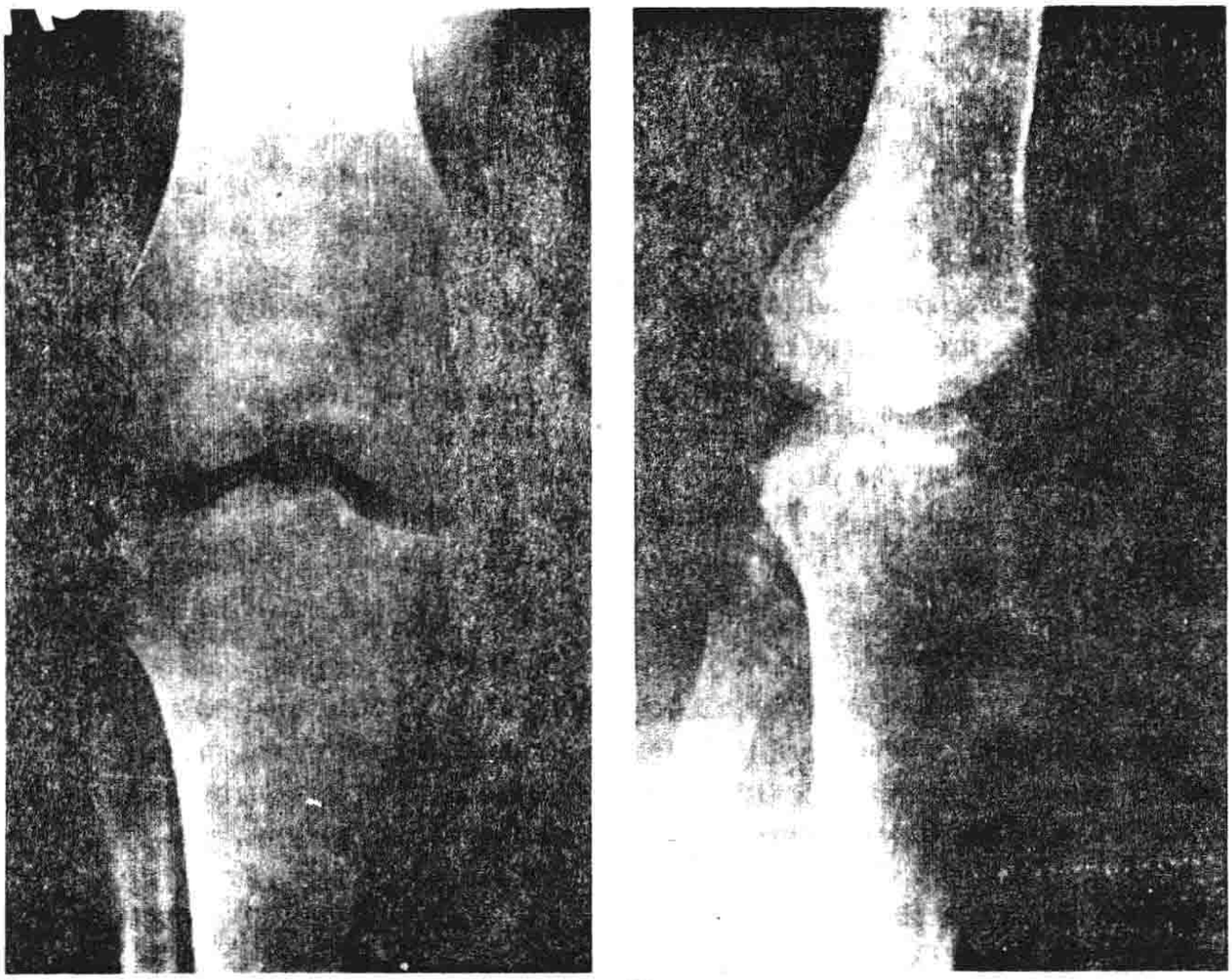


Fig. 9-9 (*Continued*). (**D,E**) Anterior-posterior and lateral radiograph (**D,E**). Radiographs show implant removal and fracture healed.

ultimately the diagnostic proof of a fracture is a radiograph showing the fracture. In evaluating radiographs, there are many pitfalls to avoid:

Adequate films. Any suspected fracture should be visualized in at least two views, anterior-posterior and lateral projections. Occasionally because of pain, deformity, and displacement, a true anterior posterior or lateral cannot be obtained, confusing even an experienced clinician. Technically poor films should never be accepted. Radiographs should show not only the grossly evident skeletal trauma but the normal and distorted soft tissue contours, fascial planes, and fat-fluid levels in the joint as well. Also, radiographs should show the joint above and the joint below the fracture, since dislocations are common with fractures of the diaphysis. An example is radial head dislocation with a fracture of the shaft of the ulna, otherwise known as the Monteggia fracture.

Splinted extremity. The extremity should be adequately splinted and protected prior to sending the child to the radiology suite. The splint should be adequate, supporting the joint above, and the joint below,

the injured bone. This is important because the technician may simply turn the unprotected part, producing two similar views.

Comparison views. (Fig. 9-3). Occasionally, the appearance of displaced apophysis may be so subtle that even an experienced observer may fail to recognize the injury initially. A comparison view is sometimes necessary to avoid excessive confusion, multiple reexaminations, and inappropriate treatment.

Stress films. Because of the elastic capacity of the child's bone and soft tissues, the injured part often springs back into its anatomic position. This is particularly common in epiphyseal fractures around the knee.[2] As in the skeletally mature patient in whom ligament injuries are tested using stress, in the skeletally immature patient stress may open a fracture to document the injury (Fig. 9-7).

Soft Tissues

Adipose tissue has a different specific gravity than the rest of the soft tissue and is more radiolucent. The shadows of fatty tissues may serve as a contrast density, partially outlining the margins of other soft tissues; thus, injury to such tissues is frequently concomitant to fractures and should be assessed as accurately as possible as part of the evaluation.

Special Techniques

Because of the difficulty in visualizing some fractures and epiphyseal contours, there is sometimes a need to use techniques other than routine radiography, if an absolute diagnosis is mandatory:

Arthrography. This modality is mainly used for chronic problems, but it has been used for acute fractures, especially in infants with a radiolucent distal humerus.

Computed tomography. This technique has been helpful in the diagnosis and management of spinal trauma.

Bone scan. This technique has been underused in children's fractures. It is simple and noninvasive, making it an attractive screening procedure. Bone scans in trauma have been used in detecting stress fractures before there is radiographic evidence of a fracture, permitting early diagnosis of these lesions.

Arteriography. Arteriography is especially helpful in adequately delineating vascular injury (Fig. 9-8).

Tomography. With the advent of the CT scan, anterior-posterior and lateral tomograms are used less frequently (Fig. 9-9).

Parents worry about the amount of radiation their child will be subjected to with radiographs. The *rad* is a unit that measures the amount of radiation absorbed by human cells. Radiation is considered low when it is below 1 rad. Most diagnostic x-rays are in low levels and are measured in millirads (one-thousandth of 1 rad). With today's equipment, physicians can

obtain more accurate information with less radiation to the patient. For example, a fractured femur will receive an average of six series of x-rays while in the hospital and about another six series (anterior-posterior views and lateral) before the fracture heals. According to the FDA's Bureau of Radiological Health, in each series the patient will receive about 3 or 4 mrad. This is a total of 48 mrad for the treatment of the fractured femur as compared with 100 mrad each American receives each year from cosmic rays, fallout, electronic products, and other sources.

TREATMENT

The Necessity of Relating to the Child and Family

Once the correct diagnosis has been made, the emergency physician can relate to the parents and to the child as to the options of treatment for the particular type of fracture, making them part of the decision-making. There are fractures that can be splinted and referred to the orthopedic surgeon and there are fractures that have to be treated on an emergency basis. The physician explains in detail the type of complications to be expected with the type of fracture, with the method of treatment, and if appropriate follow-up is not obtained. The object of the treating physician is the complete restoration of function with least risk, pain, and inconvenience to the child and the least amount of anxiety to the parents and physician.

Treatment of Specific Fractures

For treatment purposes, fractures can be divided in three groups: undisplaced, displaced, intraarticular fractures. By far most of these fractures can be managed successfully by closed methods, but there are a few indications for open reduction and internal fixation.

Undisplaced epiphyseal separations. These do not show on radiographs, but there is soft tissue swelling and tenderness over the growth plate. In children, sprains are rare, so a growth plate injury (Salter-Harris I) (Fig. 9-2) should be the diagnosis until proved otherwise and the treatment should be a cast for 3 weeks. These fractures need to be followed to make sure that there is adequate growth and that no growth plate closure has occurred.

Undisplaced diaphyseal fractures. These should be casted, including the joint above and the joint below, for a period of 3 weeks, and followed as closure of part of the growth plate or stimulation of the growth plate can occur. Adequate follow-up at 6-month intervals for the first year and then yearly visits for 3 years are adequate to pick up potential limb discrepancies.

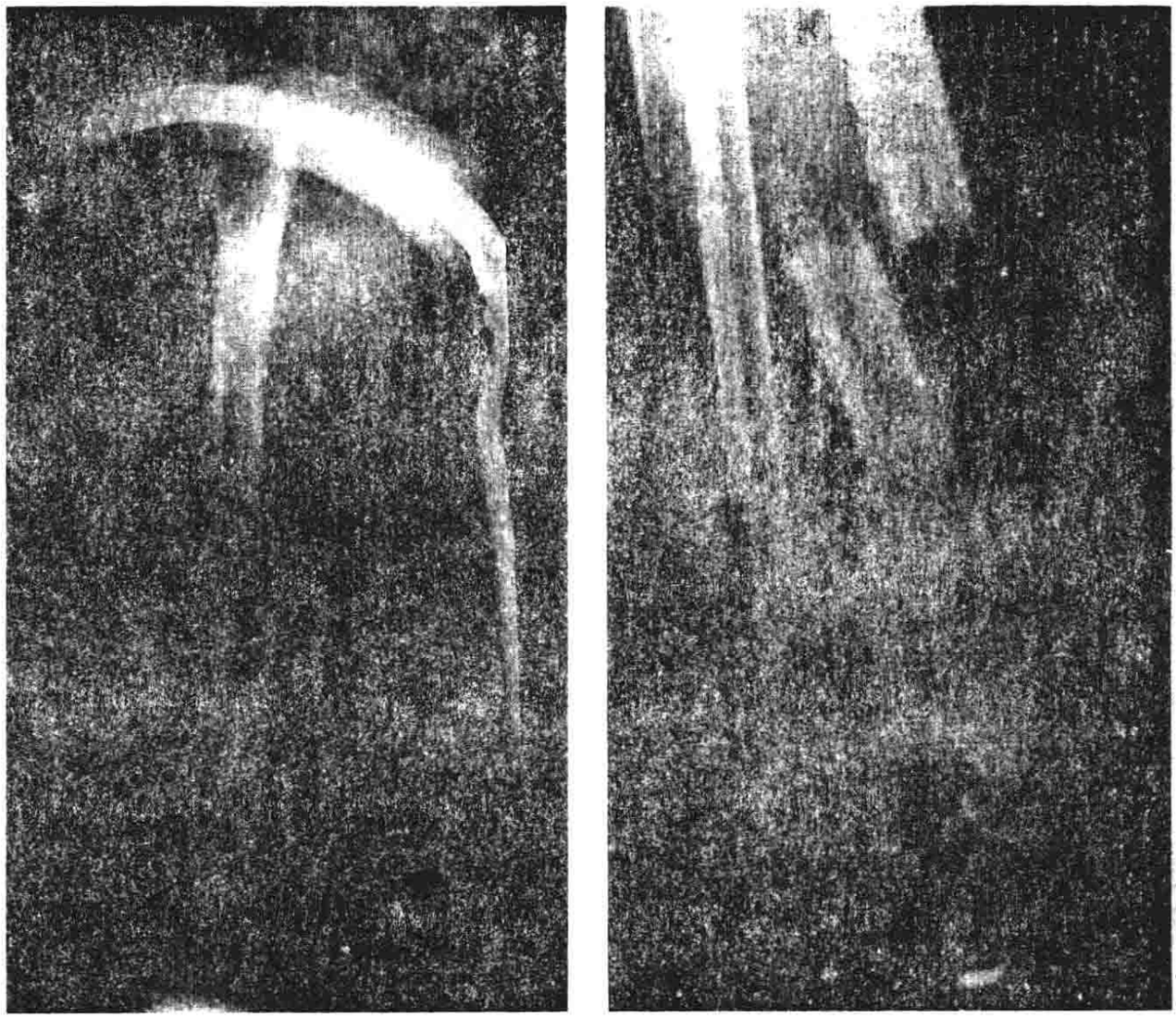


Fig. 9-10 An 11-year-old boy with a transverse fracture of the femur (**A,B**) that was treated in skeletal traction. (*Figure continues.*)

Displaced epiphyseal injuries. These can be managed by closed reduction and casting. The physician should explain to the parents and child about the technique of reducing the fracture, the type of anesthesia to be used, and the possibility that if the fracture cannot be reduced, the child might need to go to the operating room suite for a closed reduction, and possibly open reduction. An adequate anatomic reduction should be done in all children's fractures to prevent deformities and long-term disabilities. But the parents should be aware that a fracture is likely to remodel in children with two or more years of growth remaining, near the ends of the bones, in the plane of motion of the joint, in the event that the parents want to avoid manipulation and reduction. In treating this type of injury, I recommend one trial at the emergency suite and, if there is no success, the patient should be taken to the operating suite and given general or regional anesthesia. Two trials are given in the operating room; if reduction is not obtained, open reduction and casting or open reduction and internal fixation is the method of choice. The problem with serial closed reduc-

C

Fig. 9-10 (*Continued*). (**C**) Result 1 year after fracture healed.

tion is that every time it is tried, the growth plate is crushed, which can cause partial or total closure of the growth plate. After an adequate reduction, radiographs should be taken and the alignment and rotation should be matched with that of the intact limb.

Displaced diaphyseal fractures. In children these fractures can be treated by initial casting or occasionally by a period of traction and then casting (Fig. 9-10). Bending and lateral displacement by the width of the shaft are permissible, but rotation at the fracture should be fully corrected. Remodeling will not correct rotational deformities. Excessive longitudinal growth is regularly seen following fractures of the diaphysis; this is compensated for by permitting the fracture to unite with slight initial shortening. Severe angulation should be avoided and rotational deformity should be fully corrected. This is seen in forearm fractures, where bayonet apposition is acceptable and in femoral shaft fractures, where 1 cm overriding is acceptable. Cast immobilization of displaced shaft fractures always should include the joint above and the joint below. In the lower limb, 6 weeks of immobilization may be wise before unprotected weight bearing is allowed. When closed reduction fails, a period of traction or open reduction may be indicated. Difficulty in reduction is usually caused by the interposition of periosteum, muscle or tendon. Open reduction is necessary to remove these tissues from the fracture gap.

Nondisplaced intraarticular fractures. In evaluating intraarticular fractures, it must be determined whether there is an osteochondral fragment or a Salter-Harris III or IV fracture. Intraarticular fractures of less than 1 mm displacement can be treated by casting and observation. In triplane fractures of the ankle, a 2-mm gap is an indication for surgical reduction.

Displaced intraarticular fractures. When there is a loose osteochondral fragment or rotation and it is large enough to reattach, this should be done either by arthroscopic technique or by open reduction and internal fixation. If the fragment is small, surgical removal is a must because it might produce mechanical locking of the joint. Intraarticular Salter-Harris fractures (III and IV) with a displacement of more than 1 mm and joint incongruity should have open reduction and internal fixation. This will not only align the joint surface but will also align the growth plate to prevent bony bar formation and partial closure of the growth plate (Figs. 9-7, 9-9, and 9-11).

Timing of Treatment

Children's bones react more quickly to a fracture than an adult's bones. Within a few days, the bony fragments are joined by callus and a precise reduction is no longer possible. A common mistake is to see the child with a displaced fracture in the emergency department and have the child splinted and referred to the orthopedic surgeon. Because of problems with the parents or lengthy appointment time, the child is seen 2 weeks later with a fixed deformity. Delays may complicate treatment and spoil end results. The emergency physician must establish a functional referral basis with the orthopedic surgeon to avoid this outcome. Whether fractures are splinted and referred or casted initially will be determined by this relationship as well as each individual situation.

Closed Method of Treatment

Splints

A splint should be placed in a fracture only as a temporary method of immobilization. The splint should be placed after the fracture has been satisfactorily reduced or to transfer a patient prior to reduction. Before applying the splint, the extremity should be well padded and free from gaps and folds. The splint is then applied; it is important to remember in stabilizing a fracture that the splint or cast should include the joint above and the joint below.

There are many types of commercial splints, but a simple plaster-of-paris splint is usually sufficient. The physician should make sure that there are 15 layers of plaster to render it strong enough before the child is discharged from the emergency department.

Most children's fractures can be managed successfully by closed reduction, plaster immobilization, or traction treatment. There are two types of cast application: primary and secondary.

Primary cast applications. A primary cast should be applied when the fracture has been satisfactorily reduced and the limb is in the desired position, before the plaster is rolled. The cast should be applied in stages. The cast is never applied on a moving screaming child. The first stage after reduction is to apply a padding layer that is free from gaps and folds; additional padding is applied over bony prominence. The second stage is to apply the plaster of paris. It is applied in a circular fashion, forming a tube at the level of the fracture. This will hold the fracture temporarily in the required position. The water should be cold before the plaster bandage is dipped into it; cold water allows more time for molding. The tube is extended to the joint proximally and distally. It should include the joint above and the joint below. Now three-point molding is applied to make use of the periosteal hinge and reduce the fracture (Fig. 9-1). The parents should be instructed to elevate the extremity and check the circulation and the innervation distal to the fracture. It should be checked frequently and thoroughly. If there is any question about the circulation and swelling of the limb, the child should be admitted for observation. If the child is uncooperative, something is wrong. The cast should be split and the extremity checked. It is better to lose reduction than the limb. If that does not help, compartment pressures should be obtained; if elevated, fasciotomy should be considered, as a next stage.

Secondary cast application. A replacement cast is applied when the previous one no longer fits or following a period of traction. For the secondary cast, one should wait until the initial swelling has subsided. The primary cast is usually worn for the first week to 10 days. It is then changed to the secondary cast and the patient then returns in 3 weeks for a radiograph out of plaster. It is important to obtain radiographs after each cast change to make sure there are no displacement or angulations. For the lower extremities, the cast is changed at 4 weeks, and a third cast is applied. At 7 weeks, unprotected weight-bearing is allowed.

Traction

Traction is less frequently used nowadays but is still the method of choice for femoral shaft fractures.

The type of traction used depends on the age, weight, and anatomic location. In the child under 2 years of age, we use skin traction (Bryant's traction or modified Bryant's traction), making sure that the traction tape will not cause skin irritation and limb compression. The prominent areas are covered with padding and the traction tapes are held in place with an

elastic bandage. The tape should be applied over the knee joint to distribute the sheer force over a sufficiently large area to ensure maximum comfort and safety.

In the child over 2 years, I recommend split Russell's traction. In the child over 10 years of age, balanced skeletal traction with femoral and tibial pins is the treatment of choice. In proximal subtrochanteric fractures, femoral 90/90 pin traction is the treatment of choice. After 4 or 5 weeks of skeletal traction, the child is placed in hip spicas; the older child is given cast braces.

Open Treatment of Fractures

Rang believes that open reduction should follow failed closed reduction in fractures of both bones of the forearm, in teenagers, tibial spine fractures, and radial neck fractures. Rang has also stated that open reduction with internal fixation should be the initial method of treatment in fractures in which remodeling would not help: displaced intraarticular fractures, in the multiple traumatized child, and in the child with head injuries. By early immobilization of the fracture, the child can be mobilized.

Internal fixation devices should be used as infrequently as possible and should be easily removable. Smooth Kirschner wires and screws are preferred. This should be done by an experienced orthopedic surgeon. Injuries to the growth plate can occur.

At the Orthopaedic Clinic of the Cantonal Hospital in St. Gall, Switzerland, a study of 891 fractures in children was undertaken to see the percentage of open reduction and internal fixation in children fractures. Internal fixation was carried out in 16.1 percent (145 fractures) of cases.[6]

Complications

Failure of Early Diagnosis

If an incorrect diagnosis is made, everything that follows will be wrong. This is especially true of growth plate injuries, in which the cartilage is not fully ossified and hard to evaluate in simple radiographs. Here is where additional studies should come into play; arthrograms for distal humerus fractures and knee injuries, tomograms, and so forth.

A displaced intraarticular fracture can give the child deformities secondary to growth plate malalignment, with angular deformities and growth discrepancies. Therefore, early diagnosis, and the correct diagnosis, is a must.

Failure of Early Treatment

A displaced intraarticular fracture or a displaced shaft fracture should be seen by an orthopedic surgeon as soon as possible and treatment instituted if there is no contraindications. Fractures in children heal more

quickly than in adults. Within a few days, a fracture that was initially reducible becomes fixed in deformity and, to make it acceptable, a surgical procedure is indicated. As a general principle, fractures should be always reduced as early as possible, to prevent soft tissue swelling.

Failure of Treatment Method

The physician should make it a point to explain to the parents the different methods of treatment and the possibility of having to do open reduction if closed reduction is unsuccessful. It is difficult to tell the parents that the child has to go to surgery for an open reduction after they have been told that the fracture is a simple one that can be taken care of in the emergency department.

In attempting closed reductions, the child should be completely relaxed with intravenous sedation and a one-try done in the emergency department. Forceful manipulations of growth plate injuries can produce crushing of the plate. This may produce long-term problems of angular deformities. If an easy reduction cannot be achieved by sedation in the emergency department, the child should be given general anesthesia and complete relaxation. Open reduction and internal fixation should be done by an experienced orthopedic surgeon. It is crucial to avoid injuries to the growth plate and vascular supply with pins and screws.

Special Problem Fractures in Children

Child Abuse

Approximately one-third of all physically abused children require orthopedic management. Injuries to the long bones, ribs, and skull are the most common locations.[2,3]

Fractures may be classified as epiphyseal, metaphyseal, diaphyseal, and other types:

Epiphyseal fractures. These are rarely seen in the femur, tibia, and humerus. A fracture through the growth plate, especially when the epiphysis is not visible, may cause difficulty in diagnosis. Arthrographic examination of the joint may be helpful in certain cases.

Metaphyseal fractures. These are more common and are classified into impaction, buckle fractures, corner fractures (avulsion fractures), and the rare metaphyseal fractures. Corner fractures are caused by forceful downward pull of the extremity and are often bilateral. They may not be large enough to be visible in the first radiograph, but they result in periosteal separation and hemorrhage that will cause periosteal new bone formation that will be visible in about 7 to 10 days. Buckle fractures are common in the metaphyseal area and often are multiple.

Diaphyseal fractures. These are divided into three types:

Spiral or oblique fractures of the shaft—the twisting type of fractures

Multiple lesions in various stages of healing
Gross bony deformity

Spiral or oblique fractures in children, especially in nonambulatory children, are suggestive of child abuse. Many of these fractures leave permanent sequelae of gross deformities. This is partly because the parents do not seek medical care and because some of these fractures heal with rotatory or angulatory deformities. Some children may require reconstructive surgery during adult life. Fractures involving the ribs are usually posterior and posterolateral and may be in various stages of healing. As a last word for the abused child, consultation by an orthopedic surgeon is often helpful also from the legal standpoint.

Fractures of the Shoulder Girdle

Fractures of the clavicle are benign, but if they are of the outer third, they are equivalent to the acromioclavicular separations and may benefit from closed percutaneous K-wire fixation.

Fractures of the Proximal Humerus

Proximal epiphyseal separation of the humerus with less than 40 degrees of angulation can be treated in a sling, while more than 40 degrees of angulation requires closed reduction and percutaneous K-wire fixation.

Fractures of the Elbow

Fractures of the elbow in children are among the most difficult to diagnose. Errors in diagnosis in children under 2 years of age can complicate treatment with malunions and deformities. There are two types of fracture of the elbow:

Supracondylar fractures. These are fractures of the distal humeral metaphysis just above the epicondyles. They usually occur from forced hyperextension or hyperflexion of the elbow. Posterior and medial displacement of the distal fracture fragment with respect to the proximal humerus is the most common finding. One in five has a nerve injury, and subsequent complications can occur if this fracture is not treated by an experienced orthopedist. Accurate reduction is essential. A number of methods are employed in treatment, including closed reduction, overhead traction, and open reduction with internal fixation. The choice depends on the degree of displacement, the severity of associated swelling and soft tissue injury, and the experience of the physician in charge. After reduction, a long arm cast with the elbow flexed is of utmost importance, the neurovascular have to be checked because of the swelling. During the early phases of healing, displacement may occur and closed follow-up is necessary.

Fractures of the Lateral Condyle. The lateral condyle of the distal humerus or capitellum articulates with the radial head and forms the lateral half of the elbow joint. Fractures of the lateral condyle may be a Salter-Harris type III or IV. Lateral condyle fractures are intraarticular fractures. Anatomic reduction is therefore essential (Fig. 9-11).

All displaced fractures require open reduction and fixation with K wires. Even slight displacement is unacceptable. Malunion and nonunion lead to progressive valgus angulation at the elbow joint with significant late deformity.

Fractures of the medial condyle. The medial condyle or trochlea articulates with the proximal ulna and forms the medial half of the elbow joint. Isolated medial condyle fractures are uncommon. As with other articular fractures, anatomic reduction is necessary, and open reduction and internal fixation with K wires is usually the treatment of choice.

Fractures of the medial epicondyle. The medial epicondyle is a secondary ossification center on the medial aspect of the distal humerus. They are intraarticular and may result from forced lateral rotation of the forearm or medially directed of the extended elbow. There is controversy over the best method of treatment. Most surgeons will operate to reduce the duration of disability and to secure union.

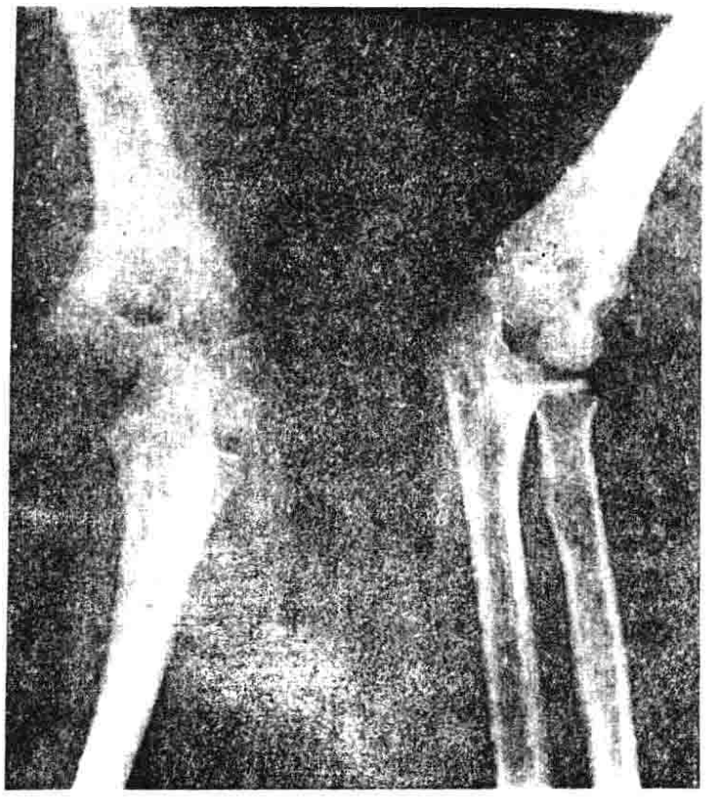
Fractures of the radial neck. Fractures of the proximal part of the radius are usually associated to other type of fractures. Nondisplaced fractures are treated in a cast. When there is more than 20 degrees of angulation, reduction is required, preferably closed if possible. Failing closed reduction, open reduction and internal fixation may be necessary.

Fractures of the olecranon. These fractures are usually intraarticular. More than 2 mm of displacement or a gap greater than 2 mm is best managed with open reduction and internal fixation.

Fractures of the Wrist

Buckle or torus fractures of the distal radial and ulna metaphysis are common in children. They frequently result from minor trauma (Fig. 9-3). Salter-Harris types I and II are common epiphyseal injuries of the distal radius and ulna. In most cases, the distal radial and ulna fracture fragments are displaced dorsally with resultant open volar angulation. Closed reduction is usually successful in closed fractures after intravenous sedation or Bier block. In open fractures, surgical debridement will be required, no matter how small the wound.

After reduction, immobilization in a bivalved long arm cast or long arm splint is appropriate to prevent neurovascular compromise from postreduction swelling. This is used for 3 to 4 weeks. In the older child a splint or short arm cast is then used for an additional three to four weeks.



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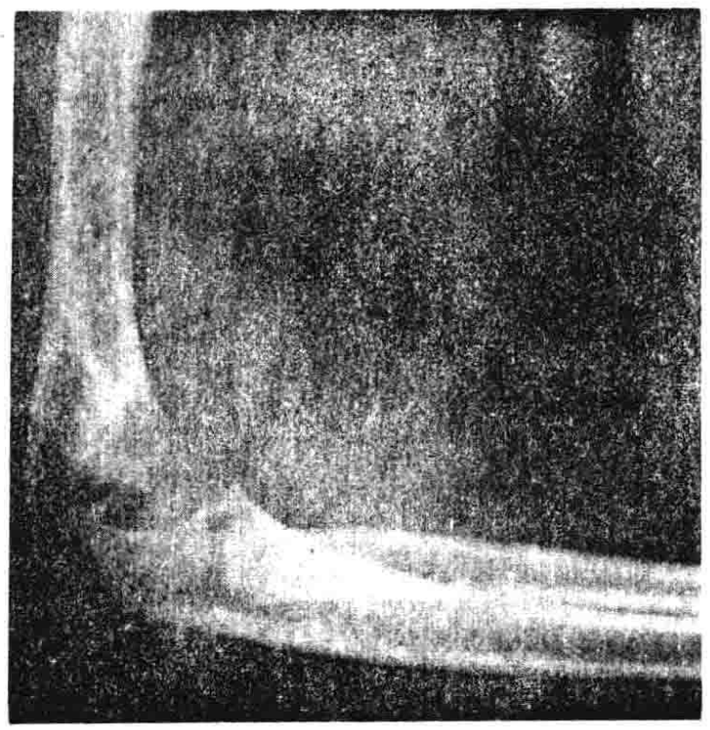

B

Fig. 9-11 (**A,B**) Patient with lateral condyle fracture. (*Figure continues.*)

Fractures of the Pelvic Girdle

Fractures of the pelvis and acetabulum. These fractures in children are usually the result of significant violence.[7] Automobile accidents are the most frequent cause. These are usually associated with multisystem injury, and the orthopedic component is but a part of the total team approach to the injured child.

There are three primary centers of ossification in the pelvis, one each

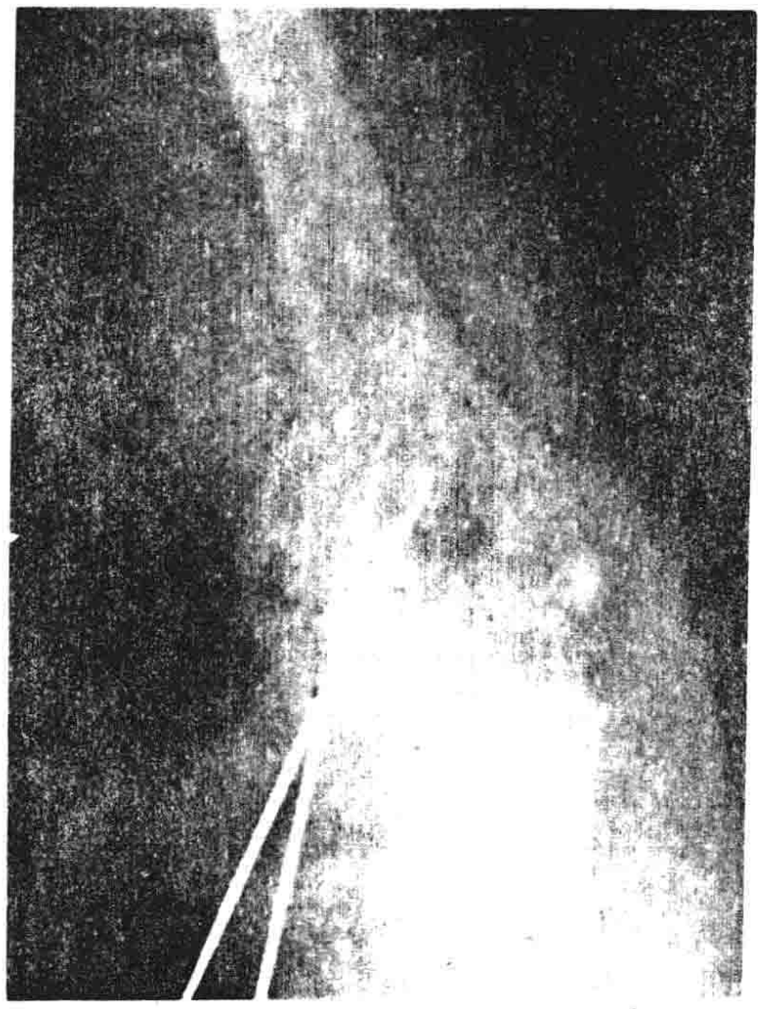

Fig. 9-11 (*Continued.*) (**C**) Intraoperative radiograph of the fragment reduced and internally fixed.

for the ischium, the pubis, and the illium. These meet at the triradiate cartilage, where fusion takes place between 18 and 23 years of age (Fig. 9-12). Secondary centers of ossification may occur in several areas of the pelvis, such as the illiac apophysis, the inguinal apophysis, the anterior inferior spine, pubic tubercle, the crest of the pubis, and the inguinal spines.

Fractures of the pelvis in children are classified as stable and unstable fractures. Stable fractures are avulsions of the secondary ossification centers, stable anteroposterior compression (open book), and lateral compression (triradiate cartilage injuries) (Fig. 9-12). Unstable fractures are classified as vertical shear fractures, as in the adult.

Stable pelvic fractures require little treatment in children. In many cases, only a brief period of bed rest is necessary. Walking may be started when the child is comfortable. Unstable or displaced fractures may require skeletal traction or open reduction to achieve alignment. Emergency external fixation have been utilized not only to stabilize the pelvic fragment but also to produce a tamponade effect in a bleeding pelvic fracture.

Fractures involving the acetabulum or lateral compression fractures of the pelvis, when they are not displaced, may cause early closure of the triradiate cartilage and may produce an acetabular dysplasia; therefore, parents may be warned of the possibility (see Fig. 9-12). Displaced fractures must be restored as nearly as possible to anatomic alignment. Internal fixation may be required.

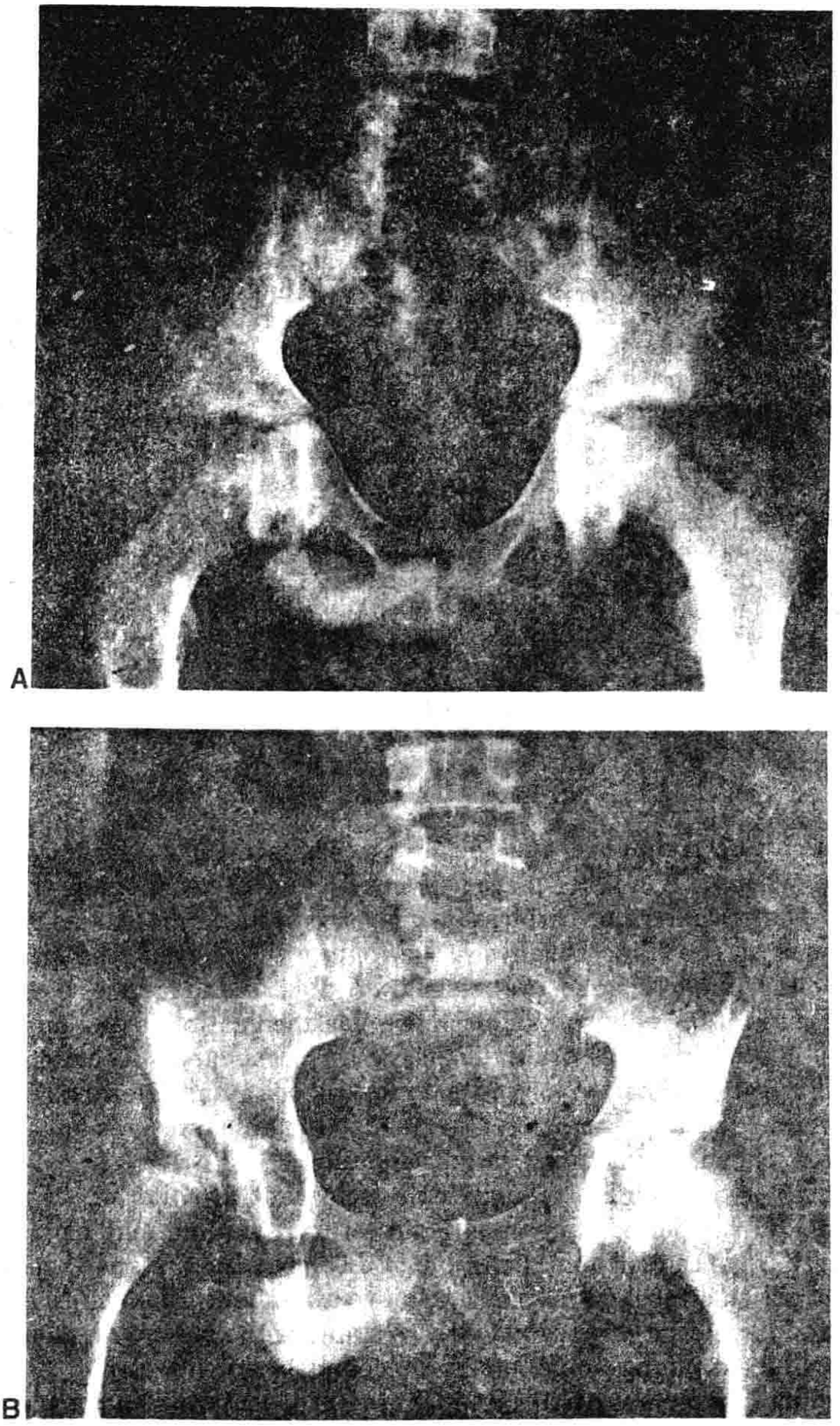

Fig. 9-12 A 7-year-old boy sustained an injury to the lateral aspect of the right hip. (**A**) Radiograph taken at the time of injury was read as normal. (**B**) Patient returned at age 16 with a bump in the lateral aspect of the right hip, and was found to have subluxation of the hip and premature closure of the triradiate cartilage.

Fractures of the hip. Fractures of the hip in children, like fractures of the pelvis, are most often the result of automobile accidents. Proximal femoral fractures may occur through the proximal growth plate, the femoral neck, and the intertrochanteric region. Hip pain, limitation of active motion, and resistance to passive motion are the consistent findings.

The diagnosis can be confirmed with anterior-posterior and lateral radiographs of the hip. It is not necessary to move the injured extremity to obtain these views. Attempts to move the injured extremities to obtain the radiograph are painful and unnecessary.

Anatomic reduction of the fracture fragments must be obtained followed by immobilization. Although closed reduction and spica cast immobilization are sometimes successful, open reduction and internal fixation are often necessary. Physical and neck fractures are often associated with damage to the vessels that supply the proximal epiphysis; the potential for avascular necrosis is high with these fractures. Children with hip fractures should be followed until they have reached skeletal masturity.

Fractures of the Knee

Fractures of the distal femur and proximal tibia are common in children. They are more frequent than ligamentous injuries. In children, a ligament disruption is the last diagnosis to make. Meniscal injuries are also rare.

Fractures of the distal femur. Fractures through the distal femoral growth plate may mimic knee ligament injuries in clinical appearance. Stress radiographs are necessary to distinguish the type of fracture and open reduction and internal fixation may be necessary. Even with anatomic reduction, the risk of angular deformity secondary to growth plate injury is high (Fig. 9-7).

Fractures of the proximal tibia. Because of the unique configuration of the growth plate separating the secondary ossification center of the proximal tibial epiphysis from the tibial metaphysis, avulsion fractures of the tibial tubercle occur occasionally in adolescence after athletic, bicycle, or motor vehicle accidents. The injuries are probably the result of violent quadriceps contractures against a flexed knee. The fracture in some cases stops short of the anticular surface. In others, the fracture extends into the joint. Minimally displaced fractures are treated in a cast. Fractures with wide separation and joint incongruity are better treated by open reduction and internal fixation.

Fractures through the anterior or posterior tibial spines may occur if load applied through the cruciate ligaments exceed the failure limits of underlying bone. Posterior spine fractures are much less common than ante-

rior spine fractures. These avulsion fractures are usually displaced and rotated 360 degrees; adequate reduction and internal fixation are required.

Proximal tibial physeal fractures are rare injuries and are usually the result of motorcycle or athletic accidents. Injuries to the popliteal vessels to the posterior border of the proximal tibia are high, and damage to the peroneal nerve may occur at the time of bony injury or as result of subsequent swelling. If distal pulses are not present, arteriography and surgical exploration may be necessary to restore blood flow.

The treatment of proximal growth plate injuries depends on the type. Salter-Harris type I and II fractures can usually be treated nonoperatively when vascular compromise is not present. Salter-Harris type III and IV fractures are intraarticular fractures and, when these are joint incongruity, open reduction and internal fixation are necessary (Fig. 9-9). The incidence of premature plate closure is high, and late joint incongruity may result in deformity and premature degenerative disease.

Other types of fractures are osteochondral fractures of the tibia or distal femur. These fractures may require open arthrotomy and removal of the fragment. If left alone, they can produce locking and joint destruction.

Fractures of the ankle joint. Fractures around the ankle joint are usually the result of a combination of twisting and bending forces. Ankle fractures require careful orthopedic treatment. The ankle is one of the major weight-bearing joints; residual joint incongruity is poorly tolerated.

Careful radiologic evaluation of patients with suspected ankle injuries is essential. Anterior-posterior, lateral, and oblique views should be obtained. Intraarticular fractures must be treated by open reduction and internal fixation. Complications are frequent.

SUMMARY

In treating children's fractures, a few guidelines should be kept in mind. First, the line of fracture should be defined and classified. This will determine the management of pediatric fractures. Next, adequate radiographs and other tests are obtained, if necessary. It is crucial to discuss with the child and parents the treatment options as well as the complications, risks of growth disturbance, the method of reduction, and follow-up. Closed reduction is done in most Salter-Harris type I and II injuries. A few will prove impossible to treat by closed method. Open reduction may be necessary. Open reduction is a must for intraarticular fractures, Salter-Harris types III and IV. Immobilization of fractures should include the joint above and the joint below. Three weeks is usually enough. In the lower extremity, 6 weeks of immobilization may be necessary before weight-bearing is allowed. Finally, whether a fracture is splinted and referred by the emergency physician or seen initially by the orthopedist, follow-up is essential. It must

be stressed to the parents that initial treatment is not necessarily definitive and that, depending on the type of fracture, the child may have to be seen repeatedly to ensure a good outcome.

REFERENCES

1. Hohl JC: Fractures and other injuries in children. Orthop Clin North Am 7:527, 1976
2. Ogden JA: Skeletal Injury in the Child. Lea & Febiger. Philadelphia, 1982
3. Rockwood CA, Wilkins KE, King RC: Fractures in Children. JB Lippincott, Philadelphia, 1984
4. Rang MC: Children's Fractures. JB Lippincott, Philadelphia, 1974
5. Salter RB, Harris WR: Injuries involving the epiphyseal plate. J Bone Joint Surg 45A:587, 1963
6. Weber BG, Brunner CH, Freuler F: Treatment of Fractures in Children and Adolescents. Springer-Verlag, Berlin, 1980
7. Tile M: Fractures of the Pelvis and Acetabulum. Williams & Wilkins, Baltimore, 1984
8. Rang MC, Willis RB: Fractures and sprains. Symposium on Common Orthopaedic Problems. Pediatr Clin North Am 24:749, 1977

10 Problems in the Management of Vomiting, Diarrhea, and Dehydration

Roger M. Barkin

Acute infectious diarrheal disease in children is common, accounting for approximately 4 percent of outpatient visits and 3 to 5 percent of inpatient admissions. Together with hemorrhagic shock from trauma, dehydration secondary to gastrointestinal (GI) losses represents the most common cause of reversible shock in infants and children. Appreciation of this fact, as well as early recognition and appropriate treatment of the dehydrated patient, may prevent further progression of the disease process. Diarrheal disease is caused by a wide range of viral, bacterial, and parasitic agents. Rotavirus or reovirus accounts for as many as 39 percent of pediatric admissions due to diarrheal disease and for 22 percent of such disease in ambulatory children. It is the etiologic agent in 70 to 80 percent of diarrheal disease in winter and 20 percent of cases in summer. *Salmonella, Shigella, Campylobacter,* and *Giardia* are also important pathogens.[1-4]

The child with vomiting and diarrhea is frequently dehydrated and requires urgent fluid therapy. The management of such patients requires a correlation of historical, physical, and laboratory data with pathophysiologic principles. The therapy must combine replacement of fluid deficits with the concurrent administration of maintenance fluids.

Specific concerns in evaluating the significance of vomiting or diarrhea in the child reflects the relative difficulty in the subjective or objective assessment of fluid losses. Abnormal losses may develop rapidly; reduction of the abnormal losses may be difficult. This chapter addresses common problems, pitfalls, and difficulties in the recognition and treatment of the dehydrated patient.

RECOGNITION OF THE DEHYDRATED INFANT

Failure to use Clinical Parameters to Determine the Extent of Dehydration

It cannot be overemphasized that the assessment of an infant for dehydration is a clinical determination. Laboratory data should generally be used to confirm the diagnosis. For this reason, therapy, especially therapy for shock secondary to vomiting, diarrhea, and dehydration, should be initiated without awaiting the results of laboratory values.

Correlation of a child's clinical status and laboratory data determines the type of therapy required after initial volume expansion. The physical examination is helpful in determining the magnitude of dehydration and must reflect a careful evaluation of mucous membranes, skin turgor, fontanelle fullness, as well as vital signs, specifically focusing on hyperapnea, orthostasis, and tachycardia. Clinical recognition must include the integration of numerous findings. Recent weights are rarely available for comparison, nor are they necessarily accurate unless done on the same scale, by the same person, with the infant fully undressed.

The findings in the child with only very mild dehydration (less than 5 percent) may be subclinical. At 5 percent dehydration, decrease in mucosal membrane moisture and prominence of the papillae of the tongue may be noted. Table 10-1 lists the clinical signs and symptoms encountered with various degrees of dehydration. For the purpose of this chapter, mild dehy-

Table 10-1. Degree of Dehydration

	Mild (<5%)	Moderate (10%)	Severe (15%)
Signs and symptoms			
Dry mucous membrane	±	+	+
Reduced skin turgor	−	±	+
Depressed anterior fontanel	−	+	+
Sunken eyeballs	−	+	+
Hyperpnea	−	±	+
Hypotension (orthostatic)	−	±	+
Increased pulse	−	+	+
Laboratory			
Urine			
Volume	Small	Oliguria	Oliguria/anuria
Specific gravity[a]	≤1.020[b]	>1.030	>1.035
Blood			
BUN	Within normal limits[b]	Elevated	Very high
pH (arterial)	7.40–7.30[b]	7.30–7.00	<7.10

[a] Specific gravity can provide evidence that confirms the physical assessment.
[b] Not usually indicated in mild dehydration.
+, present; −, absent; ±, variable.

Table 10-2. Body Fluid Compartments by Age

	Newborn	1 yr	3 yr	9 yr	Adult
Weight (kg)	3	10	15	30	70
Body surface area (m^2)	0.2	0.5	0.6	1	1.7
Body surface area ÷ weight	0.07	0.05	0.04	0.03	0.02
TBW (%)	78	65	—	—	60
ECF (%)	45	25	—	—	20
ICF (%)	33	40	—	—	40

TBW, total body weight; ECF, extracellular fluid; ICF, intracellular fluid.
(Finberg L, Kravath RE, Fleischman AR: Water and Electrolytes in Pediatrics. WB Saunders, Philadelphia, 1982.)

dration is defined as less than 5 percent, moderate as 5 to 10 percent, and severe as greater than 10 percent dehydration.

Clinically, the assessment of the degree of dehydration may be roughly delineated on the basis of defined parameters. However, because of the excellent ability of children to compensate for decreased vascular volume, abnormalities in vital signs may not be immediately evident. Vital organ functions are maintained by intrinsic compensatory functions that stabilize vital signs. As fluid loss progresses, venous capacitance may decrease by 10 to 25 percent, fluid shifts from the interstitial to intravascular compartments, and arteriolar constriction increases. With compensation, the central venous pressure (CVP) is decreased, as are stroke volume and urine output; heart rate and vascular resistance increase. Orthostatic changes may be the only evidence of these compensatory mechanisms. An acute volume loss of 10 percent may be marked by an increase in pulse rate of 20 beats/min, and a 20 percent deficit has an associated heart rate increase of 30 beats/min. A gradual volume loss produces minimal physiologic changes, while deficits of 20 to 30 percent are marked by compensatory alterations.

The clinical effect of fluid loss reflects the distribution of water loss within compartments. The percentage of total body water decreases with age, while the relative percentage of intracellular fluid increases (Table 10-2).

Table 10-3. Rapidity of Evolution of Dehydration and Compartment Fluid Source

Period of Evolution of Dehydration	Portion of Fluid Lost (%)	
	From ECF	From ICF
Rapid: <2 days	75	25
Average: 2–7 days	60	40
Long: 7 days	50	50

ECF, extracellular fluid; ICF, intracellular fluid.
(Barkin R: The dehydrated child: A management approach. J Emerg Med 1:399, 1984.)

Table 10-4. Maintenance Water Loss Components[a]

Component	Age Group			
	0–6 mo	6 mo–5 yr	5–10 yr	Adolescent
Insensible	40	30	20	10
Urinary	60	60	50	40
Fecal	20	10	—	—
Total	120	100	70	50

[a] In ml/kg/24 hr.

(Barkin R: The dehydrated child: A management approach. J Emerg Med 1:399, 1984.)

Compartmental distribution of fluid losses reflect the period of evolution of the dehydration. In general, 60 percent of fluid is lost from the extracellular fluid (ECF), while the remaining 40 percent comes from the intracellular fluid (ICF). With a more acute progression, the relative percentage lost from the ECF increases. (Table 10-3).

In determining fluid therapy, it is essential to understand normal maintenance fluids; daily water and electrolyte losses from the skin and the respiratory, urinary, and gastrointestinal (GI) tracts must be replaced. Insensible losses (approximately 30 ml/kg/day) occur through the skin and respiratory tract. About two-thirds results from skin loss. The rate of insensible loss is affected by humidity, body temperature, respiratory rate, and ambient temperature (Table 10-4). Insensible water loss increases also by 7 ml/kg/day for each degree rise in temperature above 37.2°C (99°F)[7,8] (see also Tables 10-5 and 10-6).

Limitations of Laboratory Confirmation

Laboratory data may provide confirmation of the clinical assessment of the degree and type of fluid deficit, but clinical assessment determines the urgency of emergency therapy. Examination of the urine with respect to volume and specific gravity can be useful. Urine specific gravity increases markedly in children with any degree of fluid deficit, and even minimal dehydration produces marked concentration. However, it does not provide a means of assessing the magnitude of any deficit. Serum electrolytes and BUN provide additional parameters. The BUN should decrease by at least 50 percent during the first 24 hours of appropriate rehydration (see Table 10-1). Restrictions in the use of laboratory data are further delineated in Chapter 2.

Table 10-5. Water Requirements on a Daily Basis

Subject	Weight	Fluid Requirement
Children	10 kg	100 ml/kg/day
Children	11–20 kg	1,000 ml plus 50 ml/kg/day for each kg over 10 kg
Children	20 kg	1,500 ml plus 20 ml/kg/day for each kg over 20 kg
Adults		2,400 ml/day (approximate)

(From Barkin RM, Rosen P,[8] with permission.)

Table 10-6. Electrolyte Requirements

Cation	Requirement	Compartment
Sodium	3 mEq/kg/day (adult: 80–100 mEq/day)	ECF
Potassium	2 mEq/kg/day (adult: 50 mEq/day)	ICF

(Barkin RM, Rosen P (eds): Emergency Pediatrics. 2nd Ed. CV Mosby, St. Louis, 1986.)

Special Situations Associated with Dehydration

Several situations may be associated with dehydration and vomiting and diarrhea that can exacerbate its consequences and make evaluation even more difficult. Children with pneumonia or meningitis may appear lethargic associated with the primary illness and superimposed fluid deficit. Other infections may also result in inadequate intake, such as viral stomatitis or pharyngitis.

Maternal deprivation, overfeeding, or mixing formula may incorrectly produce excessive vomiting or diarrhea, with resultant dehydration. This is rarely acute in onset, and a careful history should be useful in confirming the diagnosis.

Endocrine abnormalities, such as adrenogenital syndrome in the newborn, can result in rapid onset of shock requiring fluid therapy. Diabetes mellitus and diabetes insipidus can uniquely cause dehydration in the face of ongoing urinary output, the first on the basis of osmotic diuresis and the latter due to pitiutary dysfuncion. Hyperthermic syndromes can cause abnormal fluid losses as well.

Patients in Whom Clinical Diagnosis May Be Difficult

There are some situations in which it may be more difficult to make the clinical diagnoses of dehydration. These should be kept in mind and understood.

The Hypernatremic Patient

Because of relative sparing of the extracellular volume, the signs and symptoms of dehydration may be obscured in the hypernatremic patient. On the other hand, the hyponatremic patient might have exaggerated signs and symptoms.

Dehydration is classified into three categories based on serum sodium as a reflection of the osmolality in the moderately and severely dehydrated child. Similar degrees of dehydration may have a number of clinical signs and symptoms, partially reflecting the relative decrease in the ECF com-

Table 10-7. Types of Dehydration

	Isotonic	Hypotonic	Hypertonic
Serum sodium (mEq/L)	130–150	<130	>150
Physical signs			
Skin			
Color	Gray	Gray	Gray
Temperature	Cold	Cold	Cold
Turgor	Poor	Very poor	Fair
Feel	Dry	Clammy	Thick, doughy
Mucous membrane	Dry	Dry	Parched
Sunken eyeballs	+	+	+
Depressed anterior fontanel	+	+	+
Mental status	Lethargic	Coma/seizure	Irritable/seizure
Increased pulse	++	++	+
Decreased blood pressure	++	+++	+

+, ++, +++, relative prominence of finding.

(Barkin RM, Rosen P (eds): Emergency Pediatrics. 2nd Ed. CV Mosby, St. Louis, 1986.)

partment. Isotonic dehydration, the most common form, decreases extracellular space to only a moderate extent, whereas hyponatremia has a more marked effect on the extracellular compartment. By contrast, hypernatremic dehydration has a relatively balanced effect on both the extracellular and intracellular compartment (Table 10-7).

Isotonic dehydration is the most common category, with serum sodium ranging from 130 to 150 mEq/L. Correction of deficits must focus primarily on the replacement of ECF and ICF losses without an additional correction of the serum sodium (Table 10-8).

Hypotonic dehydration is present with a sodium below 130 mEq/L and often results from inappropriate administration of hypotonic fluids prior to hospitalization and less commonly from inappropriate secretion of antidiuretic hormone. In addition to the correction of deficits outlined in isotonic conditions, the sodium must be supplemented to normalize it (Fig. 10-9).

Hypertonic dehydration occurs most commonly from a deficit of free water. Correction of the deficit must occur within 48 hours to minimize rapid fluid shifts.

The Newborn Patient

Newborns are difficult to assess because of the nonspecific nature of signs and symptoms of illness as well as the relative lack of subcutaneous tissue in many of these cases. The evaluation of such children takes careful attention to the intake and output, with specific reference to the number of ounces ingested, the amount of stooling and the frequency and weight of wet diapers. As in all circumstances involving the acutely ill newborn child, an aggressive approach to evaluation and treatment is mandatory.

Table 10-8. Isotonic Dehydration: Management with Moderate Deficit

1. Initial findings
   a. Pre-illness weight: 10.0 kg (a 1-yr-old)
   b. Degree of dehydration: moderate (10%)
   c. Body weight on admission: 9.0 kg
   d. Electrolytes:

Na^+ 135 mEq/L	K^+ 5 mEq/L	Cl^- 115 mEq/L	HCO_3^- 12 mEq/L

2. Summary of fluids requirements

	H_2O (ml)	Na^+ (mEq)	K^+ (mEq)
Maintenance	1,000 (100 ml/kg)	30 (3 mEq/kg)	20 (2 mEq/kg)
Deficit (100 ml/kg)			
ECF (60%)	600	84 (140 mEq × 0.6)	
ICF (40%)	400		30 (150 mEq × 0.4 × 50% correction)
Total	1,000	84	30

3. Fluid schedule

	Fluids	
Phase	Calculation	Administered
I. 0–½ hr	20 ml/kg	200 ml D_5W.9%NS or D_5WLR over 45–60 min
II. ½–9 hr	½ deficit: 500 ml D_5W with 42 mEq NaCl and 15 mEq KCl ⅓ maintenance: 333 ml D_5W with 10 mEq NaCl and 7 mEq KCl Total: 833 ml with 52 mEq NaCl and 22 mEq KCl	833 ml (~100 ml/hr) of D_5W.45%NS with 22 mEq KCl (~27 mEq/L) (this approximation facilitates care)[a]
III. 9–25 hr	½ deficit ⅔ maintenance	1,167 ml (~75 ml/hr) of D_5W.45%NS with 28 mEq KCl (~24 mEq/L) (this approximation facilitates care)

[a] If patient is acidotic with HCO_3^- <11 mEq/L or pH <7.1 (on the basis of metabolic acidosis), one-third of sodium should be administered as $NaHCO_3$.

(Barkin RM, Rosen P (eds): Emergency Pediatrics. 2nd Ed. CV Mosby, St. Louis, 1986.)

The Chubby Infant

The excessive amount of subcutaneous tissue makes evaluation of hydration even more difficult, but other parameters can be used, including urinary output, and the appearance of the mucosal membrances, eyes, and fontanelle.

Table 10-9. Hypotonic Dehydration: Management with Moderate Deficit

In children the most common form of hyponatremia also is associated with hypovolemia (decreased total body water) caused by abnormal GI losses.

1. Initial findings
   a. Pre-illness weight: 10.0 kg (a 1-yr-old)
   b. Degree of dehydration: moderate (10%)
   c. Body weight on admission: 9.0 kg
   d. Electrolytes:

Na^+ 110 mEq/L	K^+ 5 mEq/L	Cl^- 90 mEq/L	HCO_3^- 12 mEq/L

2. Summary of fluid requirements

	H_2O (ml)	Na^+ (mEq)	K^+ (mEq)
Maintenance	1,000 (100 ml/kg)	30 3 mEq/kg)	20 (2 mEq/kg)
Deficit (100 ml/kg)			
ECF (60%)	600	84 (140 mEq × 0.6)	
ICF (40%)	400		30 (150 mEq × 0.4 × 50% correction)
Sodium		125	
Total	1,000	209	30

A. Na^+ required to correct 135 mEq/L = 135 mEq/L − 110 mEq/L (observed Na^+) = 25 mEq/L
B. Total body water (TBW) (L/kg) = 0.6 L/kg (preillness TBW) − 0.1 L/kg (water loss) = 0.5 L/kg
C. Pre-illness weight = 10 kg
   Sodium deficit = A × B × C = 25 mEq/L × 0.5 L/kg × 10 kg = 125 mEq

3. Fluid schedule

	Fluids	
Phase	Calculation	Administered
I. 0–½ hr	20 ml/kg	200 ml D_5W.9%NS or D_5WLR over 45–60 min
II. ½–9 hr	½ deficit; 500 ml D_5W with 104 mEq NaCl and 15 mEq KCl	833 (~100 ml/hr) of D_5W5.9%NS with 22 mEq KCl (~27 mEq KCl/L) (this approximation facilitates care)[a]
	⅓ maintenance: 333 ml D_5W with 10 ml NaCl and 7 mEq KCl	
	TOTAL: 833 ml with 114 mEq NaCl and 22 mEq KCl	
III. 9–25 hr	½ deficit ⅓ maintenance	1167 (~75 ml/kg) of D_5W.9%NS with 28 mEq KCl (~24 mEq KCl/L) (this approximation facilitates care)

[a] If patient is acidotic with HCO_3^- <11 mEq/L or pH <7.1 (on the basis of metabolic acidosis), ⅓ of sodium should be administered as $NaHCO_3$.

(Barkin RM, Rosen P (eds): Emergency Pediatrics. 2nd ed. CV Mosby, St. Louis, 1986.)

Inadequate History

The caregiver may be unfamiliar with the child's history, underlying medical problems, and normal appearance. Furthermore, they may be poor historians. Historical evidence, although only confirmatory, can be an essential component of evaluation of subclinical dehydration. The nature of the vomiting and diarrhea can lead to focusing on fulminant conditions, such as salmonella or shigellosis, which can lead to rapid progression of dehydration. Other adults who are involved in the child's care should be contacted for historical information if possible.

The Emaciated Infant

The absence of subcutaneous tissue makes it difficult to evaluate the emaciated child for subtle signs of dehydration, especially skin turgor quality. In many cases, such children have a chronic deficit and may develop an acute illness leading to acute deterioration.

Failure to Appreciate the Acuity of the Process

Perhaps the most common mistake is the failure to recognize the severity of the illness and the relative risk of the child to progress rapidly and irreversibly. The first problem is inherent with a lack of familiarity with the subtleties of taking a history and performing a physical examination on the infant compounded by an inherent anxiety. There is also an underlying assumption made by many that children are healthy and need to prove that they are ill. This is a particularly dangerous position, especially when placed in the context of a child's unique ability to compensate for intravascular depletion. Children can maintain a compensated state for an extensive period, responding with marked vasoconstriction and tachycardia, often detected only with measurement of orthostatic vital signs. Acidosis may be an early indication of the severity of the process. Once these compensatory mechanisms are no longer effective, however, uncompensated shock with rapid progression to irreversible shock occurs and management becomes increasingly difficult. The point is obviously to recognize the problem early and initiate treatment then.

TREATMENT

Failure to Treat Shock

The progression from dehydration to fulminant shock may be precipitous. Delay in initiating treatment to the dehydrated infant is unacceptable. There are several reasons why shock might go untreated.

Lack of Recognition

The diagnosis of shock secondary to dehydration is a clinical diagnosis. Lack of end-organ perfusion and respective clinical signs is the key to diagnosis.

Failure to Monitor Inadequate Therapy

Although therapy is calculated, it must be monitored. Miscalculations or ongoing losses can easily result in lack of adequate therapy.

Wrong Fluid or Fluid Rate

Initial treatment of the child with marked dehydration and shock is volume expansion, irrespective of the reason for the hypovolemic state. The initial focus must be on restoration of the vascular volume. All patients with compromised volume status, particularly those with abnormal vital signs, must be given an immediate infusion of fluids. Laboratory analysis should be initiated early, but initial fluid resuscitation must be started upon the patient's arrival and not await these confirmatory data. It may be adjusted later as more clinical and laboratory parameters become available.

Fluid therapy should include a rapid infusion of 5 percent dextrose and lactated Ringer's (D_5W LR) at a rate of 20 ml/kg (adult 1 to 2 L) over 10 to 20 minutes in the moderate or severely dehydrated child. If the child is in shock, this should be pushed as fast as possible. If there is a poor therapeutic response to the initial infusion, an additional fluid bolus of 10 ml/kg may be given over 20 to 30 minutes, assuming normal renal and cardiac function. This second infusion may be repeated for a total infusion of 40 ml/kg during the stabilization period. Laboratory values are not necessary before therapy can be started. The use of these guidelines in the child without known renal or cariac disease is a relatively safe approach to management and is certainly the desirable option when confronted with a child with intravascular depletion. The alternative is to allow shock to progress beyond salvage.

Glucose should be omitted if diabetic ketoacidosis is the underlying etiology. It is not required if the patient is over 8 years of age and has a normal glucose and nutritional status or is in acute hypovolemic shock on the basis of blood loss.

If a poor response is noted in the severely ill dehydrated child following two fluid pushes (i.e., no urine output, continued abnormal vital signs, poor perfusion, ongoing losses, or suspected cardiac or renal disease), central venous pressure or pulmonary capillary wedge pressure may be measured. Urinary output should be maximized.

Failure to Address Associated Abnormalities

Metabolic and acid-base abnormalities are commonly encountered and need to be considered in the management of the dehydrated child. However, paramount to identifying such problems must be the recognition that the

initial stabilization will be the same as described above—rapid infusion of isotonic crystalloid.

Acidosis

Dehydrated children are often acidotic, and the degree of acidosis is worthy of monitoring on an ongoing basis as one parameter in measuring the efficacy of the therapeutic plan. Furthermore, acidosis is often one of the earliest objective parameters in assessing a compensated shocklike state. Rarely is specific therapy required. Fluid resuscitation and improvement of perfusion are usually adequate without bicarbonate therapy. Serum bicarbonate should be monitored, while ABGs are indicated only if there is a marked delay in obtaining serum values or there is some question of hypoxia or hypercapnia.

Hypocalcemia

In the severely dehydrated child in shock, hypocalcemia may develop during prolonged resuscitation and support. Because of the recent evidence regarding the lack of efficacy of calcium during cardiopulmonary resuscitation, the role of calcium infusion must be weighted carefully and not initiated until there is documented evidence of a deficit.

Hypoglycemia

Children with vomiting and diarrhea as the underlying pathology for dehydration may have impaired nutritional stores and may benefit from glucose infusion early. Many recommend that the initial fluids include dextrose, while others suggest that dextrose be added to the crystalloid infusion once the initial stabilization period has been passed. Either is acceptable, but certainly it is imperative to monitor the glucose metabolism. Diabetic ketoacidosis and children over 8 years of age do not require dextrose administration.

Hyponatremia or Hypernatremia

Hyponatremia results from either decreased intake of sodium with respect to the amount of water ingested, excessive sodium loss, or excessive water intake. By contrast, hypernatremia usually stems from excessive intake or decreased body water in the face of a normal total sodium. Identification of these states is essential beyond the initial stabilization period and correction of any deficits over the subsequent 24-hour period.

Potassium

Only 1.5 to 2.0 percent of total body potassium is in the ECF and is measured by serum values. During metabolic acidosis, renal secretion of potassium is decreased. For every 0.1-decrease in pH, serum potassium increases by about 0.5 mEq/L. Abnormalities in potassium can have an impact on cardiac conduction as well as on the GI tract.

Complications of Therapy

Overhydration

With appropriate attention to the volume of fluid infused, children should not be significantly overhydrated. Volume-limiting devices such as Buretrol or Soluset should be inserted in an infusion line to minimize inadvertent excess fluid infusion. The real danger comes with the clinician who fails to pay attention to both the volume and the type of fluid infused. If care if taken, overhydration should not be a problem. In most cases, overhydration is safer than inadequate fluid resuscitation in the dehydrated child.

Sodium

Correction of sodium deficits must be done with care. Hyponatremia is usually corrected over 24 hours, using normal saline as the solution. It is essential to correct hypernatremia over 48 hours using a variety of solutions based on both personal preference and scientific data supporting the infusion of fluids ranging from 5 percent dextrose in water (D_5W) 0.2 percent to 0.9 percent normal saline (0.9% NS).

Acidosis

A conservative active correction of any noted acidosis is essential to prevent creating alkalosis. Many of the arguments that support constraint are parallel to those leading to a marked restriction in the use of sodium bicarbonate in cardiopulmonary resuscitation and diabetic ketoacidosis.

Potassium

Because of the ever-present potential for acute renal railure and hyperkalemia, the addition of potassium to intravenous fluids should usually be withheld until the patient urinates or if the history supports good urinary output.

Difficulty in Obtaining IV Access

Venous access in the dehydrated child may present substantial logistical problems to the clinician. In the severely ill child, peripheral and central sites may be considered. A more recent alternative is intraosseous infusion. Peripheral sites are commonly used, particular attention being given to the traditional sites as well as the external jugular vein, which is often overlooked.

Central access is rapidly achieved by those experienced with the techniques. The relative risks must be balanced by necessity to gain access and alternative routes. Many prefer to use the internal jugular or subclavian veins: however, this route has substantial risk in terms of pneumothorax, interferes with airway and ventilation intervention, and has a relatively

high resistance to rapid fluid infusion. The femoral vein is the preferred route in many settings.

Venous cutdown is a good option. The saphenous cutdown at the ankle can be easily monitored and performed quickly. Intraosseous infusion offers a mechanism with which to infuse fluids while a more definitive route is being established. The technique commonly involving insertion of a bone marrow or spinal needle into the tibia and has been demonstrated to be very effective in fluid resuscitation on a transient basis pending direct venous access. However, the long-term and short-term safety has not been definitely established and further research is required.[9]

Oral Routes

The use of oral rehydration techniques has been popularized of late. In selected settings there has been repeated success with substituting oral rehydration for the traditional intravenous route. Using solutions with 75 to 90 mEq/L of sodium and 2.5 percent dextrose, children with moderate, and even severe dehydration may be resuscitated using the oral route. However, such a technique requires the childs cooperation and may be difficult if vomiting is ongoing. Intravenous therapy has an identical efficacy.[10–12]

Oral hydration is usually totally adequate in the child with mild dehydration who can tolerate oral intake. If oral fluids are not initially tolerated, an initial parenteral fluid bolus of 20 ml/kg D_5W 0.9 percent NS or D_5-lactated Ringer's (LR) over 30 to 40 minutes may facilitate intake. Patients require monitoring of intake, output, and weight. Laboratory tests are rarely required. Clear liquids (Table 10-10) should be pushed as tolerated by the child, although slowly in the child who is vomiting.

Ideal fluids for the infant include Pedialyte or Lytren. Pedialyte RS is designed for initial hydration of children with fluid deficit. Other clear liq-

Table 10-10. Commonly Acceptable Clear Liquids

Solution	Na^+ (mEq/L)	K^+ (mEq/L)	Cl^- (mEq/L)	HCO_3^-/ Citrate (mEq/L)	Glucose (mg/dl)	Osmolality (mosm/kg H_2O)
Lytren	25	25	30	18	945	290
Pedialyte	45	20	35	30	2,500	250
Pedialyte RS	75	20	65	30	2,500	305
Infalyte	50	20	40	30	2,000	111
Gatorade	28	2	—	—	2,105	322
7-Up	4	0.2	—	—	3,095	525
Coca-Cola	3	0.1	—	13.4	1,495	600
Pepsi-Cola	2	0.9	—	7.3	4,900	672
Jello-O water (½ strength—strawberry)	10	0.1	—	—	118[a]	253

[a] Long-chain oligosaccharide subject to hydrolysis.

(Barkin RM, Rosen P (eds): Emergency Pediatrics, 2nd ed. CV Mosby, St. Louis, 1986.)

Table 10-11. Underlying Causes of Gastroenteritis

Congenital
Intestinal obstruction
Infection
Acute gastroenteritis
Hepatitis
Meningitis
Trauma
Concussion and head trauma
Intramural hematoma
Intoxication
Iron
Acid or alkali
Gastrointestinal irritants
Vascular
Migraine
Hypertension
Superior mesenteric artery syndrome
Endocrine/Metabolic
Acidosis
Diabetic ketoacidosis
Uremia
Inborn errors of metabolism
Psychiatric
Attention getting
Hysteria
Neoplasm
Chalasia
Improper feeding technique
Hyperthermia

uids, although less desirable because of their variability in electrolyte composition, are acceptable alternatives and work well in older children with only mild dehydration. Contraindicated foods include rice water, tea, and boiled milk; many fruit juices are hyperosmolar and may draw water into the intestinal lumen, worsening diarrhea.

Once ongoing losses are decreased, the diet may be advanced, usually within 24 hours of a restricted clear liquid diet. Milk may then be initiated slowly, many authorities suggesting that a lactose-free product is better tolerated by the child with prolonged (more than 7 days) gastroenteritis.

Failure to Address the Underlying Causes of Dehydration

Concurrent with therapy of the dehydrated child, the clinician must focus on the underlying disease process leading to the fluid deficit. Common entities that must be considered beyond acute gastroenteritis are listed in Table 10-11.

SUMMARY

The management of the dehydration in a child, usually secondary to vomiting and diarrhea, requires intervention on an emergency basis to forestall progression of disease and ultimate decompensation. This initial therapy consists of isotonic fluids and must not await laboratory confirmation. With proper initial management and systematic ongoing correction of deficits, both morbidity and mortality should be minimal and children returned rapidly to their normal state of health.

REFERENCES

1. Barkin RM: Acute infectious diarrheal disease in children. J Emerg Med 9, 1985
2. Roy CC, Silverman A: Pediatric Clinical Gastroenterology. 3rd ed. CV Mosby, St Louis, 1982
3. Kapikian AZ, Whakin H, Whatt RG, et al: Human reovirus-like agent as the major pathogen associated with "winter" gastroenteritis in hospitalized infants and young children. N Engl J Med 294:965, 1976
4. DeWitt, TG, Humphrey KF, McCarthy P: Clinical Predictors of Acute Bacterial Diarrhea in Young Children. Pediatrics 76:551, 1985
5. Finberg L, Kravath RE, Fleischman AR: Water and Electrolytes in Pediatrics. WB Saunders, Philadelphia, 1982
6. Barkin R: The dehydrated child: A management approach. J Emerg Med 1:399, 1984
7. Winters RW: Principles of Pediatric Fluid Therapy. 2nd Ed. Little, Brown, Boston, 1982
8. Barkin RM, Rosen P (eds): Emergency Pediatrics. 2nd Ed. CV Mosby, St. Louis, 1986
9. Rosetti VA, Thompson BM, Miller J, et al: Intraosseous infusion: An alternative route in pediatric intravascular access. Ann Emerg Med 14:885, 1985
10. American Academy of Pediatrics Committee on Nutrition: Use of oral fluid therapy and post treatment feeding following enteritis in children in a developed country. Pediatrics 75:358, 1985
11. Listernick R, Zieseri E, Davis AT: Outpatient oral rehydration in the United States. Am J Dis Child 140:211, 1986
12. Martin DL, Goldhagen JL: Oral rehydration therapy. In Barkin RM (ed): The Emergently Ill Child. Aspen, Rockford, MD, 1987

11 Problems in the Management of Seizures

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A seizure is an abrupt synchronous depolarization of many neurons that is propagated to affect end organs. The end-organ response may involve sensory and motor disturbance, alterations in consciousness, and behavioral disturbances in variable combinations. The polymorphic expressions of a seizure are occasionally subtle.

RECOGNITION OF SEIZURE

Most patients who have a seizure complete the sensory or motor activity or behavioral changes prior to transport to the emergency department. We become historical investigators indirectly sharing another's experience. Most parents, guardians, or caretakers who witness a seizure share their experience with unreserved willingness. However, we must appreciate that a good portion of parents believe that the child's actions reflected a terminal or near-terminal event.[1] An initial unguided sympathetic interview may serve as a catharsis but is uncommonly diagnostic. In their panic, parents typically distort temporal relationships or concentrate on specific noncontributory details. Historians may be more competent witnesses if the potentially diverse features of a seizure are independently explored. The emergency physician may be able to extract and synthesize cooperatively all the component parts of their testimony. This chapter anticipates the key elements of clinical expression. It is promoted as a partial solution to the problem of seizure recognition.

Aura

An aura consists of motor, sensory, autonomic, or psychic symptoms. It generally constitutes the initial manifestation of a seizure and occurs prior to an impairment of consciousness.[2] Auras in children are typically unpleasant. They include headache, discomfort in the chest, pins-and-needles feeling throughout the body, or complaint of a metallic taste in the mouth. Vague sensations such as a feeling of fright, of impending doom, or of detachment from the surroundings or feeling vacant are common. Perceptual distortions in olfactory, auditory, or visual sense are rare. When distinct hallucinations do occur, they are intensely unpleasant and often alarming.[3] Auras, even when present, may not be described if children are developmentally nonverbal or if the seizures occur only during sleep.[2]

Facial Expression

Involuntary facial expressions that can be quantitated and categorized according to outlined criteria may be witnessed at the onset of seizure. The largest number of patients have a cessation of facial muscle movement creating a blank or neutral expression. Of those who display some emotional response, sadness is followed in frequency by expressions of fear, surprise, happiness, and disgust.[4]

Eye Movement

A frequently encountered complaint is that the eyes turn upward and disappear behind the upper eyelids, leaving only a rim of sclera visible between the half-closed eyelids. Sustained eye opening and fixed gaze is most common with seizures in infants under 1 month of age.[5] Quiet staring at any age is generally associated with a change in facial expression. Repeated blinking may be the sole observable feature of clonic motor activity.

Unsustained wandering or seemingly voluntary eye movements are uncommon. More often there is involuntary, sustained, unnatural, tonic, conjugate deviation, which may be associated with nystagmus. The nystagmus may last for 60 to 90 seconds and may be accompanied by blinking.[6] It is more common for the eye movements to be associated with ipsilateral, forced, tonic head movement—versive movement. Involuntary movements of the eyes and head results in sustained unnatural positioning that cannot be interrupted.

Unresponsiveness

Variable degrees of clouding of consciousness occur with seizures. Patients may have either transient or prolonged loss of contact with surroundings. Disorientation or confusional states may occur without abnormal motor activity or may be associated with immobility. Patient unresponsiveness may be due to apraxia, dysarthria, or preoccupational awareness of other

seizure manifestations. Desire for uninterrupted sleep lasting for minutes to hours may also be seen.

Abnormal Speech and Vocalization

An inarticulate or gutteral sound created by forced expiration through a closed glottis may attract the attention of a caretaker. Equally alarming to an observer is the responsive patient whose verbal activity may suddenly be cut off. Those whose seizure is associated with mutism cannot be engaged in conversation, nor are they able to answer questions or repeat phrases. Mutism ends as abruptly as it was initiated.[7]

Motor Activity

Seizures without abnormal motor activity (nonconvulsive) are far less common than convulsive seizures where muscular contractions are prominent. In convulsive seizures, movement is more often rhythmic than asynchronous. With subsequent seizures, the movements are repetitive and stereotyped and often affect the same muscle groups. The contractions can occur continuously or in series and may last seconds to days.[7]

The extent of motor activity may range from isolated regions such as eyelids, mouth, and hands, without disturbing posture, to involvement of the entire trunk, face, neck, and extremities. The movement may be confined to a single area of the body in which the movement starts. Motor activity may only extend to adjacent body parts, such as conjugate eye movement, with tonic or clonic contractions of the facial muscles. The versive movement may have accompanying clonic movement or tonic posturing of the upper limbs, creating a fencing position with elbow flexion followed by abduction of the shoulder to 90 degrees with the patient looking at a fist.[8]

With some seizures, there may be an extensive progressive march in a regular way, stereotypically in every new attack from the initial focus to all other parts of the body.[3] Only newborns exhibit motor activity of one extremity that migrates randomly to other extremities.[9]

In older children, the motor activity may be generalized from the start. The motor activity may be simple, expressed as either tonic stiffening or clonic with symmetric jerking. Motor activity may occasionally be integrated into such complex activities as repetitive walking in small circles or picking up and turning objects.[10]

Loss of Body Tone

Seizures may be manifest as selective or generalized loss of body tone. Such loss of body tone is abrupt in onset. If sitting, the infant may lose only head control and have nodding spasms, or loss of motor tone of the trunk may lead to forward flexion spasms. If the child is walking the abrupt loss of motor tone may make the child fall suddenly forward in a position mimick-

ing a salaam greeting. Those patients who lose body tone may either remain on the ground motionless and unconscious for 1 to 2 minutes, or they may abruptly arise with perhaps a brief phase of confusion.[11] As these attacks are rapid and the patient has no warning, sitting or standing children will fall with no opportunity to protect themselves with their hands. Such falls often result in repeated facial or head injury. As a postictal phenomenon, paralysis may result.[12]

Behavioral Changes

It may be difficult to establish behavioral changes as a seizure, especially if the abnormal behavioral is the only or major manifestation of a seizure.[13] Children who have no mental disturbances interictally without provocation may suddenly exhibit mood alterations or abnormal behavior. The most common mood alterations observed as seizures are fear, followed in frequency by feelings of sadness and happiness. Feelings of anger and disgust are rare.[4] Behavioral disturbances that may occur as manifestation of seizures include poor memory, restlessness, agitation, irritability, uncontrolled emotional outburst with euphoria or laughing, and crying. Abnormal verbiage that includes inappropriate flow of words, delusions, or paranoid ideation may be expressed. These sudden changes in mood and behavior may end abruptly but may also gradually abate.[3]

Involuntary Actions

Involuntary actions during seizure activity are called automatisms. This repetitive and stereotypical behavior is poorly coordinated or fragmented. The types of automatic behavior that occur vary from patient to patient but tend to be similar from seizure to seizure in the same patient.[2] These disjointed behaviors include sitting, standing up, fumbling with a belt, rocking, dropping objectives, placing hands over eyes, snapping fingers, scratching, grimacing, yawning, chewing, lip smacking, and tongue movements. Neonatal automatisms include paddling, swimming, or rowing movements of the extremities. Most automatisms occur de novo and do not appear to be reactions to environmental stimuli.[2] Occasionally some ictal automatisms can persist as a continuation of the activity the patient was engaged in before the seizure.[14] Sometimes during automatisms it is possible to elicit partial responsivity to environmental stimuli.[8] However, in most cases, the patient is totally unresponsive during automatisms and is generally amnesic for the events. Automatisms may accompany versive seizures.[8] Automatisms may follow the loss of body tone.[11]

Respiratory Distress

Breathing difficulties accompany various types of seizures. Circumoral cyanosis is common during the tonic phase of generalized seizures, when respiration may be suspended. This apneic phase is typically brief and

rarely prolonged. Respirations are jerky and sonorous during the clonic phase.

Cyanosis and altered consciousness may accompany pure apneic events that are the dominant manifestation of a seizure in the newborn. Such apneic seizures may last 1 to 2 minutes.[15] Isolated apnea is unusual as the only manifestation of seizure beyond the neonatal period.[16]

Laryngospasm may be a solitary manifestation of seizure. The patient may exhibit a short cough followed by intensive inspiratory effort, with severe air hunger, generally without stridor. The attack may last 2 to 3 minutes and may be associated with cyanosis.[17]

Dysrhythmia

Patients who have primary rhythm disturbances may present with nonconvulsive or convulsive seizures. The most important dysrhythmia to recognize occurs with the congenital long QT syndrome. In children with congenital long QT syndrome, ventricular tachycardia, and fibrillation have been documented before and during frank seizure activity.[18] Such attacks are often precipitated by acute arousal reactions engendered by loud noise, emotion such as fright, or acute vigorous physical exertion.[19] Unexplained syncope, deafness, or a history of sudden death in the family or seizures in other family members should arouse suspicion of hereditary QT prolongation.[20]

Loss of Control of Body Fluids

During the clonic phase of seizures, the jaws move rhythmically; apparent chewing motion of saliva produces foaming oral secretions. If the mucous membranes have been bitten, the foam may be bloody. The foam may be pink or blood tinged if the patient has compounding pulmonary edema.[21] Young infants, particularly those under 1 month of age, may drool as a manifestation of seizures.[22]

Unexplained urinary or fecal incontinence may be clues to seizure activity.

INFLUENCE OF AGE ON DIAGNOSTIC ACCURACY

The age of the patient may influence the clinician's ability to diagnosis a seizure. The expression of neurologic dysfunction in younger and smaller infants may be nonspecific and ambiguous. The ambiguity is intensified in preterm and term infants under 1 month of age. Inconsistent neurologic response to insult in neonates is partially explained by morphologic and biochemical immaturity of the central nervous system (CNS). The very young have incomplete development of intracortical connections, delayed myelination of the corpus callosum and corticospinal tracks, and early development of inhibitory networks than of excitatory circuits.[23] Restlessness,

tremor, jitteriness, decreased motor activity, decreased motor tone, apathetic response to the environment, and high-pitched or weak cry are the principal manifestations of neonatal brain disturbance. These manifestations of underlying brain dysfunction may occur without pathologic electroencephalographic (EEG) changes. Altered suck, refusal to feed, apnea, bradycardia, tachycardia, cyanosis, peripheral vasoconstriction, and tachypnea may also be seen with neonatal brain disturbance unassociated with EEG changes.[24]

Apnea with or without bradycardia, vasomotor instability, and abrupt change in respiratory patterns may occur as ictal phenomena.[25–28] Therefore, without a concurrent EEG tracing, it may be difficult during the neonatal period to differentiate a true ictal pattern from other nonictal phenomena resembling seizures.[22,29]

Infants under 1 year of age with seizures may occasionally be difficult to diagnose accurately. These children readily display reflex activity resembling seizures with little provocation. Once older then 1 year of age, children with seizures are generally recognizable, unless the seizure lacks motor activity or altered consciousness.

DIFFERENTIAL DIAGNOSIS

Historians occasionally verbalize sequential events that may be unjustifiably attributed to seizures.[30] Diseases or syndromes that may lead to an erroneous diagnosis of seizure disorders are varied (Table 11-1). The following comments are offered about selected conditions that may arouse suspicion of epilepsy.

Reflex Anoxic Syncope

Juvenile breathholding spells and adolescent fainting attacks are clinical expressions of vasomotor instability in progressively advancing ages. For example, in breathholding the toddler strikes the head in a minor fall

Table 11-1. Conditions that Simulate Childhood Seizures

Infancy	Early childhood	Adolescence
Tremor	Breathholding spells	Vasomotor syncope (faint)
Jitteriness	Venipuncture fits	Orthostatic hypotension
Dysrhythmia	Swallowing syncope	Dystonic drug reaction
Apnea	Postmicturitional syncope	Rage attacks
Sandifer's syndrome	Cough syncope	Pseudoseizures
Micturitional shivering	Paroxysmal choreoathetosis	Hysteria
Decerebration fits	Complicated migraine	Hyperventilation
	Tics	
	Masturbation	
	Night temors	
	Narcolepsy	

but does not cry. The parent finds the child with eyes fixed upward, witnesses truncal stiffening, then brief jerking movements of the extremities associated with acrocyanosis, followed by limpness. The child may then awaken briefly and then returns to sleep. These events occur most commonly in the 6- to 8-month-old group and are rare beyond 6 years.[31] Such attacks are precipitated by external factors, such as anger, discipline, or frustration. Painful stimulation such as cuts, minor blows to the head, or spanking are precipitating events in children under age 2. Venipuncture can precipitate the event in an older child.[32]

Older children and adolescents may become syncopal from emotional stimuli or experience orthostatic difficulties. Those who are repeatedly syncopal may complain of nausea, inability to suppress yawning, dyesthesias of the face and extremities, heightened awareness of cardiac activity, and a generalized feeling of weakness prior to loss of consciousness.[33] In all age groups, the genesis of the syncope lies in the afferent–efferent loop between the brain stem reticular formation and the cardiovascular system. Increased vagal tone leads to profound bradycardia or brief asystole.[34] Carotid and vestibulobasilar insufficiency result in loss of consciousness. If the ischemia is prolonged, electrical cortical inhibition of the brain stem is suppressed allowing for tonic spasms.[32] Vagotonic procedures such as ocular compression, carotid body massage, or facial emersion in ice water may reproduce the clinical expression of reflex anoxic seizure in these patients. Those patients who have postmicturitional syncope or cough syncope can be confidently diagnosed when there is an accurate description of the precipitating events.[35]

Decerebration

Central transtentorial herniation or acute anoxia can lead to intermittent episodes of decerebrate posturing that can be misinterpreted as seizure activity.[36] Generalized tonic extension dominates. The legs are always rigidly extended, but the arm position is more variable. In the fully developed extensor spasm, there is internal rotation of the shoulder as well as elbow extension and hyperpronation. The wrist and finger positions are those of a clinched fists. Differentiation of extensor posturing from seizures may be particularly difficult in the first month of life.[37]

Dysrhythmia

Non-life-threatening bradycardic and tachycardic dysrhythmias in childhood may be associated with ineffective cardiac output and decreased cerebral blood flow.[33] As these conduction disturbances are recurrent, patients present with repeated attacks of lightheadedness or frank syncope. In some patients, brief, clonic movements occur as consciousness is lost, and the episode may be confused with a true seizure. Simultaneous monitoring

of the EEG and electrocardiogram (ECG) during an episode reveals normal electrical discharges.[38]

Dyskinesia

Involuntary movements in children occur principally with structural abnormalities of the cerebellum or impaired dopaminergic, cholinergic, and GABAminergic transmitters within the basal ganglia. With few exceptions, both congenital and acquired diseases associated with movement disorders are insidious in onset and associated with either coordination difficulties, abnormal speech, emotional lability, or mental deterioration. Only several states have clinical manifestations commonly associated with, and mistaken for, seizures. They include dystonic drug reactions, paroxysmal choreoathetosis, and dyskinesia during the acute phase of bacterial meningitis.

Phenothiazines, even in therapeutic doses, can lead to involuntary extension and twisting of the neck, shoulders, and arms, often with oculogyric crisis. This activity resembles a tonic seizure, but the patient remains alert throughout the episode.[39] Diphenylhydantoin at therapeutic levels has been reported to induce movement disorders, including choreoathetosis, dystonia, and asterixis.[40] Similarly, carbamazepine at therapeutic levels may produce multiple motor tics[41] or, at toxic levels, produces facial dyskinesias and choreoathetosis.[42]

Paroxysmal choreoathetosis is an uncommon disorder characterized by involuntary movements triggered by anxiety or initiation of movement. Episodes are paroxysmal, brief, and usually unilateral. They are characterized by arm abduction, elbow flexion, forearm pronation, wrist dorsiflexion, and digital flexion. Auras, dysarthria, or facial grimacing may accompany attacks.[43]

Focal athetoid movement of a single extremity or generalized choreiform movements of all extremities have been described in children during the course of treatment for meningitis. Such movement may be of abrupt onset and severe enough to resemble seizure activity.[44]

Tics, tremor, and jitteriness are occasionally confused with motor activities that accompany seizures. In children, tics involve primarily the head, neck, and shoulders. They consist of complex movements such as facial grimacing, eye blinking or rolling, head nodding, and shrugging of the shoulders. In contrast to seizures, these attacks are all precipitated by external factors. Tremors usually involve multiple muscle groups and have a rhythmic to-and-fro movement, in contrast to seizures, in which movements are more abrupt, with a distinct interval between each movement.[2] Jitteriness is characteristically a disorder only of the newborn period. In contrast to seizures, jitteriness is exquisitely stimulus sensitive; the alternating movements are rhythmic, not jerking.[22] Infants with jitteriness tend to have enhanced deep tendon reflexes. In addition the jitteriness may be diminished by passive limb flexion.

Complicated Migraine

Classic migraine headaches are usually diagnosed in children when transient autonomic, visual, motor, or sensory phenomena accompany the incapacitating headache. However, when migraines are atypical or complicated, they may resemble seizures.

In basilar artery migraine, the attacks are sudden. Visual disturbance, vertigo, ataxia, loss of postural tone, dysarthria, and tinnitus may be seen in varying combinations. Impairment of consciousness as part of the syndrome is variable. The headache may occur only after other symptoms resolve. Diagnosis may not be apparent until there are repreated stereotypical attacks. As with hemiplegic migraine, a strong family history is often encountered.[45]

In the hemiplegic variant, significant motor and sensory deficits of a unilateral nature accompany the headache. The paralysis, usually of several hours' duration, may precede, accompany, or follow the contralateral head pain.[46] Diagnosis may be apparent only after repeated episodes. Differentiation from a primary seizure disorder may be compounded by the occasional occurrence of tonic-clonic motor activity with an attack.[47]

In confusional migraine, children may experience aberration of time perception, distortion of body image, or impaired visual analysis of the environment while remaining lucid and oriented to place and person.[48] The confusional state may last as long as a day.[49] Behavioral alterations during episodes may resemble nonconvulsive seizures. They may only be recognizable as a complicated juvenile migraine variant when associated with scintillating scotoma or incapacitating headache.[48]

Sleep Disturbance

Periodic episodes of night terrors bear resemblance to seizures. Several hours after falling asleep, parents are alerted to difficulties as the child screams inconsolably. Those affected seem to be in a state of panic. They may tremble and stiffen out. Children exhibit semipurposeful behavior, such as walking throughout the attack, which may last several minutes. Children cannot be aroused. The terror abates, and the child falls back to sleep and is amnesic for the event.[50]

Rage Attacks

Rage attacks or the episodic discontrol syndrome may resemble nonconvulsive seizures. These attacks usually begin during adolescence. Recurrent rage attacks occur suddenly, with minimal provocation. Patients typically exhibit uncontrollable, seemingly psychotic, behavior. After the attack, they may have amnesia and fatigue, and occasionally remorse may occur.

Physical violence such as scratching, fighting, biting, kicking, or spitting, is typical.[51] This violent behavior is in contrast to true seizures, in which directed physical violence is unusual.[52]

Pseudoseizure and Hysteria

Children manifesting pseudoseizures willfully produce or feign episodic stereotypical behavior, whereas those exhibiting hysteria display the behavior involuntarily. Pseudoseizure and hysterical seizure patients mimic true seizures by their abrupt onset. However, bizarre clinical features cannot be explained on sound neurophysiologic grounds. No abnormal electrical discharges occur before, during, or after the event. Pseudoseizures may occur in children of normal intelligence, with no evidence of hysterical personality, conversion reaction, or other apparent psychiatric disorder.[53] Patients with hysteria often have associated intellectual and cognitive impairment.[54] Patients with either condition may have additional complaints of paralysis, snycope, vertigo, paresthesias, blindness, or diplopia.[55]

SIGNIFICANCE OF HEAD TRAUMA

All children, especially the young, are continuously vulnerable to head trauma during the course of their usual daily activities. The history of a recent injury may be difficult to ascertain with certainty. Nevertheless, the physician who confronts a child with seizure must interpret any information gathered from guardians, parents, playmates, and prehospital care takers about head trauma. Impact seizures closely following a head injury may present a diagnostic challenge.

An estimated 1 to 7 percent of pediatric patients under 16 years of age sustain a seizure within 1 week of a head injury.[56] Sixty to 80 percent of those who seize will have the convulsion within 24 hours of the trauma.[57] Of these, one-third seize within 1 hour and one-half within 3 hours of the head injury.[58] Of those who are provoked to seize following head trauma, focal motor seizures are seen in 75 percent of cases, and 10 percent with generalized motor seizures will present with status epilepticus. The latter is most common in children under age 5.[57]

Current practice advocates a careful neurologic evaluation of the pediatric patient with suspected impact seizure and a strong consideration for cranial computed tomography (CT). Unfortunately, there are no absolute criteria for pediatric CT scanning following isolated head injury.[59,60] If one commits all post-traumatic seizure patients to immediate CT, as advocated by some,[61] the rare space-occupying lesion will not be overlooked. However, such a decision is inflexibly rendered in isolation of the clinical environment. The efficacy of screening for mass lesions would increase in specific situations (Table 11-2). Most of the circumstances delineated are associated

Table 11-2. Indications for Cranial CT in Children who Seize After Head Injury

Multiple trauma
Unconsciousness for greater than 10 minutes
Decreasing level of consciousness
Focal neurologic deficit
Suspected, proven skull fracture
Suspected intracranial hypertension
Suspected child abuse
Previous craniotomy with shunt in place
Anticonvulsants needed to abort seizure
Age: less then 1 year

with increased likelihood of space-occupying intracerebral pathology.[57] In low-risk situations, such as a brief impact seizure following trivial head injury in a child above 1 year of age, a neurologic examination can dictate the extent of diagnostic intervention.

SEIZURE ASSOCIATED WITH FEVER

It is crucial to distinguish those previously healthy children who seize with fever who have benign intercurrent illnesses from those whose fever and seizure occur as a result of CNS infection or other serious infection. Most authorities are in agreement as to the characteristics that separate the two groups.

Ninety-five percent of children who seize unrelated to intracranial or serious infection are between 6 months to 5 years of age.[62] They have no preexisting neurologic or developmental abnormalities and are without history of epilepsy. Their convulsion occurs early in the course of the illness. The seizure may be the first sign that alerts guardians to the existence of the fever. In general, fever will be present for less than 24 hours.[63] The seizure is brief and self-limited.[12] The seizure is either single or, when multiple, occurs in a series having a total duration of less than 30 minutes.[64] In 85 percent of cases, the seizure is generalized, clonic, or tonic-clonic without focal or lateralizing features.[65] The seizure is followed by a brief postictal somnolence phase, during which no neurologic abnormality can be appreciated. An extracranial focus of infection may be apparent on examination or the source for fever may not be apparent in spite of detailed history and optimal physical examination.[66] Further seizures are unlikely within 24 hours.[67]

Several historical and clinical features of a seizure coincident with fever increase the probability of focal infection or serious illness. The first historical aberrancy is a child younger than 6 months or older than 5 years.[68] Children who have seizures 24 hours after the onset of fever are at

Table 11-3. Characteristics of Seizure with Fever that Suggests Focal Infection or Serious Illness

History
Age: less than 6 months, more than 5 years
occurrence late in illness
Visit to physician within 48 hours
Prolonged seizure, requiring anticonvulsants
Focal or akinetic seizure
Repeated seizures during one febrile illness
Prodromal CNS derangement
History of pica
Presence of explosive diarrhea
Physical Examination
Ill appearance
Abnormal physical examination
Abnormal findings on neurologic examination

increased risk of having a significant illness.[69] The history of a physician visit within 48 hours prior to seizure with fever or the occurrence of the seizure on arrival at the emergency department identifies children at risk of meningitis.[70]

Those who require anticonvulsant therapy to abort the seizure either in the prehospital setting or within the emergency department have had a prolonged seizure that increases the risk of serious infection. A focal seizure with fever increases the likelihood of intracranial infection.[71] Multiple seizures during the course of a single febrile illness should raise suspicion for CNS infection, metabolic derangement, or electrolyte disorder.[12] A history of preceding neurologic symptoms, such as headache, head tilt, morning vomiting, visual impairment, ataxia, altered mentation, or a history of pica prior to the seizure, raises a possibility of intracranial hypertension or structural lesion.[72] Those patients who seize concurrent with frequent passage of loose stools, possibly mixed with blood or mucus, are likely to have an enteric pathogen, with potential complications of bacteremia, dehydration, electrolyte imbalance, or acid-base abnormality.[73]

The risk of serious infection in febrile seizure patients is greatest in those who appear ill. The young child's quality of vocalization, eye function, motor behavior, and response to the environment are beneficial to the interpretation of the degree of illness.[74] Serious illness is predicted in infants who are inconsolable when held or fed or who are unresponsive to the examiner or parents.[75] Although subjective, identification of serious illness based on ill appearance and toxicity is facilitated as children advance in age.[76] A child who has seized and who exhibits any constellation of abnormal physi-

cal findings that includes cranial bruits, nuchal rigidity, full fontanel, tachypnea, rales, grunting respirations, gingival lead lines, petechiae, poor skin perfusion, soft tissue erythema, and induration or cyanosis is likely to have a serious illness.[70,77,78] Abnormal neurologic findings that predict serious illness include increased or decreased motor tone, nystagmus, positive doll's eye sign, inability to fix and follow, lethargy, inadequate response to verbal command, no response to painful stimuli, or focal findings such as hemiparesis.[12,70]

SEIZURE UNWITNESSED

A seizure is but one of many events that can occur with such rapidity in a child that it is possible for an observant adult not to witness the entire episode. As a partial observer, the parent may not be able to reconstruct the action with enough accuracy to make the history valid. However, unexplained facial trauma, incontinence, somnolence, headache, confusion, or confabulation should arouse suspicion of a seizure. It is appropriate for the emergency physician to convey any suspicions to the primary care physician and to arrange for an EEG. It is unlikely that the patient will have another seizure during the recording, but the interictal record may display spike discharges that provide presumptive evidence of a seizure disorder.[79] There is no justification to withhold therapy if the EEG is abnormal. Those patients who have a single seizure and an epileptiform EEG when begun on anticonvulsants have fewer recurrences than do those for whom therapy is withheld until subsequent seizures.[80]

FAILURE TO RECOGNIZE AN ACTIVE SEIZURE

Limited morbidity and mortality should be associated with failure to recognize seizures manifested primarily as sensory disturbance, autonomic dysfunction, or behavioral change. Left unrecognized, seizures that present as confusional states unassociated with motor activity, could lead to bodily injury in those who are independently mobile. Similarly, those who lose body tone without exhibiting unusual motor activity may be subjected to repeated injuries. Those patients who develop altered consciousness are at risk of an unprotected airway. Patients at greatest risk of permanent damage or death are those who present with prolonged, generalized tonic-clonic seizures—major motor status epilepticus. These children may develop significant alterations in general energy requirements, cardiovascular hemodynamics, and cerebral metabolism. Patients in status may develop hypoglycemia, hyperkalemia, hyperpyrexia, dehydration, myoglobinuria, lactic acidosis, hypotension, respiratory failure, and raised intracranial pressure.[81] Ischemic changes may appear within 15 to 30 minutes after onset of

a continuous seizure and irreversible neuropathologic changes may occur within 60 to 90 minutes.[82] The development of permanent neurologic sequelae and potential for death correlates directly with the interval between onset and termination of the prolonged seizures. Unrecognized and untreated major motor status has carried as high as 10 to 12 percent mortality rates.[83,84]

FAILURE TO PROVIDE SUPPORTIVE CARE

As in any critically ill or injured patient, the physician who confronts a child with prolonged major motor seizure must institute general care measures that include provision of an airway and assuring adequacy of breathing and circulation. Failure to optimize and protect the airway may lead to upper airway obstruction or aspiration of saliva or vomitus. Failure to combat hypoxemia and hypoventilation may lead to cerebral anoxia, refractory acidosis, respiratory insufficiency, apnea, or pharmacologic failure to abort the seizure. Failure to achieve venous access makes fluid replacement and pharmacologic termination of the seizure difficult.

When airway-preserving maneuvers and supplemental oxygen are met with lasting pallor or cyanosis, assisted ventilation with bag-valve-mask is indicated after insertion of a nasogastric tube. When clinical examination or arterial blood gas confirms inadequate respiratory function, the clinician can choose between induction of apnea with benzodiazepines or a nondepolarizing neuromuscular blocking agent. Benzodiazepine induced apnea can be overcome with bag-valve-mask ventilation.[85] Respiratory depression with benzodiazepines is preferred by many to blocking agents and endotracheal intubation. The latter facilitates respiratory control but requires ongoing EEG monitoring, since peripheral motor activity is suppressed in the face of ongoing cerebral neuronal dysfunction.

SELECTING AN INTRAVENOUS DRUG TO TERMINATE STATUS EPILEPTICUS

Termination of major motor status need not be initiated contemporaneously with general care measures. Only seizures that do not spontaneously resolve require pharmacologic abortion. The intravenous route remains the preferred route for administration of anticonvulsants. It may take up to 5 minutes to establish a peripheral percutaneous intravenous catheter in a critically ill child.[86] Therefore, a seizure that presents for 5 to 10 minutes may tax vital functions during the ensuing 5 minutes, and efforts should be initiated to abort the seizure.

Those with a new-onset seizure can often be managed with a single

drug. Lorazepam, diphenylhydantoin, phenobarbital, paraldehyde, and diazepam are available for monotherapy of status epilepticus.

Lorazepam

Ativan, a newer benzodiazepine, is the ideal drug for the treatment of status epilepticus. This fast-acting drug has a long duration of action and limited toxicity.[87] The efficacy of Ativan, first noted in adults, has been documented for children of all ages. In two major studies, doses as low as 0.05 to 0.1 mg/kg have stopped status epilepticus in 75 percent of children within 10 minutes[88] and in 92 percent of children with a median latency of 6 minutes.[89] Several doses can be administered in 15-minute intervals if necessary to a maximum of 0.15 mg/kg. Seizure control for at least 3 to 6 hours can be expected in most patients, obviating the need for an immediate choice of a traditional long-acting anticonvulsant.[89] Mild and transient adverse effects noted are sedation, confusion, hallucination, tremor, ataxia, and vomiting.[90,91] Uncommon but significant adverse effects are a loss of brain stem reflexes, decorticate posturing, and difficulty in handling secretions.[87] Cardiovascular depression and respiratory depression have been seen only when used concomitantly with other central CNS depressants.[92]

Phenobarbital

For status epilepticus outside the neonatal period, between 10 to 20 mg/kg phenobarbital infused at a rate of 25 to 50 mg/min is therapeutically efficacious in abolishing status. Such conventional therapy in the newborn may fail to control convulsive activity, unless the loading dose is increased to 30 mg/kg.[93] The latency period has individual variation, but often status epilepticus abates during the continuous administration of the loading dose. Phenobarbital has the potential to depress the activity of nerves, skeletal muscles, smooth muscles, and cardiac muscle, as well as a number of cellular metabolic functions. Cardiorespiratory compromise and impaired cerebral blood flow are the immediate limiting toxicities and are encountered infrequently at lower infusion rates.[94]

Diphenylhydantoin

Between a 13 to 18 mg/kg loading dose of Dilantin at a rate of 25 to 50 mg/min is successful in abolishing status epilepticus in more than 80 percent of children.[95] Loading doses of up to 20 mg/kg must be used in the newborn infant.[96] Dilantin can be administered manually undiluted into a vein or an IV line not containing a dextrose solution or diluted with saline and administered with a constant infusion pump.[97] The controlled infusion reduces the variability in rate of delivery and is less likely to induce cardiovascular depression and cardiac dysrhythmia.[98]

Paraldehyde

Paraldehyde is a potent anticonvulsant that has long enjoyed the reputation of being a rapidly effective drug, equipotent to phenobarbital in the treatment of status epilepticus.[99] In the absence of hypotension or significant liver or pulmonary dysfunction, the drug may be a suitable alternative in children as a first-line drug for the treatment of status epilepticus when administered at 50 mg/kg/hr.[100] When infused intravenously at a controlled rate for several hours in the form of a 4 to 5 percent paraldehyde solution in 5 percent dextrose, reported complications from prior decades of increased coagulation of the blood, as well as pulmonary edema, pulmonary hemorrhage, metabolic acidosis, hypotension, or death are not seen.[101,102] Because paraldehyde controls seizures in newborns or older children that are refractory to phenobarbital and Dilantin, paraldehyde use is often reserved as a backup medication. In refractory seizures, successful control can be achieved with 200 mg/kg IV bolus followed by 16 mg/kg/hr in the newborn[103] and 20 mg/kg/hr in older children.[101]

Diazepam

The 88 percent success rate in status epilepticus of Valium compares favorably to the response rates quoted for Ativan.[104] Valium had been the drug of choice for status epilepticus since its introduction in the mid-1960s.[105] Neonates have been successfully treated with a continuous infusion of Valium diluted in saline delivering 0.3 mg/kg/hr of drug.[106] The predominant mode of therapy in all age groups has been direct intravenous injection of 0.1 to 0.25 mg/kg. When delivered slowly up to the point of a decrease in intensity of the seizure activity or until the patient evidences an audible sigh, it abolishes the seizure within minutes without depressing respiration. When given until the seizure ceases or after the patient sighs, respiratory arrest is often encountered. Apnea after parenteral administration may also be an idiosyncratic response or an additive response, when given with other respiratory depressant drugs.[85] Other reported toxicities, such as bradycardia, hypotension, thrombophlebitis, loss of a limb, and bronchospasm, are less frequently encountered.[107,108] To the disadvantage of Valium, seizure activity may resume within 10 to 30 minutes of initial arrest of seizure activity, necessitating either repeated administration of Valium or use of one of the conventional long-term anticonvulsants.[109]

CIRCUMSTANCES THAT PREJUDICE CHOICE OF AN INTRAVENOUS ANTICONVULSANT THERAPY

Circumstances may influence the selection of an intravenous anticonvulsant to suppress status epilepticus. Patients with status from impact seizures or bacterial meningitis should receive an anticonvulsant that has

Table 11-4. Significant Anticonvulsant Interactions

Primary Drug	Secondary Drug	Primary Drug	Secondary Drug
Aspirin	Dilantin		↑/↓
Ibuprofen			↑
Imipramine			↑
Isoniazid			↑
Aminophylline		↓	↑
Chloramphenicol		↑/↓	↑
Aminophylline	Phenobarbital	↓	
Chloramphenicol		↓	
Dilantin		↑/↓	↓
Phenobarbital	Paraldehyde		↑
Dilantin			↑

no adverse influence on mental or behavioral functions. Dilantin becomes the drug of choice, as it does not impair consciousness at therapeutic levels.[110]

Patients on chronic phenobarbital who present in status are likely to have subtherapeutic levels. They are best treated with a slow infusion of phenobarbital pending a drug level. It is best to avoid the use of Valium, as the synergistic effects with barbiturates may lead to prolonged hypotension, respiratory depression, or both.[111]

Patients on chronic Dilantin therapy who present in status epilepticus may be noncompliant or, conversely, intoxicated.[112] Valium or Ativan would be preferred therapy pending a phenytoin level. Patients in status epilepticus with underlying renal or hepatic disease who have compromised metabolizing capacities may benefit from paraldehyde due to its significant rate of pulmonary elimination.[113]

For adolescent girls who would be disturbed by hirsutism and children of all ages receiving orthodontic treatment, status epilepticus can be aborted by Dilantin, but a chronic antiepileptic regimen with this drug would be inappropriate.

Caution should be exerted in administering any anticonvulsant for abolishing status epilepticus if that drug may change the metabolism of the added drug or its own metabolism may change in the presence of the second drug. One must be cognizant of the clinically significant interactions that may result from changes in drug absorption, elimination, or distribution[114,115] (Table 11-4).

LACK OF VENOUS ACCESS

The physician who fails to achieve percutaneous, peripheral, or central venous access or who is unable to gain venous access by peripheral venous cutdown must choose another route to treat status epilepticus. Alternate

routes of anticonvulsant administration include intramuscular, rectal, endotracheal, and intraosseous.

Intramuscular Route

It is possible to administer Valium,[116] Dilantin,[117] and paraldehyde.[118] Because absorption may be erratic, local toxicity to tissues may occur and the length to achieving therapeutic levels is prolonged, intramuscular administration of these anticonvulsants should be avoided. Single administration of 10 mg/kg intramuscular phenobarbital irregularly produces serum levels at the lowest therapeutic range.[119] Higher therapeutic levels can be achieved with larger doses.[120] The 45 to 90 minutes it takes to attain therapeutic levels makes the intramuscular route less desirable.

Rectal Route

The undiluted intravenous preparation of Valium, when given per rectum, is absorbed rapidly. Although variable, levels can be achieved in the same range as with an intravenous injection.[121] At a dose of 0.23 to 0.45 mg/kg, therapeutic levels are achieved within 10 minutes and maintained for 30 minutes.[122] At higher doses of 0.5 to 0.75 mg/kg, to a maximum of 20 mg/kg, seizures are interrupted in 66 percent of children in less than 15 minutes.[123] Respiratory depression, the most serious complication, is unlikely, unless the patient is chronically taking barbiturates.[123]

Paraldehyde, at a dose of 0.3 ml/kg diluted twofold in mineral oil to enhance absorption when administered by rectum achieves onset of seizure control in 5 to 10 minutes. Although mucosal penetration may be erratic, the physician can recognize effective absorption by the smell of pulmonary excretion. Gastrointestinal hemorrhage is the limiting toxicity.[124]

Endotracheal Route

Prior to 1987, only a single case of endotracheal Valium use in a human subject was recorded in the literature.[125] Recently, undiluted Valium injectable was reported to stop status epilepticus in an infant at a dose of 0.1 mg/kg endotracheally when attempts at intravenous access proved unsuccessful.[126] Several studies have demonstrated adequate blood levels in experimental animals within 30 seconds of endotracheal of 0.5 mg/kg. Levels may be maintained above therapeutic threshold for at least 30 minutes.[127] However, the high incidence of pneumonitis seen in the experimental animal may preclude endotracheal use in the currently available commercial form.[126]

Intraosseous Route

The intraosseous route has been shown in the experimental animal to be an alternate means of rapid and effective administration of Valium. Comparable blood levels and seizure suppression to the intravenous route occur with the intraosseous infusion of 0.1 mg/kg Valium.[128] Intraosseous infusion of Valium in a child has been reported when intravenous access was not available.[127]

EXTENT OF THE EVALUATION

As seizures may be a manifestation of cerebral insult from diverse affections, a proper extent of laboratory assessment of an emergency patient with a completed seizure is unclear. A rational approach groups patients into categories of either first seizure, recurrent seizure, or seizure associated with fever.

First Seizure

Recent prospective and retrospective studies have commented on the standard seizure workup in adults who present to the emergency department with a seizure. The routine acquisition of laboratory investigations regardless of clinical features including chemistries, toxic screen, cultures, radiography, and CT is deplored.[129,130] It is everyone's impression that the yield of unselected screens in the pediatric seizure patient is similarly low. Pediatric patients can be categorized into metabolic, infectious, inflammatory, traumatic, and toxic categories on the basis of history and physical examination. Laboratory attempts to confirm a specific etiology for the seizure can be tailored to the clinical circumstance. The history and physical examination can accurately guide the clinician in choosing those diagnostic evaluations most likely to have an impact on a child who experiences a seizure.

One of the two exceptions includes neonates. These subjects have a limited repertoire of responses to cerebral insult, and a relatively high proportion of their seizures result from potentially correctable metabolic or inflammatory insults to the brain.[131] The second exception is the child with hypoglycemia. Causes of hypoglycemia are myriad, and the condition may complicate toxic, metabolic, and infectious conditions. It is therefore appropriate to screen all seizure patients who evidence altered sensorium with a glucose oxidase tape.

Despite my personal opinion, current literature recites the current litany of electrolytes, calcium, phosphorous, BUN, blood sugar, liver-function studies, hematologic profile, skull radiography, cultures of blood, stool, and cerebrospinal fluid.[32,132]

Recurrent Seizure

The most common cause of a recurrent seizure in the patient with a known seizure disorder is a subtherapeutic drug level. If there is no evidence of intercurrent infection, trauma, or metabolic derangement, the diagnostic investigations can be limited to anticonvulsant levels.

Seizure and Fever

Unfortunately, wide variation in diagnostic evaluation of febrile children with seizures has been reported.[133] If no historic or clinical risk factors for serious illness exist, diagnostic evaluation for the child who experiences the seizure in association with fever should be age dependent (Table 11-5).

A well-appearing child under 1 year of age without alarming historic features and having a negative examination should receive a lumbar puncture. The lumbar puncture should be performed regardless of a history of a previous seizure with fever or the finding of a source for the fever outside the CNS.[134] A complete blood count, zeta sedimentation ratio, erythrocyte sedimentation ratio, or C-reactive protein may be used as screening criteria for acquisition of a blood culture. This should be done only if the clinician is accustomed to acquiring a laboratory profile to aid the search for occult bacteremia,[135] or the clinician believes in presumptive antibiotic therapy of only that subset of patients with confirming laboratory data at risk of occult bacteremia.[136] Alternately, blood can be cultured without performance of screening laboratory procedures when patients exhibit temperatures above 39.5°C.[137] Urinalysis should be obtained for all female patients. Urine cultures should be sent if bacteruria or pyuria are present. Chest film should be obtained, even in the absence of objective findings in those with temperatures 40°C who have had fever for more than 12 hours.

Children aged 12 to 24 months who seize coincident with fever have equal or higher rates of bacteremia than comparably febrile children without seizures.[138] Therefore, a blood culture is justified. A lumbar puncture in the low-risk group is not mandatory and may be done selectively based on experienced clinical judgment.[139] The often quoted precaution of blunted nuchal signs in the immediate postictal phase in children under 16 months of age should hold.[140] If uncertainty concerning nuchal signs persist after the initial evaluation, repeated observation is advisable. When doubt remains, performance of the lumbar puncture is the safest choice. Urinalysis remains an appropriate and noninvasive investigation. Chest radiography is optional.

Diagnostic testing in low-risk children beyond 2 years of age should be tailored to the clinical situation. No diagnostic interventions may be appropriate in those without predisposing illness, significant exposure, negative review of systems, and a benign clinical appearance. Bacteremia without apparent focus of infection is uncommon beyond the second year. Beyond

Table 11-5. Diagnostic Adjuncts for Those Without Historical or Clinical Risks Who Seize with Fever

	Age		
Diagnostic Adjunct	6–12 months	12–24 months	>2 years
Lumbar puncture	Yes	Optional	Judgment
Blood culture	Yes	Yes	No
CBC, phase reactants	Yes	Yes	No
Urinalysis	Female	Female	Female
Chest radiograph	Yes	Optional	No

age 2, those with serious illness are consistently recognized on the basis of clinical observations.[141]

The febrile patients who seize and have associated risk factors for serious illness should receive prompt investigation (Table 11-5). The yield from biochemical investigations and cultures of body fluids is significant.[142] Those with suspected noninfectious space-occupying intracranial lesions, or altered cerebral blood flow should have an immediate CT scan.[143] Those who have suspected intracranial hypertension associated with intracranial infection should have a CT scan prior to contemplating lumbar puncture. It may be ill advised to subject a patient with either altered sensorium with little or no response to painful stimuli, ophthalmoplegia, pupillary dilation, papilledema, bulging fontanel, or protracted seizures to lumbar puncture prior to a diagnostic CT.[144] Such patients should have measures to reduce cerebral edema. They should have prompt administration of empirical antibiotic therapy after blood cultures and acquisition of countercurrent immunoelectrophoresis, enzyme-linked immunosorbent assay (ELISA), or Latex partical agglutination.[145] Those stable patients in whom the seizure is completed and who are suspected of uncomplicated intracranial infection should have phlebotomy prior to performance of lumbar puncture. The patient's glucose level should be estimated with a glucose oxidase tape. The estimated value should be confirmed with a serum sample also submitted for electrolyte determination. Blood culture and complete blood count are appropriate. Those not suspected of intracranial infection but who have a probability of significant electrolyte imbalance based on history of present illness should also have electrolytes, blood sugar, and blood urea nitrogen performed.[12] Other laboratory investigations are rarely warranted.

INDICATIONS FOR ADMISSION

Guidelines for hospitalization should be based on the type of seizure, cause of the seizure, general medical profile including the evolving neuro-

logic status, as well as the projected course over the next 6 to 8 hours and the quality of parenting.

Admission should be most liberal for patients with major motor seizures, as the potential for morbidity and mortality is greatest in this group. Those with prolonged or repetitive major motor seizures who require anticonvulsant administration to abolish the seizure are potential candidates for prolonged observation. Patients with seizures associated with critical underlying diseases (i.e., adrenal insufficiency, thyroid storm, or intracranial or metastatic tumor) require hospitalization. Those with seizures secondary to ingestion (i.e., theophylline, Isoniazid), exposure to environmental intoxicants (i.e., organophosphates), and overzealous therapeutics (diphenylhydantoin intoxication) and who require antidote administration or prolonged supportive care should be hospitalized. Patients who seize due to rapidly reversible metabolic derangement (i.e., hypoglycemia, hypocalcemia) may require admission for evaluation of biochemical imbalance. Children with tenuous vital signs, poor airway control, or general debilitation may require hospitalization for preventive or supportive care. Patients who experience a disturbing postictal phenomenon, such as prolonged altered sensorium, visual impairment, aphasia, or hemiparesis, are best admitted for observation. Those with poor guardianship may have to be admitted if immediate observation is to be guaranteed. Patients who may have been abused should be admitted.

Hospitalization of previously healthy febrile children with seizures is carried out more often than necessary.[146] General practitioners admit more patients than do pediatricians.[147] No data exist for emergency physicians. Hospital admission should be reserved for febrile children who have true hyperthermia illness or who meet high-risk criteria and have incomplete investigations of their present illness. Hospitalization is warranted for proven or suspected extracranial infection associated with significant risk of intracranial spread or for proven intracranial infection. If a holding area admission is not possible for febrile patients who seize, inpatient management may occasionally be necessary for alleviating parental anxiety.[148]

TRANSFER CONCERNS

The decision to transfer to another facility is best made in concert with the physician responsible for the patient's ongoing care. If the seizure activity is completed and the patient has an active gag reflex and exhibits no cardiorespiratory depression, transportation in the left lateral decubitus position under the watchful eye of a skilled attendent who has intravenous access is acceptable. Patients who are actively seizing, who lack life-sustaining blood pressure, pulse, and temperature, who have unsecured airways, respiratory depression, and unprotected cervical spine in the face of trauma, or who lack intravenous access should not be transferred.

References

1. Rutter N, Metcalfe DH: Febrile convulsions—What do parents do? Brit Med J 2:1345, 1978
2. Holmes GL: Partial seizures in children. Pediatr 77:725, 1986
3. Millichap JG: Drug treatment of convulsive disorders. N Engl J Med 286:464, 1972
4. Strauss E, Wada J, Kosaki B: Spontaneous facial expressions occurring at onset of focal seizure activity. Arch Neurol 40:540, 1983
5. Rodvanyi-Bouvet MF, Valleculle MH, Morel-Kahn F, et al: Seizures and electrical discharges in premature infants. Neuropediatrics 16:143, 1985
6. Thurston SE, Leigh JR, Osoric I: Epileptic gaze deviation and nystagmus. Neurol 35:1518, 1985
7. Rothner AD, Erenberg G: Status epilepticus. Pediatr Clin North Am 27:593, 1980
8. Wyllie E, Luders H, Morris HH, et al: The lateralizing significance of versive head and eye movement during epileptic seizures. Neurology 36:606, 1986
9. Volpe JJ: Neonatal seizures. Clin Perinatol 4:43, 1977
10. Donaldson IM: Volvular epilepsy: a distinct and underreported seizure type. Arch Neurol 43:260, 1986
11. Pazzaglia P, D'Alessandro R, Ambrosetto G, et al: Drop attacks: an ominous change in the evolution of partial epilepsy. Neurology 35:1715, 1985
12. Bettis DB, Ater SB: Febrile seizures: emergency department diagnosis and treatment. J Emerg Med 2:341, 1985
13. Lee SI: Nonconvulsive status epilepticus: ictal confusion in later life. Arch Neurol 42:228, 1985
14. Jacome DE, Risko MS: Absence of status manifested by compulsive masturbation. Arch Neurol 40:523, 1983
15. Watanabe K, Hara K, Hakamada S, et al: Seizures with apnea in children. Pediatrics 79:57, 1982
16. Coulter DL: Partial seizures with apnea and bradycardia. Arch Neurol 41:173, 1984
17. Amir J, Askenazi S, Schonfeld T: Laryngospasm as a single manifestation of epilepsy. Arch Dis Child 58:151, 1983
18. Horn CA, Beekman RH, Dick M, et al: The congenital long QT syndrome: an unusual cause of childhood seizures. Am J Dis Child 140:659, 1986
19. Moss AJ: Prolonged QT interval syndromes. JAMA 256:2985, 1986
20. Bricker JT, Garson A, Gillette PC: A family history of seizures associated with sudden cardiac deaths. Am J Dis Child 138:866, 1984
21. Mulroy JJ, Mickell JJ, Tong TK: Postictal pulmonary edema in children. Neurology 35:403, 1985
22. Volpe J: Neonatal seizures. N Engl J Med 289:1413, 1973
23. Jacobson M: Developmental Neurology. Plenum Press, New York, 1978
24. Singer JI: Acute bacterial meningitis. p. 1591. In Rosen P, Baker FJ (eds): Emergency Medicine Concepts and Clinical Practice. CV Mosby, St. Louis, 1983.
25. Watanabe K, Hara K, Shuji M: Electroclinical studies of seizures in the newborn. Folia Psychiol Neurol Japonica 31:383, 1977
26. Fenichel GM, Olson BJ, Fitzpatrick JE: Heart rate changes in convulsive and nonconvulsive apnea. Neurology 7:577, 1980

27. Vorhies TM, Vanucci RC: Perinatal cerebral hypoxia-ischemia: diagnosis and management. p. 61. In Sarnat HB (ed): Topics in Neurology. Grune & Stratton, Orlando, 1984
28. Phizackerly PJR, Poole EW, Whitly SWM: Sino-auricular heart block as an epileptic manifestation. Epilepsia 3:89, 1954
29. Dehkhani F, Sarnath B: Neonatal seizures. p. 209. In Sarnat HB (ed): Topics in Neonatal Neurology. Grune & Stratton, Orlando, 1984
30. Snyder CH: Conditions that simulate epilepsy in children: some bedside observations and reflections. Clin Pediatr 11:487, 1972
31. Oppenheimer EY, Rosman NP: Seizures and seizure-like states in the child: an approach to emergency management. Emer Med Clin North Am 1:125, 1983
32. Roddy SM, Ashwald S, Schneider S: Venipuncture fits: a form of reflex anoxic seizure. Pediatrics 72:715, 1983
33. Woody RC, Kile EA: Swallowing syncope in the child. Pediatrics 78:507, 1986
34. Lombroso CT, Lerman P: Breath holding spells (cyanotic and pallid infantile syncope). Pediatrics 39:563, 1981
35. Catz RM: Cough syncope in children with asthma. J Pediatr 77:48, 1970
36. Jennett B: Head injuries in children. Develop Med Child Neurol 14:137, 1972
37. Sarnat HB: Pathogenesis of the decerebrate "seizures" in the premature infant with intraventricular hemorrhage. J Pediatr 87:154, 1975
38. Gastau TH, Fischer-Williams M: Electroencephalographic study of syncope: Its differentiation from epilepsy. Lancet 2:1018, 1957
39. Waugh WH, Metts JC: Severe extrapramidal motor activity induced by prochlorperazine. N Engl J Med 262:353, 1960
40. Chalhub EG, DeVivo DC, Volpe JJ: Phenylhydantoin-induced dystonia and choreoathetosis in two retarded epileptic children. Neurology 26:494, 1976
41. Neglia JP, Glaze DG, Zion TE: Tics and vocalizations in children treated with carbamazepine. Pediatrics 73:841, 1984
42. Crosley CJ, Swender PT: Dystonia associated with carbamazepine administration: experience in brain-damaged children. Pediatrics 63:612, 1979
43. Kinast M, Erenberg G, Rothner AD: Paroxysmal choreoathetosis: report of five cases and review of the literature. Pediatrics 65:74, 1980
44. Burstein L, Breningstall GN: Movement disorders in bacterial meningitis. J Pediatr 109:260, 1986
45. Golden GS, French JH: Basilary artery migraines in young children. Pediatrics 66:722, 1975
46. Kremenitizer M, Golden GS: Hemiplegic migraine: cerebrospinal fluid abnormalities. J Pediatr 85:139, 1974
47. Verret S, Steele JC: Alternating hemiplegia in childhood: a report of eight patients with complicated migraine beginning in infancy. Pediatrics 47:675, 1971
48. Golden GS: The Alice in Wonderland syndrome in juvenile migraine. Pediatrics 63:517, 1979
49. Gascon G, Barlow C: Juvenile migraine, presenting as an acute confusional state. Pediatrics 45:628, 1970
50. Kales A, Kales JD: Sleep disorders: recent findings in the diagnosis and management of disturbed sleep. N Engl J Med 290:487, 1974
51. Elliott FA: The episodic discontrol syndrome and aggression. Neurol Clin 2:113, 1984

52. Delgado-Escueta AV, Matson RH, King L, et al: The nature of aggression during epileptic seizures. N Engl J Med 305:711, 1981
53. Sackellares JC, Giordani B, Barent S, et al: Patients with pseudoseizures: intellectual and cognitive performance. Neurology 35:116, 1985
54. Slater E: The diagnosis of hysteria. Brit Med J 1:1395, 1965
55. Schneider S, Rice DR: Neurologic manifestations of childhood hysteria. J Pediatr 94:153, 1979
56. Shulman K: Late complications of head injuries in children. p. 371. In Shillito J, Mosbery WH (eds): Clinical Neurosurgery. Williams & Wilkins, Baltimore, 1972
57. Jennett B: Trauma as a cause of epilepsy in childhood. Develop Med Child Neurol 15:56, 1973
58. Hendrick EB, Hardwood-Nash DC, Hudson AR: Head injuries in children: a survey of 4,465 consecutive cases at the Hospital for Sick Children, Toronto, Canada: p. 46. In Shillito J, Mosbery WH (eds): Clinical Neurosurgery. Williams & Wilkins, Baltimore, 1964
59. Bruce DA, Schut L: The value of CAT scanning following pediatric head injury. Clin Pediatr 19:719, 1980
60. Mills ML: Russo LS, Vines FS, et al: High-yield criteria for urgent cranial computed tomography scans. Ann Emerg Med 5:1167, 1986
61. Diechman NR: Seizures in children. p. 1017. In Decker BD (eds): Current Therapy in Emergency Medicine. CV Mosby, St. Louis, 1987
62. Gururaj VJ: Febrile seizures: current concepts. Clin Pediatr 19:731, 1980
63. Wolf SM, Carr A, Davis DC, et al: The value of phenobarbitol and the child who has had a single febrile seizure: a controlled prospective study. Pediatrics 59:378, 1977
64. Fishman MA: Febrile seizures: the treatment controversy. J Pediatr 94:117, 1979
65. Nelson KB, Ellenberg JH: Prognosis in children with febrile seizures. Pediatrics 61:720, 1978
66. Felter RA, Asch SM: Febrile seizures: a protocol for emergency management. Ped Emerg Care 2:93, 1986
67. Lennox-Buchtal MA: Febrile convulsions: a reappraisal. EEG Clin Neurophysiol 23(Suppl):1, 1973
68. Ouellette EM: The child who convulses with fever. Pediatr Clin North Am 21:467, 1974
69. Anderson AB, DeSisto J, Marshall P, et al: Duration of fever prior to onset of a simple febrile seizure: a predictor of significant illness and long term outcome. (Abst) American Pediatric Association, 1986
70. Joffe A, McCormick M, DeAngelis C: Which children with febrile seizures need lumbar puncture. Am J Dis Child 137:1153, 1983
71. Gerber MA, Berliner BC: The child with a "simple" febrile seizure. Am J Dis Child 135:431, 1981
72. Lorin M: Febrile convulsions. p. 153. In The Febrile Child: Clinical Management of Fever and Other Types of Pyrexia. John Wiley and Sons, New York, 1982
73. Ashkenezi S, Dinari G, Zevulunova, et al: Convulsions in childhood shigellosis: clinical and laboratory features in 153 children. Am J Dis Child 141:208, 1987

74. McCarthy PL, Jekel JF, Staswick SA, et al: Further definition of history and observation variables in assessing febrile children. Pediatrics 67:687, 1981
75. Caspe WB, Chamudes O, Louie B: The evaluation and therapy of the febrile infant. Pediatr Infect Dis 2:131, 1983
76. Wright PH: Patterns of illness in highly febrile young child: epidemiologic, clinical and laboratory correlates. Pediatrics 67:694, 1981
77. McCarthy PL, Rembo RM, Barren MA, et al: Predictive value of abnormal physical examination findings in ill appearing and well appearing febrile children. Pediatrics 67:167, 1985
78. Tomlanovich MC, Rosen P, Mendensohn J: Simple febrile convulsion. J Am Coll Emer Phys 5:347, 1976
79. Wright ES: Epilepsy in childhood. Pediatr Clin North Am 31:177, 1984
80. Canfield PR, Canfield CS, Smith EL, et al: Newly treated childhood epilepsy: a prospective study of recurrences and side effects. Neurology 35:722, 1985
81. Plum F, Posner JB, Troy B: Cerebral metabolic and circulatory responses to induced convulsions in animals. Arch Neurol 18:1, 1968
82. Meldrum BS, Brierley JB: Prolonged epileptic seizures in primates. Arch Neurol 28:10, 1973
83. Oxbury JM, Whitty CW: Causes and consequences of status epilepticus in adults: a study of 86 cases. Brain 94:733, 1971
84. Delgato-Escueta AV, Bojorek GJ: Status epilepticus: mechanisms of brain damage and rationale management. Epilepsia 23(Suppl 1): 29, 1982
85. Hall SC, Ovassapian A: Apnea after intravenous diazepam therapy. JAMA 238:1032, 1977
86. Kanter RK, Zimmerman JJ, Strauss RH, et al: Pediatric emergency intravenous access. Am J Dis Child 140:132, 1986
87. Levy RJ, Krall RL: Treatment of status epilepticus with lorazepam. Arch Neurol 41:605, 1984
88. Crawford TO, Mitchell WG: Lorazepam for status epilepticus in serial seizures in children: 218 consecutive cases. Ann Neurol 18:412, 1983
89. Lacey DJ, Singer WD, Horwitz SJ, et al: Lorazepam therapy of status epilepticus in children and adolescents. J Pediatr 108:777, 1986
90. Sorel L, Mechler L, Harmant J: Comparative trial of intravenous lorazepam and clonazepam in status epilepticus. Clin Ther 4:326, 1981
91. Walker JE, Holman JW, Vasko MR, et al: Lorazepam and status epilepticus. Ann Neurol 6:207, 1979
92. Griffith PA, Carp HR: Lorazepam in therapy for status epilepticus. Ann Neurol 7:493, 1980
93. Donn SM, Grasela TH, Goldstein GW: Safety of a higher loading dose of phenobarbital in the term newborn. Pediatrics 75:1061, 1985
94. Tomlanovich M, Yee AS: Seizures. p. 1339. In Rosen P, Baker FJ (eds): Emergency Medicine Concepts and Clinical Practice. CV Mosby, St. Louis, 1983
95. Earnst MP, Marx JA, Drury CR: Complications of intravenous phenytoin for acute therapy of seizures: recommendations for usage. JAMA 249:762, 1983
96. Painter MJ, Pippenger C, McDonald IT, et al: Phenobarbital and diphenlyhydantoin levels in neonates with seizures. J Pediatr 92:315, 1978
97. Cloyd JC, Gumnit RC, McLain Jr W: Status epilepticus: the role of intravenous phenytoin. JAMA 244:1479, 1980
98. Carducci B, Hedges JR, Beal JC, et al: Emergency phentoin loading by constant intravenous infusion. Ann Emerg Med 13:1027, 1984

99. McGrial DA: Emergency therapy of convulsions in childhood. Practitioner 181:719, 1985
100. Curless RG, Holzman BH, Ramsey RE: Paraldehyde therapy in childhood: status epilepticus. Arch Neurol 40:477, 1983
101. Bostrom B: Paraldehyde toxicity during therapy of status epilepticus. Am J Dis Child 136:414, 1982
102. Giacoia GP, Gessner PK, Zaleski MM, et al: Pharmacokinetics of paraldehyde distribution in the neonate. J Pediatr 104:291, 1984
103. Koren G, Butt W, Rajchgot P, et al: Intravenous paraldehyde seizure control in newborn infants. Neurology 36:108, 1986
104. Delgotta-Esqueta AV, Wasterlain C, Treiman DM, et al: Management of status epilepticus. N Engl J Med 306:1337, 1982
105. Lombroso CT: Treatment of status epilepticus with diazepam. Neurology 16:629, 1966
106. Gamstorp PI, Sedin G: Neonatal convulsions treated with continuous intravenous infusion. Ups J Med Sci 87:143, 1982
107. Schneider S: Loss of limb following intravenous diazepam. Pediatrics 53:112, 1974
108. Grenblat DJ, Coch-Weser J: Adverse reaction to intravenous diazepam: a report of the collaborative drug surveillance program. Ann Intern Med 266:261, 1973
109. Canfield PR: Treatment of status epilepticus in children. Can Med Assoc J 128:671, 1973
110. Wallis W, Kutt IT, McDowell F: Intravenous dilantin therapy of acute repetitive seizures. Neurology 18:513, 1968
111. Duffy TH, Lombroso CT: Therapy of status epilepticus. Clin Neuropharmacol 3:41, 1978
112. Holcomb R, Lynn R, Harvey B, et al: Intoxication with dilantin. J Pediatr 80:627, 1972
113. Lang DW, Borgstedt HH: Rate of pulmonary excretion of paraldehyde in man. Toxicol Appl Pharmacol 15:269, 1969
114. Conney AH: Pharmacologic implications of microzomal enzyme induction. Pharmacol Rev 19:317, 1967
115. Garrettson LK: Pharmacology of anticonvulsants. Pediatr Clin North Am 19:179, 1972
116. Hillestad L, Hansen T, Milson H, et al: Diazepam metabolism in normal man: serum concentration and clinical effects after intravenous, intramuscular, and oral administration. Clin Pharm and Therap 16:479, 1974
117. Cantu RC, Schwab RS, Timberland WH: Comparison of blood levels with oral and intramuscular diphenylhydantoin, and paraldehyde intramuscularly. Neurology 18:782, 1968
118. Hitchcock P, Nelson E: The metabolism of paraldehyde. J Pharmacol Exp Ther 67:299, 1939
119. Brachet-Liermain A, Grutieres F, Aicar DIJ: Absorption of phenobarbital after the intramuscular administration of single doses in infants. J Pediatr 87:624, 1975
120. Jalling GB: Plasma and cerebrospinal fluid concentrations of phenobarbital in infants given single doses. Dev Med Child Neurol 16:781, 1974
121. Dulac O, Aicardi J, Ray E, et al: Blood levels of diazepam after single rectal administration in infants and children. J Pediatr 93:1039, 1978

122. Aqurell S, Berlin A, Ferngren H, et al: Plasma levels of diazepam following parental and rectal administration in children. Epilepsia 16:277, 1975
123. Hoppu K, Santavuori P: Diazepam rectal solution for home treatment of acute seizures in children. Acta Pediatr Scand 70:369, 1981
124. Gardner HL, Levine H, Bodanski M: Concentrations of paraldehyde in the blood following its administration during labor. Am J Obstet Gynecol 40:435, 1940
125. Pasternak SJ, Heller MB: Endotracheal diazepam in status epilepticus (letter). Ann Emer Med 14:485, 1985
126. Rusli M, Spivey WH, Bonner H, et al: Endotracheal diazepam: absorption in pulmonary pathologic effects. Ann Emerg Med 16:314, 1987
127. Barsan WG, Ward JI, Otten EJ: Blood levels of diazepam after endotracheal administration in dogs. Ann Emerg Med 11:242, 1982
128. Spivey WH, Unger HD, Later CM, et al: Intraosseous diazepam suppression of phentylmeterazol-induced epileptogentic activity in pigs. Ann Emerg Med 16:156, 1987
129. Powers RD: Serum chemistry abnormalities in adult patients with seizures. Ann Emerg Med 14:416, 1985
130. Eisner RF, Turnbull TL, Howes DS, et al: Efficacy of a "standard" seizure work up in the emergency department. Ann Emerg Med.15:33, 1986
131. Dodson WE, Prensky AL, DeVido DC: Management of seizure disorders: selected aspects. J Pediatr 89:527, 1976
132. Nigro MA: Pediatric seizures and status epilepticus. In Tintanalli, Rothstein, Krome (eds): Emergency Medicine: A Comprehensive Study Guide. McGraw Hill, New York, 1985
133. Chessare JB, Berwick DM: Variation in clinical practice in the management of febrile seizures. Ped Emer Care 1:19, 1985
134. Pichichero ME: Problems in diagnosing occult bacteremia in the febrile child. ER Reports 1:119, 1980
135. Singer J, Bucchino JJ, Chabali R: Selected laboratory examinations in pediatric emergency medicine care. Emer Med Clin North Am 4:377, 1986
136. Carroll WL, Farrell MK, Singer JI, et al: The treatment of occult bacteremia: a prospective randomized clinical trial. Pediatrics 72:608, 1983
137. Waskerwitz S, Berkelhomer JE: Outpatient bacteremia: clinical findings in children under 2 years with temperature of 39.5 C or higher. J Pediatr 99:231, 1981
138. Crocker BJ: Occult bacteremia in the emergency department. Ann Emer Med 13:45, 1984
139. Lorber J, Sunderland R: Lumbar puncture in children with convulsions associated with fever. Lancet 1:785, 1980
140. Sampson JH, Apthorp J, Finley A: Febrile seizures in perulent meningitis. JAMA 210:1918, 1969
141. McCarthy PC, Grundy GW, Spiesel SZ, et al: Bacteremia in children: an outpatient clinical review. Pediatrics 57:861, 1976
142. Jaffe M, Bar-Joseph G, Tirosh E: Fever and convulsions: indications for laboratory investigations. Pediatrics 67:729, 1981
143. Kirks DR, Harwood-Nash DL: CT in pediatric radiology. Pediatric Annals 9:53, 1980
144. Heldrich FJ, Walker SH, Crosby RM: Risk of diagnostic lumbar puncture in acute bacteria meningitis. Ped Emerg Care 2:180, 1986

145. Dogbjartsson A, Ludrigsson J: Bacterial meningitis and initial antibiotic therapy. Pediatr Clin North Am 34:219, 1987
146. Surpure JS: Febrile convulsions: what happens to the infant admitted to the hospital. Clin Pediatr 19:361, 1980
147. Hirtz DG, Young JL, Ellenberg JH, et al: Survey on management of febrile seizures. Am J Dis Child 140:909, 1986
148. Wolk SM: Laboratory evaluation of the child with febrile convulsions. Pediatrics 62:1074, 1978

12 Problems in the Management of Respiratory Distress

Kim A. Ogle

RESPIRATORY DISTRESS

Respiratory failure is the preceding event in most pediatric arrests. Because respiratory failure most commonly follows a variable period of respiratory distress, identification of the child with respiratory distress and early intervention may prevent the progression to respiratory failure and cardiac arrest. Assessment of the patient must be rapid and many times is done simultaneously with treatment. The child's color, mental status, and work of breathing should be quickly assessed.

Suprasternal and infrasternal retractions are the hallmark of increased work of breathing or respiratory distress. As the pediatric patient's work of breathing increases, accessory muscles are recruited. Initially, intercostal retractions appear, followed by suprasternal and supraclavicular retractions and nasal flaring as the distress worsens. The severity and depth of the retractions may lessen in the child with severe respiratory distress who becomes fatigued and can no longer maintain the increased work of breathing. This child will have other signs of impending respiratory failure (i.e., behavioral changes and/or cyanosis) (Fig. 12-1). The patient who presents with respiratory complaints, looks well, and has no signs of increased work of breathing is not in respiratory distress. Cyanosis is an ominous sign, if present. Significant hypoxemia can exist in infants and young children

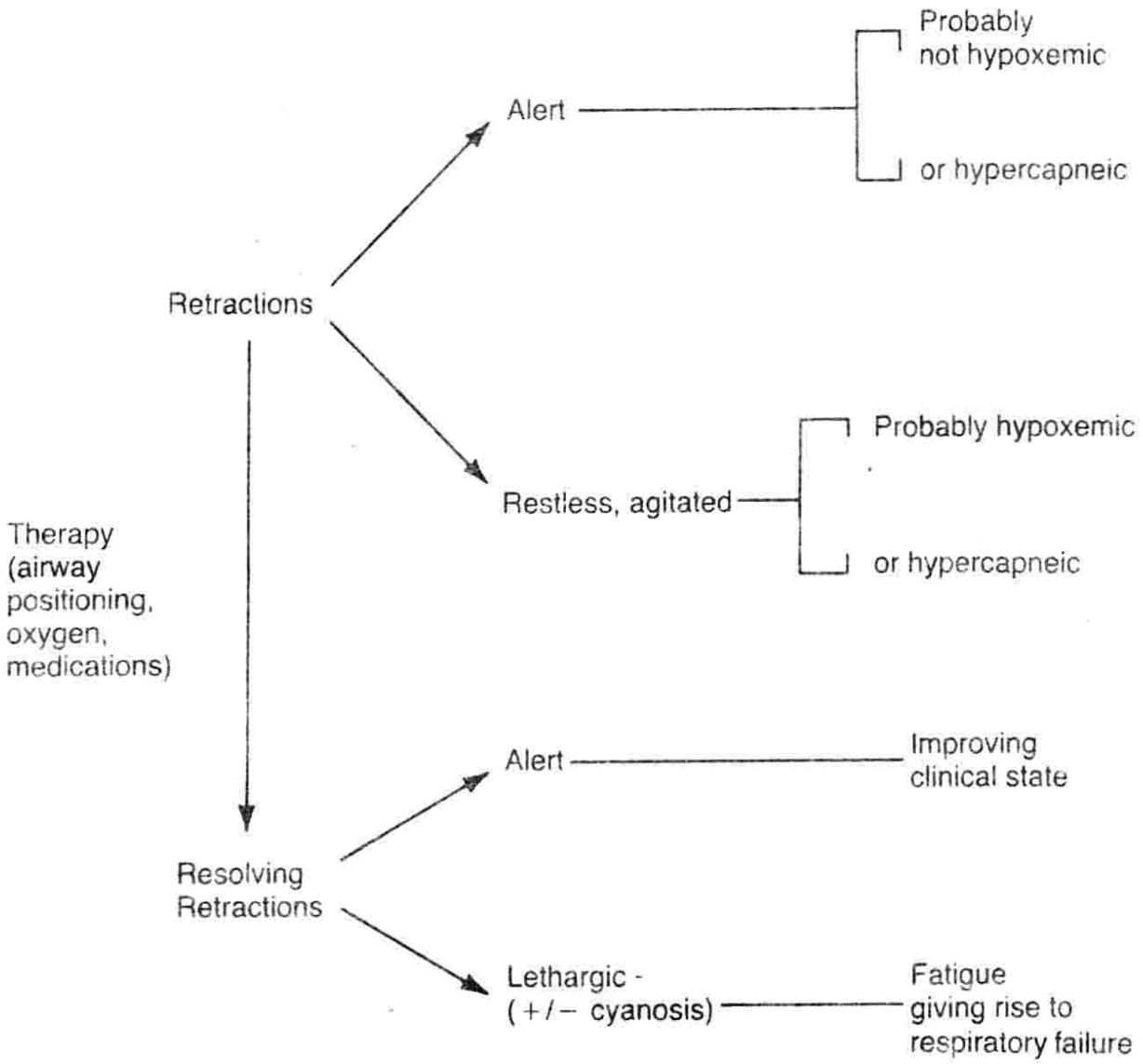

Fig. 12-1 Evaluation of the child with increased work of breathing.

without cyanosis because of their relative anemia and ability to increase cardiac output, compared with the older child and adult. Behavior is an extremely important clue to hypoxemia and respiratory acidosis. The young pediatric patient who is restless and agitated may be hypoxemic. Headache and mental confusion in an older child are signs of decreased PaO_2. Increased $PaCO_2$ usually follows hypoxemia as respiratory distress worsens. The child with CO_2 retention is lethargic and somnolent.

All children in respiratory distress should receive high concentrations of supplemental oxygen, but only if tolerated. The oxygen should not be administered if it causes the pediatric patient to become agitated because crying and agitation worsen airway obstruction. If the child is alert the oxygen should be delivered by whatever method causes the least agitation. Young children and infants tolerate oxygen tubing held close to the face by the parent. Older children usually tolerate a face mask. In the patient with a depressed sensorium airway patency must be ensured by suctioning and positioning maneuvers, before administering oxygen. The patient should be allowed to assume a position of comfort. A child with respiratory distress

should not be forced to lie down on a stretcher. Infants and young children are more comfortable on a parent's lap. All children should be allowed to remain close to their parents. Noxious stimulation should be kept to a minimum.

The child who is apneic on arrival in the emergency department needs rapid airway and breathing assistance to prevent cessation of cardiovascular function and irreparable central nervous system damage. The child in respiratory failure should not have airway intervention delayed. The problem is to decide when overt respiratory failure is present. It is a clinical diagnosis, which can be supported by laboratory values. The clinical signs of respiratory failure include (1) decreased or absent breath sounds, (2) severe retractions and accessory muscle use, (3) cyanosis in 40 percent oxygen, (4) decreased level of consciousness and response to pain, (5) poor muscle tone, and (6) weak to absent cough or gag reflex.[1] Any child presenting with respiratory distress should be assumed to have the potential for deterioration to respiratory failure and have immediate intervention that may preclude a complete history and physical examination. Once the patient is stabilized, the details of the history and a complete physical examination may be performed.

Respiratory distress can result from disturbances in any of the systems contributing to the normal function of breathing. Causes of respiratory distress include CNS disorders that alter the state of consciousness, resulting in either hypoventilation or hyperventilation; metabolic disturbances causing acidosis; neuromuscular and structural disorders altering the mechanics of respiration and disorders within the respiratory tract. A careful history and physical examination should identify the system dysfunction responsible for the respiratory distress. Dysfunction within the respiratory tract is divided into three areas: obstructive disorders of the upper and lower airways and alveolar/interstitial lung disorders. Physical examination should determine the area of disorder within the respiratory tract. Posture is an important identifying sign. The child with upper airway obstruction often sits upright with the neck slightly flexed and head slightly extended on the neck. The tripod position is assumed by patients with lower airway obstruction disease. Upper airway obstruction causes suprasternal and supraclavicular retractions; intercostal and subcostal retractions occur with lower airway obstructive diseases. Severe obstruction of either upper or lower airways causes retractions of all the accessory muscles. Grunting is a sign of respiratory distress, caused by an infant exhaling against a partially closed glottis in an attempt to generate positive end expiratory pressure. Grunting is associated with interstitial and aveolar diseases such as pneumonia or pulmonary edema. Grunting usually signals that the child is in severe distress.

Assessing the depth of respiration in children may aid in determining the cause of respiratory distress. Metabolic acidosis causes deep rapid respirations while obstructive lesions usually cause a decreased depth of respiration. An estimation of the depth of respiration can be made by placing a

hand in front of the child's nose. Normally airflow can be felt a measurable distance away, usually 2 to 3 inches in infants under 3 months, 4 inches up to 12 months of age, and 5 to 6 inches for 12 to 24 months.[2] The quality of breath sounds, length of inspiration and expiration, and phase of respiration in which abnormal breath sounds occur, offer important clues to the origin of the respiratory distress. Stridor is a musical inspiratory noise associated with upper airway obstruction. Lower airway obstruction initially causes expiratory wheezing but as the obstruction worsens, airway collapse occurs in inspiration as well. The normal inspiration expiration ratio is 1 : 1.5. During inspiratory obstruction the ratio may be 1 : 1 or as much as 2 : 1. Lower airway obstruction prolongs the expiratory phase altering the ratio to 1 : 3 or 1 : 4.[3] Patients who have severe airway obstruction may not have sufficient air entry to generate wheezes or stridor. Difficulty occurs in differentiating inspiratory and expiratory sounds in young children and infants with rapid respiratory rates. Correllating the auscultatory findings with observation of the chest and abdominal wall motion may help in determining whether the sounds are inspiratory or expiratory. Rales are associated with aleveolar/interstitial processes but often are heard in patients with obstructive disorders as well.

UPPER AIRWAY OBSTRUCTION

Assessment

The causes of upper airway obstruction in the pediatric patient are many (Table 12-1). Acute airway obstruction is generally due to viral or spasmodic croup, acute supraglottitis, and foreign body aspiration. Differentiating between the causes of upper airway obstruction is important because their management is different.

A foreign body in the extrathoracic airway can cause inspiratory stridor and a croupy cough. There is often a history of the child playing with small objects followed abruptly by choking and coughing. The child is often unable to talk.[4]

The typical course of viral croup begins with URI symptoms followed by the development of a barky high-pitched cough; 6 to 24 hours later, the child develops hoarseness and inspiratory stridor. Wheezing may develop if the bronchi are involved in the infectious process. Low grade fever is variable and mild leukocytosis is occasionally seen. The inspiratory stridor and cough typically get worse at night, with crying and agitation. This picture usually lasts 3 to 5 days, during which time the symptoms may remain unchanged or progress, and is followed by a convalescent period of 2 to 3 days.[5]

Supraglottitis is an acute rapidly progressive bacterial illness. After a brief period of sore throat and URI symptoms, usually less than six hours, the child with supraglottitis rapidly develops a high fever, lethargy and dysphagia. With progression of the illness, the child becomes toxic appear-

Table 12-1. Differential Diagnosis of Upper Airway Obstruction

Congenital
Subglottic hemangioma
Cystic hygroma
Pharyngeal cysts
Laryngomalacia
Tracheomalacia
Acquired
Neoplastic conditions
Inflammatory conditions
Laryngotracheobronchitis
Supraglottitis
Bacterial tracheitis
Pharyngeal abscess
Miscellaneous
Spasmodic croup
Vocal cord paralysis
Subglottic stenosis
Foreign bodies

ing, has a muffled voice or is unable to speak. The child begins to drool and assumes a characteristic posture, sitting upright with the head and neck leaning slightly forward. If inspiratory stridor is present, it is usually softer and lower in pitch than the stridor from croup. Cyanosis and suprasternal retractions are present with severe obstruction.[6]

The child who presents with the classic history and appearance of croup or supraglottitis is not difficult to recognize, but the presentation is not always so clear cut. Younger children may not have the typical signs and symptoms. Singer and McCabe[7] reviewed the cases of epiglottitis in patients less than 24 months seen in their institution over a 10-year period. Only 5 of the 19 children exhibited an abrupt onset of a febrile illness associated with impairment of swallowing, vocalization, and inspiration. Seven of the 19 patients had prodromal symptoms, including cough, rhinorrhea, and anorexia for more than 12 hours. Dysphagia, retention of oral secretions, and drooling were uncommon. Only four of the children assumed the characteristic sniffing posture. The only sign present in all the children was high fever, temperature greater than 39.3°C. In only 8 of the 19 patients was the diagnosis of epiglottitis suspected after the initial history and physical examination. The other 11 children were erroneously thought to have croup, reactive airway disease, or bacteremia. Among this group of 11 children, three had respiratory arrests and one child died while being evaluated or treated for these other illnesses. In a series of 33 cases of epiglottitis reported by Welch and Price,[8] 13 of the children had a prodromal coryzal phase of 24 hours or longer. The clinical pictures of croup and epiglottitis are not always distinct. The clinician evaluating young children with signs and symptoms of upper airway obstruction needs to be cognizant that atypi-

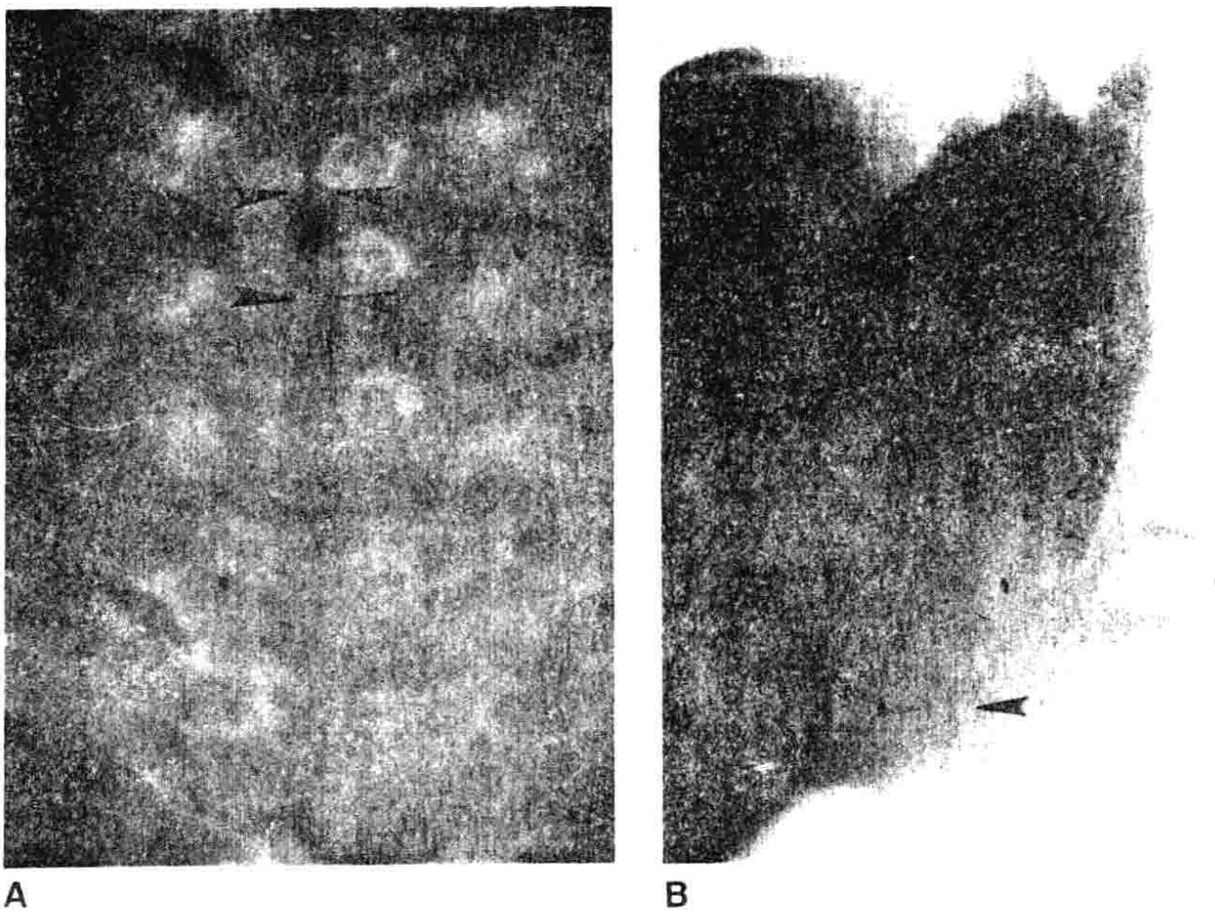

Fig. 12-2 (**A**) Croup frontal view, showing typical funnel-shaped glottic and subglottic narrowing in infant croup. (**B**) Croup lateral view, with typical findings including marked overdistention of hypopharynx and paradoxical narrowing of the subglottic trachea (arrows). The epiglottis and aryepiglottic folds are normal. (Swischuk LE: Emergency Radiology of the Acutely Ill or Injured Child. Williams & Wilkins, Baltimore, 1987.)

cal presentations do occur. Early recognition of supraglottitis in this special group of patients should improve their outcome.

Soft tissue radiographs of the neck are very helpful in the evaluation of upper airway obstruction in the pediatric patient. Radiologic examination may locate foreign bodies, identify subglottic edema or stenosis and other causes of acute and nonacute obstruction. Lateral neck films aid in differentiating between croup and epiglottitis. The films are helpful in the management of the child in whom the diagnosis of croup or supraglottitis is not apparent. The lateral neck radiograph of the patient with supraglottitis and croup has features in common, i.e. hypopharyngeal distention and trachael collapse on inspiration. The anterior-posterior cervical film of the child with croup may show a long segment of tracheal narrowing (steeple sign)[9] (Fig. 12-2). Initially, the lateral neck radiograph of the patient with supraglottitis may demonstrate only swelling of the aryepiglottic folds, which is a key to early diagnosis of the disease, with progression the epiglottitis becomes swollen resembling the size and configuration of an adult thumb[10] (Fig. 12-3).

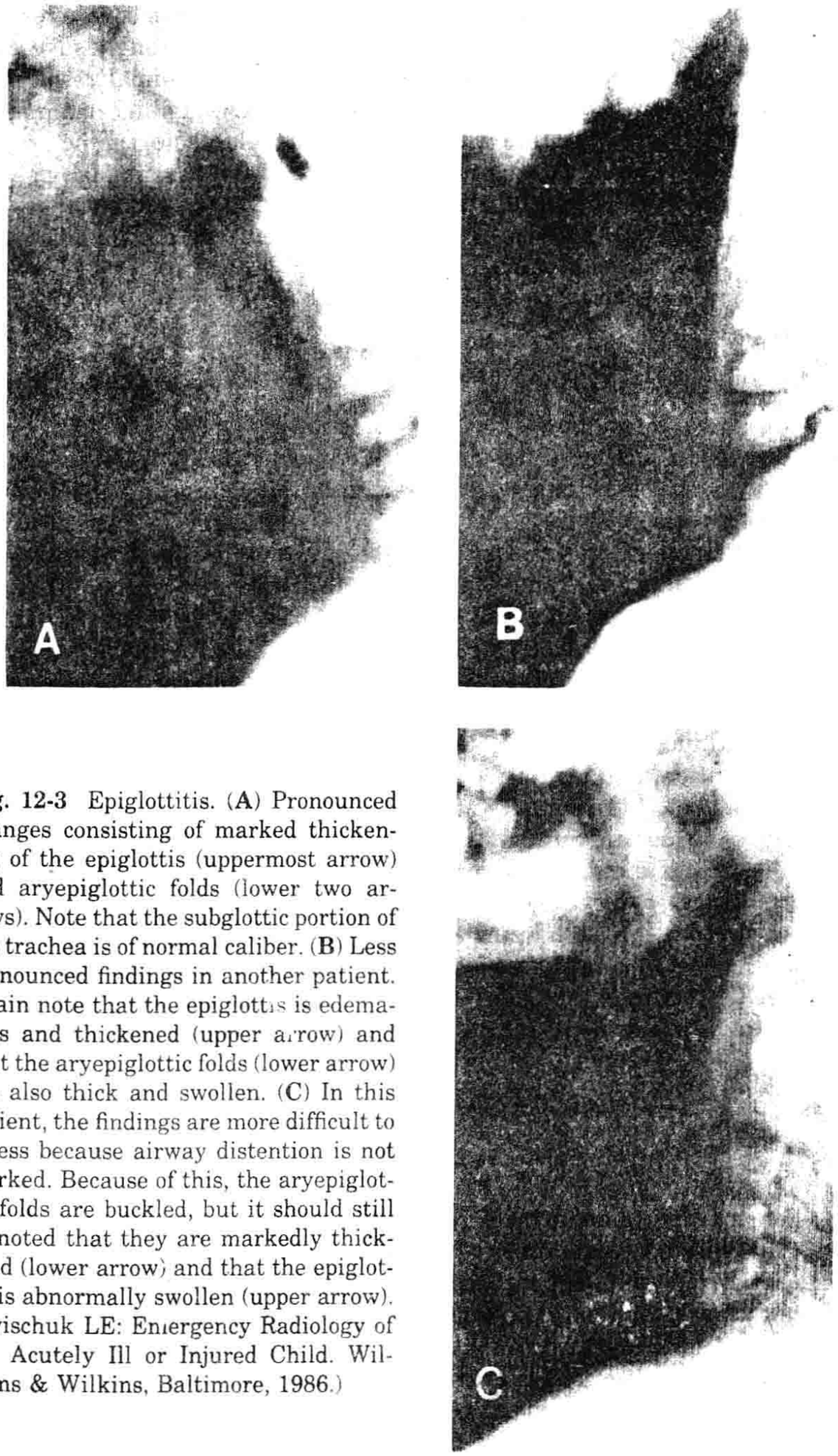

Fig. 12-3 Epiglottitis. (**A**) Pronounced changes consisting of marked thickening of the epiglottis (uppermost arrow) and aryepiglottic folds (lower two arrows). Note that the subglottic portion of the trachea is of normal caliber. (**B**) Less pronounced findings in another patient. Again note that the epiglottis is edematous and thickened (upper arrow) and that the aryepiglottic folds (lower arrow) are also thick and swollen. (**C**) In this patient, the findings are more difficult to assess because airway distention is not marked. Because of this, the aryepiglottic folds are buckled, but it should still be noted that they are markedly thickened (lower arrow) and that the epiglottis is abnormally swollen (upper arrow). (Swischuk LE: Emergency Radiology of the Acutely Ill or Injured Child. Williams & Wilkins, Baltimore, 1986.)

The child who presents to the emergency department in severe respiratory distress, or in whom the diagnosis of epiglottitis is strongly suspected, should not have radiographs done.[6] For the child in minimal respiratory difficulty and an unclear clinical picture, the lateral neck film can aid in his management. These children should have portable radiographic studies done in the emergency department, preferably in the parent's lap or with the parent near by. The child who has to be taken to the radiology department should be transported with airway equipment and accompanied by a person skilled in airway management.[5,6,11–13]

CROUP

Management

The clinical picture of the child with croup is related to the severity of his airway obstruction. The patient with mild croup has only a hoarse voice, croupy cough and stridor when agitated. With worsening obstruction stridor at rest develops. As the disease progresses, the child's distress increases. The child becomes tachypneic and tachycardic and develops suprasternal retractions and sternal recession. As the child becomes fatigued, stridor and retractions diminish and signs of hypoxemia develop. An unwary observer seeing the child at this stage may conclude that the child is improving because the stridor is quieter and the retractions milder. If this downhill course continues interrupted, the hypoxemia will worsen, resulting in $PaCO_2$ retention, and leading to respiratory arrest.

Assessment of the severity of croup is often difficult. Air entry and the intensity of stridor are difficult to assess objectively.[13,14] The intensity of the noise the child makes does not correlate well with the degree of obstruction. Respiratory rate and heart rate have been found to correlate with the severity of the hypoxemia, both rising as PaO_2 declines.[9] Clinical scoring systems have been developed to aid the clinician in judging severity and monitoring response to treatment[15,16] (Table 12-2). Arterial blood gas analysis is useful in the initial assessment and to follow the child's course and response to therapy.[8]

MIST

For inpatient management of croup, the child is placed in a cool mist tent. The efficacy of cool mist is debated and its mode of action unknown, but some children seem to benefit from the humidified air. Animal studies have shown that droplet deposition on upper airways may reflexly alter the pattern of breathing and improve air flow rates.[5] Mist tents are generally not available in the emergency department, but the child with mild croup may be given aerosolized normal saline. The child with moderate to severe croup

Table 12-2. Subjective Assessment of Clinical Severity of Croup[a]

	0	1	2	3
Stridor	None	Mild	Moderate at rest	Severe on inspiration and expiration or none with markedly decreased air entry
Retraction	None	Mild	Moderate	Severe, marked use of accessory muscles
Air entry	Normal	Mild decrease	Moderate decrease	Marked decrease
Color	Normal	Normal (0-score)	Normal (0-score)	Dusky or cyanotic
Level of consciousness	Normal when undisturbed	Restless when disturbed	Anxious, agitated; restless when undisturbed	Lethargic, depressed

[a] Score: 5,mild; 5–6, mild to moderate; 7–8, moderate—most cases admitted; 8, severe—admit (or if child has any one sign in severe category).

(David HW: Croup and epiglottitis. Pediatr Clin North Am 28:866, 1981.)

Fig. 12-4 Aerosol delivery using paper cup mask.

can receive nebulized normal saline with oxygen in between racemic epinephrine treatments while awaiting admission. The aerosol can be administered by face mask held in front of the child's face (Fig. 12-4). The parents of the patient with mild croup should be instructed in the use of a cool mist vaporizer and sheets to make a mist tent in the child's crib and using the steamy bathroom for exacerbations at home.

RACEMIC EPINEPHRINE

Racemic epinephrine has been used in the treatment of croup for more than 15 years. Racemic epinephrine is a mixture of L and D isomers of epinephrine. The L isomer is 30 times more active. Racemic epinephrine was originally chosen because it was believed to have fewer cardiovascular side effects than L-epinephrine; however, there is no pharmacologic basis for this belief.[16] The α-adrenergic effect of the racemic epinephrine is believed to shrink the edematous subglottic mucosa and the β-agonist effect may help by reflexly reducing laryngospasm or tracheal muscle constriction.[5,17]

Racemic epinephrine is administered as an aerosol, 0.5 ml of the 2.25 percent solution diluted with 3.5 ml normal saline. Early in its use, racemic epinephrine was administered by intermittent positive-pressure breathing (PPB), but several studies have found simple face mask nebulization to be as effective.[18–20] Racemic epinephrine can be given as often as every 20 to 30 minutes or can be spaced 4 to 6 hours apart, depending on the severity of the croup and the patient's heart rate.[5] Any child who receives racemic epinephrine in the emergency department should be admitted, since the effects of racemic epinephrine wear off in 2 hours and the patient may rebound with worsened respiratory distress.[18] If racemic epinephrine is not immediately available, L-epinephrine may be used instead. The dosage equivalent is 5 ml L-epinephrine 1 : 1000.[21]

STEROIDS

The role of corticosteroids in croup is controversial. Despite numerous studies published in the literature the effectiveness of steroids in croup is not known. A recent review in the literature suggests that the controversy arises from methodologic inadequacies in the nine studies analyzed by the authors. The studies fail to define diagnostic criteria adequately for croup, often including spasmodic croup and viral croup together. The dose of steroids, the steroid preparation used and the dosing interval vary between the studies and make comparisons difficult. The choice of outcome event was poorly defined and varies among the studies.[22] Koren et al.[23] compared the effects of dexamethasone and placebo in pediatric patients with viral laryngotrachetitis and spasmodic croup. They found that dexamethosone lowered the respiratory rate of the children with spasmodic croup at 6 hours but did not affect the patients with viral croup. The clinical significance of this study is questioned[24,25] as the difference in respiratory rate while statistically significant was a decrease from 31 breaths/min to 27 breaths/min, and is not clinically important. Steroids have no role in the outpatient management of croup.[5] If steroids are to be given to admitted children, the initial dose should be equivalent to at least 100 mg cortisone.[22]

Disposition

Most children presenting to the emergency department with croup can be managed at home. Ten percent will require hospital admission, and of those admitted 3 percent will require establishment of an artificial airway. The following are disposition guidelines for children with croup.

1. All children with croup score of 8 or greater should be admitted.
2. Most children with croup scores of 6 or 7 should be admitted, especially if the parents are extremely anxious and unable to care for the child properly. The availability of the family to emergency health care should also be considered.
3. Children in a high-risk category and with a history of prematurity, neonatal pulmonary disease, or postintubation should be admitted with lower croup scores.
4. All children who receive racemic epinephrine in the emergency department should be admitted.

SUPRAGLOTTITIS

Management

The child with supraglottitis requires an artificial airway. Observation and antibiotic therapy of a pediatric patient with supraglottitis in mild respiratory distress is not recommended, as this carries a significant morbidity and mortality.[5,6,26,27] The hospital should have a supraglottitis team that is called as soon as epiglottitis is suspected. Composition of the team depends on the resources within the hospital and community. The team might consist of a pediatrician, an otolaryngologist or general surgeon and an anesthsiologist. The hospital should have an supraglottitis protocol agreed upon by the emergency department and the members of the team. The current literature advocates airway management by a nasotracheal tube, with trachestomy only if intubation is not possible.[5,6,26,28] Airway placement should be done under controlled conditions, in the operating room, intensive care unit, or the emergency department. Each institution's protocol should address airway placement and where it is performed.

While awaiting the team's arrival, the child should be kept under constant surveillance by nursing or physician personnel. Equipment for airway maintenance should be readily accessible. The child should be allowed to maintain his position of comfort on parent's lap or near parents. Because the child with supraglottitis may suddenly have a respiratory arrest all procedures that can cause crying or agitation, for example taking blood, starting an intravenous line, giving injections, giving antipyretic medication, undressing the child, restraining the child for a neck radiography or examination of the throat and separation from parents should be avoided until a

stable airway is established.[29,30] Oxygen should be administered in a manner that does not agitate the patient. Racemic epinephrine should not be given to pediatric patients with supraglottitis in an effort to improve the respiratory distress while waiting for placement of a secure airway. A recent report in the literature describes four children with epiglottitis who received racemic epinephrine by nebulization and within 60 minutes of treatment each had a respiratory arrest.[31]

Once the team is assembled, the child, accompanied by team members and airway equipment, should be taken to the operating room or intensive care unit. The child should be transported with the parent, in a wheelchair on the parent's lap for a young child. If the child deteriorates or has a respiratory arrest in the emergency department prior to arrival of the team, airway management should be done by the most skilled person available. It is important to keep in mind that most pediatric patients with supraglottitis can be effectively oxygenated with a bag-valve-mask apparatus.[32,33] If this is unsuccessful, oral intubation should be done using an endotracheal tube 1 to 2 mm smaller than appropriate for the child's size. After the posterior pharynx is visualized with a laryngoscope, gentle compression of the chest, forcing an air bubble out of the glottis may help to identify the glottic opening if the anatomy is distorted. A needle cricothyrotomy can be done if intubation is unsuccessful as a last resort.

Ideally, patients needing transfer to a medical center for pediatric intensive care should have an airway established prior to transfer. The child should then be restrained and sedated during transport. The parent should accompany the child during transport to decrease the child's agitation. Not all hospitals will be able to provide definitive airway management at their institution. If transport times are brief and the child is not in severe distress, transfer via ambulance without an airway may occur. There is significant risk associated with transferring patients without a protected airway.[6] Community hospitals should have an agreement with their referral centers concerning the airway management and transport of children with supraglottitis.

LOWER AIRWAY OBSTRUCTION

Assessment

The three major causes of lower airway obstruction in children are asthma, bronchiolitis, and tracheobronchial foreign bodies. When a young child presents with his first episode of wheezing it is often difficult to determine the etiology.

A history of a choking or coughing episode followed by the abrupt onset of respiratory distress suggests a foreign body aspiration. Of 50 children with tracheobronchial foreign bodies evaluated at Denver Children's Hospital, 75 percent had a history of a choking episode. The classic triad of unilateral wheezing, cough, and decreased breath sounds was present in less than

half of the patients. All 50 children had an abnormal physical examination: decreased breath sounds in 39, wheezing in 38, cough in 18, and a variety of the other findings in 14 children.[34] The chest radiograph of the child with a tracheobronchial foreign body is often normal or the findings subtle or nonspecific. Very few aspirated foreign bodies are radiopaque. Unilateral hyperexpansion, atelectasis, and an infiltrate may be seen after aspiration.[35] Any child who presents with a history or physical examination compatible with a foreign-body aspiration or who has persistent unequal breath sounds after therapy for bronchospasm should have inspiratory/expiratory or decubitus radiographs taken. Decubitus films are often easier to obtain in the uncooperative or young child. Normally, the dependent hemidiaphragm moves in the decubitus position, decreasing the dependent lung size. When a foreign body is present in the dependent lung, the hemidiaphragm is unable to elevate, and the lung cannot empty. The dependent lung appears radiolucent compared with the other lung. The child with a possible aspirated foreign body should be admitted and referred to a subspecialist for bronchoscopy.

Bronchiolitis is a viral infection of the lower distal respiratory tract, most commonly caused by respiratory syncytial virus. It affects children under 2 years of age and tends to occur in epidemics during fall and winter. The virus causes necrosis of the respiratory epithelium, destruction of the ciliated layer and edema of the submucosa and adventitia of the bronchioles. Airway obstruction is caused by the edema, cellular and fibrin debris, and secretions. The contribution of bronchial smooth muscle constriction to obstruction is uncertain.[36] Infants do have the ability to decrease airflow in response to noxious stimuli and to increase flow with bronchodilator therapy.[37] The physiologic manifestations are hyperinflation, decreased lung compliance, and increased airway resistance, resulting in an increased work of breathing. Ventilation-perfusion mismatch occurs because of the patchy pattern of involvement.

Infants with broncholitis usually have a history of a few days of nasal congestion, cough, and fever with a gradual onset of wheezing and respiratory distress. The infants are tachypneic, tachycardic, and coughing, with chest retractions, prolonged expiration, wheezing and rales. Tachypnea of 60 to 100 breaths/min is not unusual as the infant compensates for airway obstruction by increasing his respiratory rate rather than tidal volume. Wheezing may not be a prominent sign because the involved airways are small peripheral bronchioles where airflow is not sufficient to produce turbulent noisy flow. The sicker infants with bronchiolitis have respiratory rates over 60/minute, are anxious or irritable, are unable to take a bottle or suck without frequent interruptions for coughing or choking. They have subcostal and intercostal retractions and as the severity increases, sternal and supraclavicular retractions and nasal flaring occur. Infants in impending respiratory failure are pale and lethargic and have poor air entry.[38] These infants are hypoxic and have CO_2 retention.

More than 50 percent of asthmatic children have the onset of symptoms

in the first two years of life.[37] Because the physiologic manifestations of asthma are similar to bronchiolitis, the presentation and appearance of a young child with asthma may be identical to a child with bronchiolitis. A careful history may help distinguish between the two entities. A history of previous wheezing or bronchitis episodes, other atopic manifestations (such as atopic eczema) and a family history of reactive airway disease all suggest asthma. Fever is not a sign specific for bronchiolitis. Young asthmatic children often have fever and nasal congestion with their bronchospasam because viral respiratory infections are the common initiating events for an asthmatic episode in a young child.

Clinically, it is probably not necessary to distinguish between these two entities. Their initial management is the same. The standard treatment for asthma in the emergency department involves subcutaneous or aerosolized β-agonists. A trial of one of these agents should be administered to all infants and young children with wheezing. Young asthmatics may respond to β-agonist therapy relatively less well than older children and adults. This lesser effect may be explained by the predominence of mucosal edema and increased secretions rather than bronchospasm in the pathophysiology of their asthma.[40,41] The response of infants with bronchiolitis to bronchodilator therapy is unpredictable. Several investigators have failed to show a consistent response to aerosolized and oral bronchodilators.[42–44] If the patient responds by a decrease in respiratory rate, work of breathing, or a decrease in wheezing, the child should receive further therapy in the emergency department and, if discharged, should be continued on bronchodilator therapy.

Assessment of the severity of a child's acute asthmatic episode can be difficult. Patients, parents, and physicians may not agree on the degree of distress the child is experiencing.[45] The objective measurements of severity used in adults such as spirometry and arterial blood gases are often difficult to obtain in children.

Historic factors are useful in assessing severity. The duration of the wheezing episode is important, especially in young children and infants, in whom fatigue can contribute significantly to the development of respiratory failure. The child's current drug therapy should be noted, especially the use of steroids. An asthma attack that develops after a recent decrease in steroid dose should be considered severe.[46] A pediatric patient who is unable to sleep or continue usual activities should be considered to have a serious exacerbation of asthma. A history of a previous intensive care unit admission suggests the potential for a severe attack.

Tachypnea and tachycardia occur with acute asthma. Several studies in adult asthmatics have correlated tachycardia with severity.[47,48] Rees et al.[47] noted that a heart rate greater than 130 beats/min was associated with a PaO_2 value less than 40 mmHg in 83 percent of their patients. Collins et al.[48] reported a mean heart rate of 126 beats per minute in patients with severe asthma. The clinical scoring system developed by Fischl et al.[49] uses a heart rate greater than 120 beats/min as a criterion. A heart rate greater

than 150 beats/min in a child should alert the physician. It may signify prior abuse of β-agonist therapy, hypoxemia, and/or derangements in acid-base status.[50]

The quality of breath sounds and the work of breathing are used to assess severity. Wheezing is not a reliable sign. When obstruction is minimal and airflow great, wheezing is loud. As obstruction increases, wheezing becomes high pitched and of low intensity. The most severe asthmatics have decreased, distant breath sounds with diminished wheezing.[51] As the severity of the attack increases, the child uses the accessory muscles of respiration. In adults, sternocleidomastoid muscle retraction is associated with severe airway obstruction; however, severe obstruction can exist without sternocleidomastoid muscle use.[52]

The presence of pulsus paradoxus correlates well with severity. A fall of more than 10 mmHg in adults is associated with an $FEV_{1.0}$ of less than 40 percent of predicted.[53] Among acutely ill asthmatic children studied by Gluck et al.,[54] all patients with a decrease of 20 mmHg or greater were admitted to the hospital in status asthmaticus.

One study in adult subjects found derangments in patterns of speech to correlate well with severity. Too dyspneic to speak was labeled severe; monosyllabic speech, moderately severe; and frequent pauses, mild. All three groups were found to be severe by spirometric criteria.[46] In an older child this may be a very helpful assessment tool.

A change in mental status usually heralds severe obstruction. The child who is restless or agitated is hypoxemic. As the obstruction progresses with impending respiratory failure, the pediatric patient becomes fatigued, lethargic, and somnolent. This occurs as the child can no longer maintain the increased work of breathing and respiratory acidosis develops.

Clinical scoring systems have been developed to aid in identifying the severely ill child or child with impending respiratory failure. Most of the scoring systems are modeled after the system developed by Woods et al.[55] (Table 12-3). The indices are based on assessment of the child's work of

Table 12-3. A Clinical Asthma Score[a]

Cyanosis (PaO_2)	None (70–100 mmHg)	In air (< 70 mmHg)	In 40% O_2 (< 70 mmHg)
Inspiratory breath sounds	Normal	Unequal	Decreased or absent
Use of accessory muscles	None	Moderate	Maximal
Expiratory wheezing	None	Moderate	Marked
Cerebral function	Normal	Depressed or agitated	Coma

[a] Score: 5 or more suggests impending respiratory failure; 7 or more with PCO_2 65 indicates existing respiratory failure.

(Downes JJ, Raphaely RC: Pediatric intensive care. Anesthesiology 43:328, 1975.)

breathing, quality of breath sounds and mental status. Recently, the ability of these scoring systems to detect significant hypoxemia has been questioned. Hurwitz et al.[56] examined the correlation between the initial clinical score and the initial PaO_2 determined by arterial blood gas analysis. Four of the 37 children who had ABG analysis had PaO_2 less than 60 mmHg. The mean clinical score of these four children was not significantly different from the mean score of the remainder of the children, whose average PaO_2 was greater than 80 mmHg. The authors conclude that the scoring system did not predict the degree of hypoxemia in the pediatric asthma patient and suggest that significant hypoxemia can exist in the absence of clear clinical signs of respiratory distress.

Arterial blood gas analysis is a valuable adjunct to assessing severity of an asthmatic episode. The ABGs in an early asthma attack show a mild decrease in PaO_2 and a low $PaCO_2$ usually less than 35 mmHg. As the obstruction increases the $PaCO_2$ rises toward normal values and the PaO_2 continues to decrease from the worsening ventilation perfusion mismatch. The point where the $PaCO_2$ is normal has been called the crossover point, where the asthmatic patient's compensatory mechanisms are no longer adequate. The sickest asthmatic children will have severe hypoxemia, respiratory acidosis, and often a metabolic acidosis. It is important to use the ABG analysis in conjunction with the clinical assessment of the patient. A normal $PaCO_2$ in a wheezing retracting child is an ominous sign. It needs to be emphasized that most pediatric asthmatic patients will not need arterial blood gases drawn. However, when there is a question concerning the severity of the attack or response to therapy, they may be extremely helpful.

Spirometric measurements provide an objective assessment of severity. They are not routinely obtained in most acute pediatric asthma patients. Spirometry cannot be performed by infants or young children. An acute episode is also not the optimum time to teach an anxious child how to use a spirometer. The severely ill child may be too dyspneic to accomplish a full expiratory maneuver.

Chest radiographs are often obtained in the acutely wheezing child. The information obtained from this study rarely alters the emergency treatment of the patient. The frequently found abnormal findings, hyperinflation, atelectasis, and peribronchial cuffing do not alter the management. The appearance of the chest radiograph does not correlate with the severity of the asthma attack.[57] Chest radiography should be delayed until after therapy is instituted, unless a pneumomediastinum or pneumothorax is suspected. A radiograph should be taken if there is a poor response to therapy.

Fatal outcomes in asthmatics do occur. A significant number of childhood asthmatic deaths are sudden and unexpected, often without time to seek medical attention. Because the large majority of asthmatic episodes in children will resolve uneventfully irrespective of the therapy they receive it is easy to become complacent in treating asthma. The possibility of respiratory failure should always be kept in mind. Although severe hypoxemia ($PaO_2 < 50$ mmHg) or hypercapnia ($PaCO_2 > 50$ mgHg) clearly define respi-

ratory failure, findings that should lead one to suspect its development at an earlier stage include (1) faint or absent breath sounds, (2) a normal arterial $PaCO_2$ in the face of strenuous respiratory effort, (3) appearance of fatigue, (4) history of previous respiratory failure, and (5) failure to respond to therapy promptly.[50]

MANAGEMENT

Oxygen

Oxygen should be given generously to acutely ill pediatric asthma patients. The patient with minimal airway obstruction has a mildly decreased PaO_2. As the obstruction worsens, the PaO_2 continues to decline. β-Agonist therapy administered to the child may increase his hypoxemia by worsening the ventilation perfusion mismatch. Patients should receive their nebulization therapy with oxygen.

Fluids

Many asthmatic children presenting to the emergency department are mildly dehydrated. This results from poor oral intake secondary to their dyspnea, from vomiting and increased respiratory losses. Most children's fluid requirements will be met if they are given $1\frac{1}{2}$ times daily maintenance requirements. There is no evidence to suggest that large amounts of intravenous fluids aid in liquefying secretions. Overhydration may predispose the child to pulmonary edema or exacerbation of the syndrome of inappropriate antidiuretic hormone secretion.[51]

β-Agonist Agents

β-Adrenergic drugs are the primary therapeutic agents for an acute asthmatic attack. Epinephrine, given subcutaneously, has been the first-line treatment for more than 40 years in the United States. The standard regimen was two or three subcutaneous injections of epinephrine twenty minutes apart followed by Susphrine. This regimen has not been found to produce greater bronchodilation than Susphrine alone. Ben-Zvi et al.[58] concluded that repeated injections of epinephrine sustain the improvement provided by the initial injection but do not add to its action. Susphrine provided a pattern of response (as measured by spirometry) similar to multiple injections of epinephrine. If the clinical situation warrants epinephrine, one dose of Susphrine should be given instead of multiple doses of epinephrine.

With the introduction of more selective inhaled β_2-agents into the United States, the use of epinephrine, which has both α- and β-activity has declined. The inhaled beta agonists offer several advantages over epineph-

Table 12-4. Clinical Pharmacology of the β-Adrenergic Aerosols

Agent	Relative Potency[a]	Duration (h)	β-Selectivity
Isoproterenol	4	1–2	$\beta_2 = \beta_1$
Isoetharine	2	2–3	$\beta_2 \geq \beta_1$
Metaproterenol	3	3–4	$\beta_2 > \beta_1$
Terbutaline	4	4–6	$\beta_2 \gg \beta_1$
Albuterol	4	4–6	$\beta_2 \gg \beta_1$
Fenoterol	4	4–6	$\beta_2 \gg \beta_1$

[a] The highest potency is equal to 4.

rine therapy. Nebulization of the drug delivers it to its site of action, thereby decreasing systemic absorption and side effects. The prolonged duration of the aerosol treatment may permit delivery of the drug into parts of the airway that were initially obstructed. Multiple studies have shown that inhaled β-agonists are at least as effective as, if not more effective than, subcutaneously administered epinephrine, but with fewer side effects.[59–63] Severe airway obstruction does not alter the response to these inhaled agents compared with the response to injected agents.[62,64] Nebulization therapy offers a psychological benefit to the young child who is scared and in respiratory distress. The avoidance of painful injections may make the child's experience in the emergency department less threatening and frightening and lessen respiratory distress.

Several β-agonist aerosols are used in this country. They differ in their potency, β_2-specificity, onset and duration of action (Table 12-4). Albuterol has been widely used in Canada and Europe for several years. It has proved effacacious and safe, with minimal side effects.[59,65] Albuterol aerosol solution is now available in the United States. It may well replace the other β-adrenergic agents and become the treatment of choice for acute asthma in the pediatric patient.

The β-adrenergic aerosols should be administered by oxygen-powered nebulizers. Most older children can use a hand-held mouthpiece to administer the aerosol. In younger children or infants, the inhalation can be given with a face mask held close to the face. Some children become very agitated and upset by the face mask. The aerosol may be successfully given in these children by using a paper or foam cup instead of the face mask (see Fig. 12-3). If the child continues to cry, increasing respiratory distress, the aerosol should be stopped and a subcutaneous β-agonist given. Oxygen flow of at least 6 L/min, and a chamber volume of 4 ml provides optimum particle size and delivers the aerosol over 5 to 10 minutes. With the best technique only 12 percent of the nebulized solution is deposited in the lungs and 20 to 40 percent of the solution remains in the nebulizer.[66] The β_2-specific aerosols have a wide margin of safety; thus, precise dosing is not critical. The amount of drug a child receives is based on the tidal volume and respiratory rate, which are related to the child's size. The dosage schedules recommended for the β_2-agents are based on age rather than weight[67] (Table 12-

Table 12-5. Dosing and administration guidelines for nebulized β-$_2$ agents

Drug	Dose (ml)
Metaproterenol (5% inhalant solution, 50 mg/ml)	
2 yr	0.1
2–9 yr	0.2
9 yr	0.3
Terbutaline (0.1% parenteral ampule, 1 mg/ml)	
2 yr	0.5
2–9 yr	1.0
9 yr	1.5
Albuterol (salbutamol) (0.5% respirator solution, 5 mg/ml)	
2 yr	0.2
2–9 yr	0.4
9 yr	0.6
Fenoterol (0.5% respirator solution, 5 mg/ml)	
2 yr	0.1
2–9 yr	0.2
9 yr	0.3

(Bolte RG: Nebulized β-adrenergic agents in the treatment of acute pediatric asthma. Pediatr Emerg Care 2:250, 1986.)

5). The β_2-agonists can be given every twenty minutes for a total of three or four treatments, depending on clinical response and heart rate. Spirometric studies show that this shorter dosing interval provides for a smooth increase in forced expiratory volume in 1 second (FEV_1) and achieves an earlier peak response which is maintained.[65]

Theophylline

The role of theophylline in the initial management of an acute asthmatic episode is in question. Intravenous aminophylline should be given to the asthmatic child who has failed to improve enough for discharge from the emergency department after adequate treatment with β-adrenergic agents and who has a subtherapeutic theophylline level. Oral theophylline or intravenous aminophylline have not been shown to improve upon the bronchodilation achieved in the emergency department from potent nebulized β-agonist therapy.[68–71] If a child has failed to respond to β-adrenergic therapy sufficient enough to be sent home, hospital admission should be arranged and intravenous aminophylline given. Aminophylline should be given as a loading dose of 6 to 7 mg/kg over a 20- to 30-minute period. The full loading dose should be given to children who are not currently taking a theophylline preparation, who have not received a short-acting preparation within 6

Table 12-6. Total Daily Theophylline Doses

Age	Dose mg/kg/kg
6–12 mo	[0.2 (age in wks) + 5]
1–9 yr	20
9–12 yr	16
12–16 yr[a]	16
12–16 yr[b]	13

[a] Smoker.
[b] Nonsmoker.

hours or a long-acting preparation within 12 to 18 hours. If the patient has received theophylline in the recent past, the loading dose should be decreased to 2 to 3 mg/kg. A theophylline level should be checked prior to giving theophylline to any patient in whom the dose, time of last dose, or medication name is uncertain. Once the child is fully loaded an intravenous continuous infusion should be started at a rate of 0.7 to 1.1 mg/kg/hr, via a rate minder. The dose should be adjusted based on the patient's age, concurrent medications and preexisting chronic illnesses (Table 12-6).

Steroids

The contribution of corticosteroids in the management of acute asthma is controversial. Various authorities have found them beneficial,[71–73] while others have not.[9] The precise mechanism of action is unknown. A portion of its effect is probably due to potentiation of β-adrenergic stimulation. Pierson et al.[73] found that steroids administered to children in status asthmaticus produced a more rapid recovery from their arterial hypoxemia. An additional effect was not found on airway obstruction. Other investigators have not found a improvement in oxygenation but have documented an additional improvement in airway obstruction greater than that produced by β-adrenergic agents alone.[71,72] Until recently, steroids were not thought to play an important role in management of the asthmatic in the emergency department because their onset of action was felt to be several hours to a day.[72,73] Littenberg and Gluck[71] administered methylprednisolone in a double-blind placebo-controlled study to acutely ill asthmatic adults within 30 minutes of their arrival in the emergency department. The patients then received standard emergency treatments for asthma. After an average stay of 4 hours for both groups, 81 percent of patients in the steroid-treated group were discharged, as compared with 53 percent in the placebo-treated group. The patients receiving steroids had improved scores on subjective symptom assessment. Littenberg and Gluck[71] conclude that "prompt use of intravenous methylprednisolone in the emergency care of acute asthma can help to terminate the attack, to alleviate symptoms, and to reduce the need for hospitalization."[11] Steroids should be given to every asthmatic child who is

hospitalized.[74] There is no agreement on which steroid, how much or how often it should be given. One to 2 mg/kg IV methylprednisolone can be given safely in the emergency room.

Disposition

Early identification of pediatric asthma patients requiring hospital admission has several potential advantages. Patients at high risk of admission could initially be treated more vigorously, perhaps reducing the length or eliminating the need for hospitalization. Delays in admitting patients could be reduced. Several studies have attempted to identify factors that would predict the need for hospitalization of asthmatics early in their emergency department course.[75–77] The two pediatric studies used initial spirometric measurements, clinical asthma scores and historical factors to identify the children that need admission.[74,75] Fischl et al.[76] used an index for adults based on pulse rate, respiratory rate, pulsus paradoxus, peak expiratory flow, dyspnea, use of accessory muscles and wheezing. Scores to predict admission have had poor success in prospective studies.[75,77] A reliable valid scoring system to predict hospital admission needs to be developed.

A few admission guidelines do exist: (1) all children who remain in respiratory distress after three or four treatments with beta agonist agents should be admitted; (2) any child who repeatedly vomits asthma medications probably needs admission; (3) a child receiving emergency treatment in the preceding 24 hours should be considered a strong candidate for admission; and (4) a low threshold for admission should exist for wheezing infants in high-risk categories (congenital heart disease, bronchopulmonary dysplasia, neuromuscular disease and cystic fibrosis).

The goal of treatment in an acute exacerbation of asthma is not complete relief of airway obstruction. Most patients are discharged with mild to moderate obstruction.[14] Because significant small airway obstruction may remain for days after an acute episode, patients need to be discharged on medication for several days. The child on a maintenance regimen who has an acute exacerbation needs a therapeutic change either to ensure compliance with current therapy, a dosage adjustment, or additional therapy. Pediatric patients currently not taking asthma medications should be started on therapy.

Discharge medications may include oral or inhaled β_2-agents, theophylline, or steroids. The preparation used depends on the age of the child, current therapy, and past medication intolerance for failures. For patients discharged on a theophylline preparation, an attempt should be made to obtain a therapeutic level (10 to 20 μg/ml) prior to discharge either by administering a loading dose of an oral short-acting theophylline or intravenous aminophylline. The loading dose should be based on the child's previous theophylline doses and current level. A loading dose is especially important for children discharged on sustained-release theophylline prepa-

rations. Theophylline should be used cautiously in infants less than 12 months because of their variable clearance rates.

Oral β-adrenergic drugs such as metaproterenol or albuterol are as effective as theophylline in controlling asthma.[78] β-Agonists can be used as single-agent therapy or in conjunction with theophylline. They are well tolerated with fewer side effects than theophylline preparations. Inhaled β-agonists can be used with success in children older than 5 to 6 years of age.

REFERENCES

1. Thompson AE: Respiratory distress. p. 230. In Fleisher G, Ludwig S (eds): Textbook of Pediatric Emergency Medicine. Williams & Wilkins, Baltimore, 1983
2. Barness LA: Pediatric Physical Diagnosis. Yearbook Medical Publishers, Chicago, 1976
3. Fireman P: The Wheezing Infant. Pediatr Rev 7:247, 1986
4. Gay BB, Atkinson GO, Vanderzalm T: Subglottic foreign bodies in pediatric patients. Am J Dis Child 140:165, 1986
5. Hen J: Current management of upper airway obstruction. Pediatr Ann 15:274, 1986
6. Barker GA: Current management of croup and epiglottitis. Pediatr Clin North Am 26:565, 1979
7. Singer JI, McCabe JB: Epiglottitis at the extremes of age.
8. Welch DB, Price DG: Acute epiglottitis and severe croup. Anesthesia 38:754, 1983
9. Mills JL, Spackman TJ, Borns P, et al: The usefulness of lateral neck roentgenograms in laryngotracheobronchitis. Am J Dis Child 133:1140, 1979
10. Podgore JK, Bass JW: The "thumb sign" and "little finger sign" in acute epiglottitis. J Pediatr 1:1354, 1976
11. Outwater KM: Diagnosis and management of acute epiglottitis in children. Crit Care Med 14:995, 1986
12. Diaz JH: Croup and epiglottitis in children: The anesthesiologist as diagnostician. Anesth Analg 64:621, 1985
13. Couriel J: Acute stridor in the preschool child. Br Med J 288:162, 1984
14. Goldhagen JL: Croup: Pathogenesis and management. J Emerg Med 1:3, 1983
15. Downes J, Raphaely R: Pediatric intensive care. Anesthesiology 43:242, 1975
16. Ellis EF, Taylor JC, Lefkowitz MS: Racemic epinephrine in croup. Pediatrics 53:291, 1974
17. Lenny W, Milner AD: Treatment of acute viral croup. Arch Dis Child 53:704, 1978
18. Taussig LM, Castro O, Beaudry PH: Treatment of laryngotracheobronchitis (croup). Use of intermittent positive pressure breathing and racemic epinephrine. Am J Dis Child 129:790, 1975
19. Wesley CR, Cotton EK, Brooks JG: Nebulized racemic epinephrine by IPPB for the treatment of croup. A double blind study. Am J Dis Child 132:484, 1978
20. Fogel JM, Berg IJ, Gerber MA, Sherter CB: Racemic epinephrine in the treatment of croup: Nebulization alone versus nebulization with intermittent positive pressure breathing. J Pediatr 101:1028, 1982

21. Remington S, Meakin G: Nebulized adrenaline 1 : 1000 in the treatment of croup. Anesthesia 41:923, 1986
22. Tunnessen WW, Feinstein AR: The steroid-croup controversy: An analytic review of methodologic problems. J Pediatr 96:751, 1980
23. Koren G, Frand M, Barzilay Z, MacLeod SM: Corticosteroid treatment of laryngotracheitis v spasmodic croup in children. Am J Dis Child 137:941, 1983
24. Lacroix J, Massicotte P: Corticosteroid treatment of croup. Am J Dis Child 138:699, 1984
25. Orr ST, Caplan SE: Laryngotracheitis and croup. Am J Dis Child 138:991, 1984
26. Clark WD, Bailey BJ, Clegg TJ: Epiglottitis and laryngotracheobronchitis. Am Fam Pract 28:189, 1983
27. Selbst SM: Epiglottitis: A review of 13 cases and a suggested protocol for management. J Fam Pract 19:333, 1984
28. Vernon DD, Carnaik AP: Acute epiglottitis in children: A conservative approach to diagnosis and management. Crit Care Med 14:23, 1986
29. Tarnow-Mordi WO, Berrill AM, Darby CW, et al: Precipitation of laryngeal obstruction in acute epiglottitis. Br Med J 290:629, 1985
30. Bass JW, Fajardo JE, Brien JH: Sudden death due to epiglottitis. Pediatr Infect Dis 4:447, 1985
31. Kissoon N, Mitchell I: Adverse effects of racemic epinephrine in epiglottitis. Pediatr Emerg Care 1:143, 1985
32. Szold PD, Glicklich M: Children with epiglottitis can be bagged. Clin Pediatr 15:792, 1976
33. Sweeney DB, Allen TH, Steven, IM: Acute epiglottitis: management by intubation. Anesth Intens Care 1:526, 1973
34. Janik JS, Burrington JD, Wayne ER et al: Foreign body aspiration in children. Colo Med 10, 1986
35. Cotton EC, Yasuda K: Foreign body aspiration. Pediatr Clin North Am 31:937, 1984
36. Wohl ME, Chernick V: Bronchiolitis. Am Rev Respir Dis 118:759, 1978
37. Eigen H: Clinical Pharmacology of the wheezing infant. Pediatr Ann 14:325, 1985
38. Wohl ME: Bronchiolitis. Pediatr Ann 15:307, 1986
39. Consensus Statement Section on Allergy and Immunology, American Academy of Pediatrics: Management of Asthma. Pediatrics 68:874, 1981
40. Lenny W, Milner D: At what age do bronchdilators work? Arch Dis Child 53:532, 1978
41. Radford M: Effect of salbutamol in infants with wheezy bronchitis. Arch Dis Child 50:535, 1975
42. Spier S, LaPierre JG, Lamarre A: Response to salbutamol during a first or second episode of wheezing in infancy. Am Rev Respir Dis 131:A259, 1985
43. Phalen PD, Williams HE: Sympathomimetic drugs in acute viral bronchiolitis: their effect on pulmonary resistance. Pediatrics 44:493, 1969
44. Brooks LJ, Cropp GJ: Theophylline therapy in bronchiolitis. Am J Dis Child 135:934, 1981
45. Rebuck AS, Braude AC, Chapman KR: Evaluation of the severity of the acute asthmatic attack. Chest 82(suppl I):285, 1982
46. Corre KA, Rothstein RJ: Assessing severity of adult asthma and need for hospitalization. Ann Emerg Med 14:45, 1985

47. Rees HA, Millar JS, Donald KW: A study of the clinical course and arterial blood gas tensions of patients in status asthmaticus. Q J Med 37:541, 1967
48. Collins JV, Clark TJ, Brown D, et al: The use of corticosteroids in the treatment of acute asthma. Q J Med 64:259, 1975
49. Venturelli J: Acute epiglottitis in children. Crit Care Med 15:86, 1987
50. Leffert F: The management of acute severe asthma. J Pediatr 96:1, 1980
51. Stempel DA, Mellon M: Management of acute severe asthma. Pediatr Clin North Am 31:879, 1984
52. Baggs PB, Stephens AL, Bhat KD, Vekovius WA: A classification system for asthmatic patients: Clinical-physiological correlation. Ann Allergy 47:307, 1981
53. Rebuck AS, Read J: Assessment and management of severe asthma. Am J Med 51:788, 1971
54. Gluck JC, Busto R, Marks MB: Pulsus paradoxus in childhood asthma—Its prognostic value. Ann Allergy 38:405, 1977
55. Wood DW, Downes JJ, Lecks HI: A clinical scoring system for the diagnosis of respiratory failure. Am J Dis Child 123:227, 1972
56. Hurwitz ME, Burney RE, Howatt WF, et al: Clinical scoring does not accurately assess hypoxemia in pediatric asthma patients. Ann Emerg Med 13:1040, 1984
57. Eggleston PA, Ward BH, Pierson WE, Bierman CW: Radiographic abnormalities in acute asthma in children. Pediatrics 54:442, 1974
58. Ben-Zvi Z, Lam C, Spohn WA, et al: An evaluation of repeated injections of epinephrine for the initial treatment of acute asthma. Am Rev Respir Dis 127:101, 1983
59. Turpeinen M, Kwokkanen J, Backman A: Adrenaline and nebulized salbutamol in acute asthma. Arch Dis Child 59:666, 1984
60. Baughman RP, Ploysongsang Y, James W: A comparative study of aerosolized terbutaline and subcutaneously administered epinephrine in the treatment of acute bronchial asthma. Ann Allergy 53:131, 1984
61. Tinkelman DG, Vanderpool GE, Carroll MS, et al: Comparison of nebulized terbutaline (terb) and subcutaneous epinephrine (epi) in the treatment of acute asthma. Ann Allergy 50:398, 1983
62. Ben-Zvi Z, Lam C, Hoffman J, et al: An evaluation of the initial treatment of acute asthma. Pediatrics 70:348, 1982
63. Schwartz AL, Lipton JM, Warburton D, et al: Management of acute asthma in childhood: a randomized evaluation of β-adrenergic agents. Am J Dis Child 134:474, 1980
64. Fanta CH, Rossing TH, McFadden ER: Treatment of acute asthma. Am J Med 80:5, 1986
65. Robertson CF, Smith F, Beck R, Levison H: Response to frequent low doses of nebulized salbutamol in acute asthma. J Pediatr 106:672, 1985
66. Reiser J, Warner JO: Inhalation treatment for asthma. Arch Dis Child 61:88, 1986
67. Bolte RG: Nebulized β-adrenergic agents in the treatment of acute pediatric asthma. Pediatr Emerg Care 2:250, 1986
68. Siegel D, Sheppard D, Gelb A, Weinberg PF: Aminophylline increases the toxicity but not the efficacy of an inhaled beta-adrenergic agonist in the treatment of acute exacerbations of asthma. Am Rev Respir Dis 132:283, 1985
69. Rossing TH, Fanta CH, Goldstein DH, et al: Emergency therapy of asthma: Comparison of the acute effects of parenteral and inhaled sympathominietics and infused aminophylline. Am Rev Respir Dis 122:365, 1980

70. Fanta CH, Rossing TH, McFadden ER: Emergency room treatment of asthma. Am J Med 72:416, 1982
71. Littenberg B, Gluck EH: A controlled trial of methylprednisolone in the emergency treatment of acute asthma. N Engl J Med 314:150, 1986
72. Fanta CH, Rossing TH, McFadden ER: Glucocorticoids in acute asthma. Am J Med 74:845, 1983
73. Pierson WE, Bierman CW, Kelley, VC: A double-blind trial of corticosteroid therapy in status asthmaticus. Pediatrics 54:282, 1974
74. Silver RB, Ginsburg CM: Early prediction of the need for hospitalization in children with acute asthma. Clin Pediatr 23:81, 1984
75. Ownby DR, Abaruza J, Anderson, JA: Attempting to predict hospital admission in acute asthma. Am J Dis Child 138:1062, 1984
76. Fischl MA, Pitchenik A, Gardner LB: An index predicting relapse and need for hospitalization in patients with acute bronchial asthma. N Engl J Med 305:733, 1981
77. Rose CC, Murphy JG, Schwartz JS: Performance of an index predicting the response of patients with acute bronchial asthma to intensive emergency department treatment. N Engl J Med 310:573, 1984
78. Schuller DE, Oppenheimer PJ: A comparison of metaproterenol and theophylline for control of childhood asthma. Clin Pediatr 21:135, 1982

13 Problems in the Management of Intoxications

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The 1985 annual report of the American Association of Poison Control Centers[1] has once again defined the spectrum of human toxic exposures relative to age as a variable. Sixty-three percent of reported exposures occurred in children below the age of 5 years; 8.3 percent in children under 1 year old of age, and 55 percent in the 1- to 5-year-old group. This is distinctly different from the 5.2 and 4.1 percent incidence in the 6- to 12- and 13- to 17-year-old groups, respectively. Confirming what we already know to be true, 72.6 percent of toxic exposures occurred in children and adolescents under 17 years of age. Ninety percent of the exposures were termed accidental, 93.3 percent involved exposure to only one substance, and in 79.2 percent of cases the product was ingested. It is clear that most of these toxic dilemmas appear within the pediatric age group. Herein lies our basic problem.

A clinician, comfortable with management of toxic exposures in adults, may make some serious errors in deduction when translating the diagnostic, prognostic, and therapeutic management guidelines from adult to pediatric intoxications. Failure to appreciate the inherent differences between these two groups of patients may result in either an underaggressive or overaggressive approach to a potentially toxic situation. The presence of age-dependent variables may influence the pharmacokinetic or pharmaco-

dynamic handling of the toxin. This is further complicated by the variability and unpredictability in the maturation process within the pediatric age groups. Indeed, children may be more sensitive or more resistant to toxic effects when compared on a milligram-per-kilogram basis with their adult counterparts. The differences in the handling of toxic substances may dictate a more aggressive or lenient approach to the management of the poisoned pediatric patient.

FAILURE TO APPRECIATE DIFFERENCES BETWEEN PEDIATRIC AND ADULT TOXIC EXPOSURES

Differences in Pharmacokinetic Parameters

Failure to recognize inherent age-related variability in the pharmacokinetic and pharmacodynamic handling of compounds may lead to serious errors in judgment when attempting to predict the toxic potential of an exposure. It is beyond the scope of this chapter to discuss the pharmacokinetic and toxicokinetic parameters characterizing toxic exposures in detail. Highlights of the age-related changes in the pharmacokinetic handling of toxins are presented to familiarize the nonpediatric-trained physician with certain parameters which may explain differences in clinical presentation, as well as the decision process needed in dealing with pediatric poisonings or overdoses.

Age-related modification in the absorption, distribution, metabolism, or excretion (ADME) of a compound will influence the pharmacokinetic handling of a drug or toxin. These alterations in the ADME appear to be most marked in the newborn, with a progressive trend toward the normal adult situation.[2] In addition to the magnification of toxic effects seen with certain compounds in these age groups, other entities may demonstrate an age-related protective effect (e.g., the sulfate route for conjugation of acetaminophen). Pediatric patients demonstrate shifts in elimination rate kinetics due to age-related differences in the amount of enzyme stores, enzyme activity, and hepatic and/or renal maturity.

Factors known to influence the absorption of an orally ingested compound include variations in gastric pH and emptying time, intestinal pH and motility, absorptive surface area, biliary functioning, cell membrane permeability, and gut flora.[3] Gastrointestinal (GI) absorption is influenced in the first few weeks of life by a relative achlorhydria, delayed gastric emptying time, and irregular and unpredictable intestinal mobility.[4] Absorption of acidic drugs may be decreased. Adult values for gastric acidity are usually not reached until 2 to 3 years of age.[5,6] Gastric emptying time may be as long as 6 to 8 hours in the newborn and may not approach adult values until 6 months of age.[7] Both pH and motility are also greatly influenced by individual diet and feeding patterns.[7] The bioavailability of orally ingested compounds exhibiting high hepatic clearances may also display

great interindividual variability, consistent with alterations in the hepatic first-pass metabolism (e.g., propranolol, propoxyphene).[2,3,7]

Percutaneous absorption of certain compounds, such as those related to hexachlorophene, analine dyes, naphthalene, pentachlorophenol, boric acid, potent corticosteroids, and phenylmercury diaper rinses, are known to produce intoxication in infants.[4,6] There is increased potential for dermal absorption in infants as compared with adults, apparently due to the presence of an immature stratum corneum, and a thinner more well-hydrated epidermis.

The distribution characteristics of a potential toxin can be markedly affected by changes in the proportion of body water, fat, protein and tissue binding, circulatory factors, blood-brain barrier development, and other concurrent physiologic and pathologic conditions.[2,3] Since drugs are distributed between extracellular water and fat according to the lipid/water partition coefficient, the relative variability of these parameters can alter the distribution characteristics, hence the concentration found in the plasma and tissue compartments. Throughout infancy and childhood there is a continual change in the body's water compartment with age.[4,6,8] Blood flow, tissue mass, and fat content will determine how much of the drug actually reaches the tissue receptor sites. These receptors in turn may vary with age with respect to sensitivity and binding affinity characteristics. A relative change in the amount of tissue mass, especially lymphatic and renal tissue, which occurs throughout childhood may further alter distribution characteristics.[4]

Total body water may be responsible for as much as 85 percent of premature infant weight and 70 to 75 percent of full term infant weight, while accounting for only 55 percent of adult body weight.[6,8] Intracellular water remains fairly constant throughout life. The most dramatic changes appear in the postnatal and infancy periods, with 50 percent of body weight in premature newborns accounting for extracellular water, 35 percent in the 4 to 6-month-old, 25 percent in the 1-year-old, and only 20 percent in adult life,[6] which is reached at approximately 13 to 15 years of age.[6] Lower plasma levels of water-soluble compounds occur when comparing doses on a milligram-per-kilogram basis. Conversely, the fraction of total body weight present as fat increases with age; therefore, the volume of distribution for lipid-soluble drugs is smaller in the neonate than in the adult.[4]

Infants and young children will display reduced plasma protein binding as compared with their adult counterparts; therefore more free drug will be available for distribution and interaction with tissue receptors. In addition to the reduced plasma protein concentration, the serum albumin is qualitatively different, exhibiting a lower binding capacity for acidic drugs.[9–11] This characteristic results in an increased free fraction of drug (unbound) with an enhanced potential for toxicity. The binding of basic drugs to α-1-glycoprotein also exhibits age-related effects. Adult values are usually not reached until 7 to 12 years of age.[2]

The development of the blood-brain barrier is incomplete in newborns, resulting in increased permeability for certain substances into the brain (e.g., lipid soluble drugs).[6] This may make the infant or young child more sensitive to direct central nervous system (CNS) effects than the older child or adult.

Metabolic degradation in humans enhances the water solubility of a compound to make it more easily excretable. Apparently, the rate of oxidative metabolism (hydroxylation) and glucoronidation is often reduced in newborns, resulting in persistent drug- or toxin-related effects of compounds metabolized via this pathway,[3,4] while sulfate conjugation and demethylation seem to proceed at adult levels.[4,6] Decreased oxidation in newborns is characteristic for acetaminophen, phenobarbital, phenytoin, diazepam, lidocaine, and nortriptyline, while immaturity of the glucuronidation pathway may influence the kinetic handling of chloramphenicol as well as salicylic and nalidixic acids. After 1 month of age, there is a dramatic increase in the enzyme efficiency, approaching adult levels at approximately 1 year of age. Differences in acetaminophen metabolism in children under 9 to 12 years may be responsible for the relative hepatotolerance seen in this age group.

Pharmacogenetic differences, such as glucose 6-phosphate dehydrogenase deficiency, as well as genetic differences in oxidation, acetylation, and pseudocholinesterase activity, also play a role in the characteristics elicited by certain drugs or toxins in different segments of the population.[4]

Drug or toxin elimination occurs by either the excretion of metabolites of the parent compound (active or nonactive) or by elimination of the unchanged parent product. Developmentally, renal function is incomplete at birth, with glomerular filtration rates for newborns 35 to 50 percent of that of older children and adults (newborn 10 ml/min/m^2 versus 70 ml/min/m^2 for adults.[2,4] Tubular function, both secretory and absorptive, is decreased slightly by adult standards. Renal function comparable to that of adults is not achieved until approximately 6 months to 1 year of age.[12] Newborn renal plasma flow is only 20 to 40 percent of adult values, improving quickly by 4 weeks of age. Full adult capacity may not be realized until 12 months of age.[4]

Recognition of Normal Values in Children

One of the preventable pitfalls associated with detecting toxicity in the pediatric age group is related to changes in vital signs and basic physical examination parameters. It is imperative that the nonpediatrician taking charge of the poisoned or overdosed child be aware of the normal values in relation to these patients in order to appropriately detect, assess, and diagnose the toxic syndromes. Table 13-1 lists the appropriate normal values for just that purpose.

Table 13-1. Normal Values for Children

Age	Weight (kg)	Pulse (per min)	Systolic BP (mmHg)	Respiratory Rate (per min)
NB	3.5	94–175	60 +/– 10	30–60
1 mo	4.0	100–190	80 +/– 10	30–60
6 mo	7.0	111–179	90 +/– 30	24–30
1–2 yr	10–12	98–163	96 +/– 30	20–24
3–5 yr	14–18	65–132	99 +/– 25	16–22
6–9 yr	20–28	70–115	105 +/– 13	14–20
10–12 yr	32–34	50–110	112 +/– 19	12–20
12–14 yr	34–40	50–105	120 +/– 20	12–20
15–16 yr	50	50–100	120 +/– 20	12–20
> 16 yr	60–70	50–100	120 +/– 20	10–20

Parameter	Neonate/Infant	Child
Central venous pressure	10 cm H_2O	20 cm H_2O
Pulmonary capillary wedge	3–6 mmHg	6–12 mmHg

FAILURE TO RECOGNIZE THE DIFFERENCES IN THE ASSESSMENT OF A POISONING OR DRUG OVERDOSE IN CHILDREN

History

The spectrum of incidence and etiologic reasons behind pediatric poisonings and drug overdoses is different from those encountered in adults. Therefore, a different approach to the history is warranted in this age group. A recent review of pediatric exposures admitted to a children's hospital confirmed that history is the most important indicator of poisoning.[13] History may be more accurate with accidental ingestions because it often occurs under observation. However, as with most unknown ingestions, a precise history is often unobtainable or unreliable. It should be common practice to ask for the specific container in question. This is important, since toxic and nontoxic products may share the same phonetic name, product formulations may be changed over time by the manufacturer, and certain product lines have numerous varieties of the same product, with small changes in their ingredients. One should not accept patient's or parent's word as gospel concerning the product ingested; it is important to insist on reading the ingredients for oneself. In evaluating the information on the manufacturers label, one must remember that these labels are notoriously inaccurate on first-aid advice for poisoning.

During the initial contact with the poisoned patient, assessment of the severity can be made with the help of other experienced health care practitioners, such as those found in a regional poison control center. Symptoms should be assessed first. If present, it mandates a medical examination (with

the possible exception of vomiting). It may not be necessary for the child to be brought into the emergency department or, for that matter, even treated. Seventy-five percent of human exposures reported to the American Association of Poison Control Centers data collection system were treated in a non-health care facility.[1] The following information should be obtained to permit accurate assessment of whether the victim is in immediate or potential danger or is in no danger at all:

1. Demographic data and source of the exposure
2. Symptomatology present with an assessment of severity
3. The nature of the toxin as assessed by the labeled ingredients, place and date of purchase, name of the company or person who purchased, obtained, or used the product; color, odor, and intended use of the product
4. What other medications or toxic products are available in the environment
5. Time of or since exposure
6. The amount involved (How many tablets or milliliters are left? How many/much was initially in the container? How much was spilled on floor or clothes?
7. Actions taken prior to presentation to the emergency department
8. Assessment of the reliability of informant
9. Assessment of intent: accidental, therapeutic overdose, or suicide attempt

The literature derived from poison control center experience suggests that "accidental" ingestions in children over the age of 5 years are often not truly accidental but rather intentional, requiring investigation into the motivational aspects for the act. This type of event may actually be a "cry for help" indicating an intolerable home situation.[14] Psychological factors may also play a role in children even younger than 5 years of age.[15] "Accidental" ingestion in children under 1 year of age warrants investigation, since it is highly unlikely that a child under 1 year of age would possess the developmental skills necessary to accomplish an accidental ingestion. The "chemical maltreatment" of young children appears to be much more prevalent than was previously believed.[16] Therefore, "accidental" ingestions, whether toxic or nontoxic, should be investigated under the following circumstances:

1. The ingestion occurs in a child over the age of 5 years
2. There is an occurrence of more than one episode of "accidental ingestion"
3. Bizarre clinical manifestations are apparent (possibly Munchausen by proxy syndrome)
4. The "accidental" ingestion occurs in a child under 1 year of age

It is distressing to think of poisoning as a form of child abuse but, for the aforementioned reasons, necropsy in child abuse cases should always include toxicologic analysis.

Table 13-2. Nontoxic Ingestions

Adhesives
Antacids
Antibiotics
Baby product cosmetics
Ballpoint pen inks
Bathtub floating toys
Batteries (flashlight, if bitten)
Bath oil (castor oil and perfume)
Bleach (< 5% sodium hypochlorite)
Body conditioners
Bubble bath soaps (detergents)
Calamine lotion
Candles (beeswax or paraffin)
Caps, toy (potassium chlorate)
Chalk (calcium carbonate)
Cigarettes or cigars
Clay (modeling)
Colognes
Contraceptive pills
Corticosteroids
Crayons (marked A.P., C. P., C.S. 130–46)
Dehumidifying packets (silica)
Detergents (phosphate/anionic)
Deodorants
Deodorizers (spray/refrigerator)
Elmer's glue
Etch-A-Sketch
Eye make-up
Fabric softeners
Fertilizer (with no caution label)
Fish bowl additives
Fluoride, caries preventative
Glues and pastes
Golf ball (possible mechanical injury)
Grease
Hair products (dyes/sprays/tonic)
Hand lotions and creams
Hydrogen peroxide (3%)
Indelible markers
Ink (blue, black)
Iodophor disinfectant
Laxatives
Lipstick
Lubricant
Lubricating oils
Lysol brand disinfectant (not toilet cleaner)
Magic markers
Make-up (eye, liquid facial)
Matches
Mineral oil
Newspaper
Paint, indoor latex/acrylic
Pencil (lead-graphite, coloring)
Perfumes (dependent on alcohol content)
Petroleum jelly
Plaster (nonlead content)
Play-Doh
Polaroid picture fluid
Porous-tip marking pens
Prussian blue
Putty (less than 2 ounces)
Rouge
Rubber cement
Sachets (essential oils/powder)
Shampoos (liquid)
Shaving creams and lotions
Soap and soap products
Spackles
Suntan preparations
Sweetening agents
Teething rings
Thermometers (mercury)
Toilet water
Toothpaste
Petroleum jelly
Vitamins (with/without fluoride)
Warfarin
Water colors
Zinc oxide
Zirconium oxide

(Mofenson HC, Greensher J, Caraccio TR: Ingestions considered nontoxic. Emerg Med Clin North Am 2:159, 1984.)

Assessment of NonToxic Ingestions

Failure to properly designate an exposure as nontoxic may result in an overly aggressive approach to treatment, exposing the patient to undue risk from unnecessary therapeutic modalities; on the other hand, it may result in inadequate handling of a potentially toxic situation. The decision to treat or not to treat is based on history, presenting symptomatology, potential to

Table 13-3. Signal Words Relating to Toxic Potential

Signal Word	LD_{50} Oral (mg/kg)	Household Measure
No label	5,000	Pint or more
Caution	50–5,000	Ounce to pint
Warning	50–5,000	Teaspoon to ounce
Danger–Poison	<50	Taste to teaspoon

(Adapted from Mofenson HC, Greensher J, Caraccio TR: Ingestions considered nontoxic. Emerg Med Clin North Am 2:159, 1984.)

cause severe toxicity, the risk versus benefit ratio relating to invasive and noninvasive therapeutic and diagnostic maneuvers, and the experience of the clinician. A nontoxic ingestion is said to occur when the victim either consumes a nonedible product which does not usually produce toxic symptoms (Table 13-2), a medication in therapeutic or otherwise subtoxic amounts, or as characterized by the following discussion. Caution must always be exercised when an evaluation of a case is termed nontoxic, since (1) no product or drug is entirely safe, (2) all products can produce undesirable effects if ingested in a sufficiently great quantity, and (3) a variety of clinical manifestations may be displayed. The designation of an ingestion as nontoxic requires satisfaction of the following criteria[17]:

Absolute identification of the product
Absolute assurance that only a single product was ingested
Assurance that there is no signal word on the container (as described in Table 13-3)
Good approximation of the amount ingested
Assurance that the victim is free of symptoms
Ability to call back at intervals to ensure that no symptoms have developed

When to Suspect a Poisoning or Overdose in a Child

Various elements in the history and clinical presentation of the sick child should raise clinical suspicion for the possibility of an overdose or poisoning—especially if the child is in the at-risk age group of 1 to 5 years, if there is a previous history of ingestions of nonedible substances, or if the child is in the socioeconomic at-risk group. Clinical suspicion should be aroused under the following circumstances:

1. The child has an abrupt onset of an illness, particularly the appearance of an afebrile or complex febrile convulsion, or coma without any apparent cause or explanation.
2. The illness involves multiple organ systems without an apparent cause or explanation.

3. There is an unusual odor from the mouth, there are stains on the clothing, or stains/burns of the skin or mucous membranes around or in the mouth.
4. There is unexplained hematemesis.

The importance of history and an assessment of toxin availability is critical to the awareness that a poisoning has indeed occurred. Assessment of a product ingested from an unlabelled or illegible container is truly a frustrating experience. For the most part, if there is any doubt, emesis, lavage, and/or administration of charcoal and a cathartic may be warranted, unless absolute or relative contraindications to the implementation of these procedures exist, such as the involvement of a caustic or corrosive product as evidenced by symptoms of dysphagia, drooling, and burns on the skin or in the mouth, or if the product has the distinctive odor of petroleum distillates.

Assessment of Symptomatology and Severity in Children

Equally important to the evaluation of the variables and factors previously discussed is the ability to assess physical, neurologic, and laboratory examinations accurately in the different pediatric age groups. To the nonpediatrician, this may result in one of the most common pitfalls. It has already been established that differences exist in the evaluation, ability, and willingness to give a history when young children are compared with older children, adolescents, or adults. In addition, the ability to exhibit toxin-related symptomatology may vary with age and chronicity of exposure.

The American Academy of Pediatrics has extended the age of pediatric care to 21 years of age; therefore, the physical examination of the pediatric poisoned patient requires a basic knowledge of the normal variations within these age groups. It should be remembered that the poisoned child can present with or without symptoms. Those without symptoms may turn out to be a nontoxic exposure or, on the other hand, a potentially toxic exposure where the decision to treat the patient requires specific knowledge about the toxicity of the agent and the degree of exposure. The seriously poisoned patient may present comatose, with convulsions, respiratory depression or failure, cardiac dysrhythmias, GI disturbances, or any combination of these manifestations. The poisoning may also be complicated by trauma or an underlying medical condition. Clues to the diagnosis may be helpful when the poisonous agent is unknown in single entity exposures.

When evaluating the poisoned patient, stabilization procedures should always precede definitive poison management. One of the common causes of death occurring outside the hospital in poisoned/overdosed patients is upper airway obstruction, which may result from CNS depression and aspiration, convulsions, muscle paralysis, or the presence of foreign bodies. Therefore, it is imperative that establishment and maintenance of an adequate airway and ventilatory function, as well as restoration of adequate heart rate,

rhythm, and blood pressure, be part of every initial patient assessment. Evaluation of these parameters should also be obtained with adequate and due caution for the cervical spine. Any patient presenting in coma or with severe CNS or respiratory depression should receive an endotracheal tube, cuffed over the age of 9 years to protect against aspiration. If the patient does not tolerate the tube, chances are he or she does not need it. Endotracheal intubation is therefore the best test of protective airway reflexes. It should be remembered that an appropriate gag reflex may be absent in an otherwise normal individual, thus it may not be a good test of protective airway reflexes. Subsequently, adequate ventilation and perfusion should be assessed by clinical evaluation and arterial blood gases (ABGs).

Cardiovascular dysfunction of varying degrees may be induced by toxins which cause a loss of vascular tone (phenothiazines), have a direct myocardial depressant effect (barbiturates) and/or produce dysrhythmias (cyclic antidepressants). Best initial assessment of the cardiovascular status in a patient is by blood pressure, heart rate, and rhythm; assuming one knows the normal and acceptable ranges for the particular age group. The minimum acceptable systolic blood pressure in an adult may be 80 mmHg, or 90 mmHg in age 40 and over. Whereas in children 1 to 8 years of age and in infants under 1 year, 70 mmHg and 60 mmHg respectively, are considered adequate. The appearance of hypotension in a previously well and healthy child may mean that circulatory shock and collapse are imminent. Adequacy of circulation and tissue perfusion may be inferred from skin moisture, temperature, capillary refill, and the urinary output (at least 0.5 to 1 ml/kg hr). Catheterization of the bladder, while not usually required in the conscious and cooperative patient, may be required in the comatose patient to avoid bladder distention, as well as providing a monitoring parameter for urinary output.

In cases of poor circulatory function that fails to improve after establishment of adequate ventilation and oxygenation, the patient should be placed in the Trendelenburg position in an attempt to aid venous return. Fluid challenge should be used and will improve the circulatory function if hypovolemia is a problem. If the child does not respond to adequate fluid challenges (20 ml/kg), additional invasive critical care monitoring (e.g., CVP, PCWP) may be considered to guide further fluid administration. Vasopressors may be required; it should be remembered that the choice of the appropriate vasopressor may depend on the toxicologic derangement.

If the patient is comatose, the following are indicated as soon as vital functions are stabilized:

1. Administration of 100 percent oxygen
2. Administration of 0.5 g/kg glucose as 10 to 25 percent dextrose in water (after a blood specimen for glucose determination is obtained); too large an amount of glucose delivered too rapidly may result in reactive hypoglycemia in the child who does not have diabetes mellitus[18]
3. Assessment of ABGs, BUN, creatinine, liver-function tests, blood

ammonia and electrolytes, blood culture, osmolality, toxicology screen, and an additional tube of blood held in reserve

4. Administration of naloxone 0.1 mg/kg, up to 2 mg IV in a child or 2 mg initially up to 10 mg IV in an adult

5. Administration of thiamine 100 mg IV if the patient is malnourished or a chronic alcoholic (in adolescence)[19]

6. Careful recording of the negative or positive response to glucose and naloxone

Physical Examination of the Poisoned Child

Failure to recognize the subtle differences, as well as normal age-related phenomena in the pediatric age group may result in the loss of a crucial assessment parameter. A thorough examination should be conducted, recording the precise time of the examination. Repeated examinations and clinical assessments, including continuous monitoring of vital signs, are mandatory, since changes in the patient's condition may develop with dramatic rapidity. The following vital functions should be recorded:

1. Respiratory pattern—rate and depth (requires 1 full minute to evaluate because of variability in infants)
2. Heart rhythm, rate, strength of pulse
3. Blood pressure—correct size cuff important for accurate determinations (alternately Doppler method may be used)
4. Temperature—the rectal temperature determined with an instrument that can detect the extremes of hyperthermia and hypothermia
5. Weight—a baseline weight extremely important in children
6. The child's cry—strength of the cry and the response to procedures an important parameter of evaluation
7. Capillary filling—determined by pressure on a fingernail, a reliable indicator of tissue perfusion

The physical examination of the child should concentrate on the following (the order of which may vary with the patient's presentation):

1. Determine evidence of:
   a. An unusual odor
   b. External trauma
   c. Needle marks, tracts, tatoos and other evidence of chemical abuse
   d. Skin burns or bullae
   e. Abnormal skin color (pigmented, pallid, flushed, cyanotic or jaundiced) and degree of abnormality
   f. Temperature
   g. Diaphoretic or dry skin
   h. Skin turgor (is often used to determine the degree of dehydration in infants and children)

2. Assessment of the breathing rate, depth, pattern and the quality of breath sounds. Children under 6 to 7 years have diaphragmatic breathing, therefore the physician should evaluate the abdominal excursion. Note the ease of respiration and the presence of upper respiratory protective reflexes (cough, swallow, contraction of the pharyngeal muscles with or without gagging). Comatose patients should have airway protection. Patients who have ingested materials that contraindicate emesis should not be made to vomit by assessing the gag reflex.

3. Assessment of the heart rate, rhythm and sounds. Irregularities of the cardiac rhythm due to normal sinus arrhythmia and extrasystoles are common in healthy children. Innocent heart murmurs occur in well over 50 percent of healthy children. Investigation of the dysrhythmia or murmur in a poisoned patient depends on the history, the clinical state of the patient and the toxic agent. All seriously poisoned patients deserve an electrocardiogram and continuous monitoring.

4. The presence of the following during gastrointestinal tract examination should be noted:

1) Circumoral or oral staining or burns.
2) Drooling and dysphagia.
3) Any evidence of the toxic agent (odor, color, pill fragments, blood, coffee ground material, etc.) in the vomitus or aspirated material.

5. A thorough abdominal-rectal examination for organomegaly, tenderness, bladder distention, and the presence or absence of bowel sounds should be performed and findings recorded.

6. The neurological survey is particularly important, noting:

1) Level of consciousness and mental status. If the patient appears comatose, check response to noxious stimuli in the form of pressure on fingernail with a blunt object. Note relaxation of the angle of the mouth or if comatose, blowing out of cheek (cranial nerve VII). In children, it is important to note their behavioral state and their interaction with their parents and the examiner. Language and cognitive function can be examined in older children and adolescents.
2) Pupil size, reactivity to bright and dim light and accommodation, shape, equality, consensual reflex, corneal reflex (cranial nerve V), nystagmus, ocular movements, deviation of eyes (cranial nerves IV, III, VI), oculogyric crisis and fundoscopic examination (for papilledema and retinal hemorrhages).
3) Motor system function; convulsions and involuntary movements. Look for tremors, myoclonus, asterixis in toxic metabolic disorders; increased muscle tone, decorticate and decerebrate rigidity or focal weakness in neurological structural lesions, and fasiculations of anterior horn cell disease. Some types of convulsions in children may be subtle. Note the position of the patient.
4) Reflexes; deep tendon, superficial abdominal and cremasteric, vestibulo-ocular brain stem reflexes (cold caloric and doll's eye;

cranial nerve III, VI, medial longitudinal fasciculus and vestibular part of cranial nerve VIII, midbrain). Always exclude cervical spine injury prior to doll's eye testing.

5) The presence of focal neurologic findings and pathologic reflexes and clonus warrants consideration of structural neurologic lesions. The plantar flexion of the toe reflex (Babinski reflex) is present up to 12 to 24 months of age in healthy infants and appears to be dependent on the state of central nervous system myelination. A limited amount of ankle clonus (5 beats) may be present normally up to 4 months of age.
6) Check for meningeal signs. Always exclude cervical neck injury prior to testing of meningeal signs.
7) Autonomic nervous system function; indicated by evaluation of cardiorespiratory systems, the bladder and bowel function, and the temperature control

Coma in the Poisoned Pediatric Patient

The Glasgow Coma scale underestimates the patient with intoxications and should not be used to evaluate the poisoned patient. The comatose poisoned patient is better monitored by the Modified Reed Coma Scale (Table 13-4).

One of the most important diagnostic considerations is to differentiate a structural primary central nervous system disorder, which requires advanced radiologic evaluation (CT scan) and may require neurosurgical consultation, from a toxic-metabolic or systemic disorder, which requires meticulous supportive and possibly antidotal therapy. The incidence of the toxic metabolic disorder exceeds the structural etiology by 15 to 1.[20] It should be realized that both situations may coexist. Table 13-5 delineates the differences between neurologic coma and toxic metabolic coma.

Table 13-4. Modified Reed's Coma Scale

Stage	Conscious State	Pain Reaction	Reflexes	Respiration	Circulation
0	Asleep	Arousable	Intact	Normal	Normal
1	Comatose	Withdraws	Intact	Normal	Normal
2	Comatose	None	Intact	Normal	Normal
3	Comatose	None	Absent	Normal	Normal
4	Comatose	None	Absent	Abnormal (Cyanosis)	Abnormal (shock)

(Adapted from Reed CE, Driggs MF, Foote CC: Acute barbiturate intoxication: A study of 300 cases on a physiologic system of classification. Ann Intern Med 37:290, 1952.)

Table 13-5. Differentiation of Structural Neurologic Coma from Toxic Metabolic Coma

Signs	Structural Neurologic	Toxic Metabolic
Motor signs precede conscious changes	Usually	Rarely
Brain stem compression	May be present	Absent
Focal signs	Often present	Usually absent[b]
Cranial nerve palsies	May be present	Usually absent
Brain level dysfunction	Consistent	Inconsistent
Rostrocaudal progression[a]	Present	Absent
Pupillary reflex	Develop nonreactive	Preservation of reactivity[c]
Neurologic signs (symmetry)	Asymmetric	Usually symmetric
Neurologic signs; variability from one examination to another	Usually do not vary	May vary
Spasticity	Present until reaches lower pons	Usually flaccid[b]

[a] Rostrocaudal progression is a progression of the respiratory pattern, pupil size, reactivity, oculovestibular response, and motor response from one level to another (i.e., from the diencephalon to the mid-brain to the pons and medulla).

[b] Exceptions to flaccidity and focal signs may occur with phenothiazines, glutethimide, cyclic antidepressants, methaqualone, and stimulants and the sequelae to intoxication.

[c] Exceptions to pupil reactivity are anticholinergics and glutethimide which may give a unilateral dilated pupil.

FAILURE TO APPRECIATE THE PARTICULARS OF PEDIATRIC DECONTAMINATION PROCEDURES

Failure to appreciate the differences in adult versus pediatric decontamination modalities may permit evolution of incorrect and even dangerous treatment modalities in the child. In discussing the basic management of poisoning, it will be assumed that an attempt to diagnose the intoxication has been made, although the exact identity of the agent may still not be known. It is also assumed that the management and maintenance of vital functions, the first priorities in the management of the poisoned patient, have received appropriate attention. It should be remembered that most ingestions in young children involve nontoxic products or those that cause only minor toxicity.[21] A frequent error is to treat all ingestions aggressively, even when unnecessary.

General External Decontamination

Ocular exposures warrant immediate attention by irrigation with copious amounts of water or saline solution. Wrapping the child in a sheet or blanket, papoose style, may help control the writhing infant and may also be used in other procedures in which control of flying limbs is preferred. In these instances, the help of the parent or guardian may be beneficial in calming the child and making him or her more amenable to health care personnel and procedures. Dermal exposure requires removal of clothes, followed by complete soap and water washing of the child (including the hair, fingernails, and navel), in an attempt to limit further percutaneous absorption. Again, the help of the parent is suggested and may limit the fear reaction of the child.

General Internal Decontamination

Evaluation of the need for GI decontamination in the child must include assessment of various factors concerning the toxic versus nontoxic potential. Although transit time through the GI tract is shorter in the infant, it has not been systematically investigated in pediatric intoxications. The shorter transit time in infants appears to approach adult values at approximately 2 to 3 years of age, with considerable individual variation. Absorptive properties of the infant reach adult levels at 3 to 6 months of age, which may have some impact on substances left in the gut that have the potential for prolonged or continued absorption. These factors are of importance, since GI exposure appears to be the most common route of poisoning in children.[1] Whereas adult and adolescent suicide attempts often involve polydrug ingestions and the history obtained is often unreliable, most children ingest only a single substance, and histories may be more reliable, since the ingestion often occurs while being observed. The accidental ingestion of a bad-tasting household product often results in only one swallow (5 ml) in the child. Medications thought of as candy by children may be ingested in multiple quantities.

Dilutional Therapy

In caustic or corrosive ingestions, the use of dilution techniques with milk or water may be warranted. Contraindications to the use of this dilutional therapy include an inability of the patient to swallow, signs of upper airway obstruction or esophageal perforation, and shock. The administration of greater than 15 ml/kg in a child (up to a maximum of 250 ml) may result in vomiting, the event the physician was trying to avoid. Dilutional therapy also has other drawbacks; consequently, it should not be used routinely. Evidence indicates that ingestion of a large volume of water has the

potential to enhance GI absorption of certain toxins by increasing the solubilization of the product.[21-25] The use of specific neutralization techniques with caustics or corrosive ingestions should be abandoned. An exothermic reaction will result in a thermal burn in addition to the chemical burn.

Emesis

Controversies concerning the use of syrup of ipecac for pediatric intoxications have included discussions on appropriate dosage, use in infants under 1 year of age, co-administration of fluids, and ambulation after ipecac administration. Although no one method of GI decontamination demonstrates more than 30 to 50 percent effectiveness in the removal of orally ingested toxins, it is generally accepted that emesis or lavage should be performed up to 4 hours postingestion, and in some cases up to 12 hours postingestion.[26] It should be well noted that certain substances, including anticholinergic agents and opioids, as well as the ingestion of a large amount of tablets or capsules, may result in a delay in gastric emptying, leaving the compound available for removal long after ingestion. This element has been demonstrated in well-controlled adult studies,[27] but not in children.

Emesis is generally accepted as being more effective in removing ingested substances in children than lavage, if only due to the absolute size of the tube tolerated by the child. It is most effective in preventing further systemic absorption of a toxin if induced within 30 minutes of ingestion. Children often cannot tolerate large-bore tubes, which make the removal of partially dissolved tablets, capsules, and plant parts difficult, if not impossible. Emesis is not without its own inherent dangers; consequently, the unnecessary and routine administration of syrup of ipecac should be avoided. The following remain as contraindications to the use of ipecac syrup for induction of emesis.

Relative Contraindications:

1. Ingestion of a high-viscosity petroleum distillate that is usually not systemically absorbed but may result in lipoid pneumonia if aspirated during emesis
2. Ingestion of an agent that is likely to produce rapid depression of consciousness, loss of protective airway reflexes, or convulsions (i.e., propoxyphene, camphor, isoniazid, strychnine, ethanol, cyclic antidepressants)
3. Significant vomiting occurring prior to presentation
4. A child under 6 months of age who may have immature protective airway reflexes[21]
5. Ingestion of a foreign body, in which emesis is usually ineffective in the removal of the object, while the risk of creating or worsening of airway obstruction and aspiration exists
6. Certain other petroleum distillates in small quantities, which have a

much greater potential to cause pulmonary and CNS toxicity if aspirated than if left in the gastrointestinal tract

Absolute Contraindications:

1. Ingestion of a caustic or corrosive product
2. A comatose or seizing patient
3. Presence of hemetemesis
4. Neurologically impaired children who may lack or have depressed upper airway protective reflexes, hence enhanced aspiration potential

Emesis in a child under 1 year of age was not recommended in the unsupervised home setting, for fear of immature protective airway reflexes. However, this concept has recently been challenged, with some clinicians recommending use of ipecac at home in a child 6 months or older.[28–30] The appropriate dose of ipecac syrup should be based on the age of the child as shown in Table 13-6.[21,29] This dose of ipecac may be repeated once if the child does not vomit within 15 to 20 minutes. Further investigations have now suggested that 30 ml may be employed safely in patients 12 to 60 months of age, resulting in a more rapid onset of vomiting.[32,33] The mean time for emesis reported in pediatric studies range from 14 to 37 minutes, with an average of 20 minutes. Increasing the dose from 15 to 30 ml in children has been shown to decrease the time to emesis by 10 minutes.[32] The induction of emesis seems to be delayed in infants,[29] possibly making lavage or the preferential initial use of activated charcoal a more advantageous modality in this instance.

Although still controversial,[34] most authorities will administer 5 ml/kg body weight of water after the ipecac dose in infants and children, and 250 ml in older children or adults.[22] In an attempt to improve the efficacy of the emetic agent, patients are kept ambulatory in the belief that this will hasten the onset of emesis. It is further believed that this procedure will aid in decontamination by washing the pill fragments and debris from the gastric mucosal folds, permitting more forceful and complete emptying of the stomach. Recent studies have challenged the efficacy and practicality of this procedure.[21,34,35] Tepid water should be used preferentially over cold water, since the latter can decrease gastric emptying time.[21,22] For the child who refuses to drink syrup of ipecac, a small nasogastric tube may be inserted, whereupon the dose of ipecac and an appropriate volume of water may be

Table 13-6. Pediatric Dosages for Syrup of IPECAC

Age	Amount (ml)
6–9 mo	5
9–12 mo	10
1–10 yr	15
>10 yr	30

easily introduced into the stomach. This tube is removed after the dose is administered.

If vomiting does not occur after two doses of syrup of ipecac, lavage should be performed, based on the toxicity of the ingested substance. Although normal doses of ipecac syrup pose little threat of toxicity, ipecac has reportedly produced mild adverse effects, such as protracted vomiting, diarrhea, lethargy, irritability, diaphoresis, and fever.[36] Symptoms, if noted, should not be mistaken for part of a toxic syndrome. Lethargy, the appearance of which we associate with a depressed level of consciousness, is reported to occur in 20 percent of patients after ipecac-induced vomiting.[29,33,37] However, in spite of the good track record of syrup of ipecac, studies have suggested that the safety of ipecac should be reevaluated and that possibly charcoal should be used as the sole agent for GI decontamination. Death was reported in a child who was inadvertently given a massive dose of syrup of ipecac.[38] In a subsequent case report, when given to a child with a congenital diaphragmatic hernia, syrup of ipecac caused death following protracted vomiting.[39] Gastrointestinal decontamination by the induction of emesis may be effective if induced within 2 to 4 hours after ingestion. Emesis may be considered the treatment of choice over gastric lavage for solid ingestions in children, because the smaller diameter lavage tubes will not permit removal of intact tablets, capsules, or other ingested debris. With the imminent onset of emesis, the child's head should be lowered 15 to 20 cm below the hips (spanking position) and the vomitus inspected for remnants of pills or other toxic substances, and the appearance, color, and odor noted.

Emetic agents, other than syrup of ipecac, have been advocated in the past. Salt water should not be used as an emetic in children, reportedly producing fatal hypernatremia and other electrolyte abnormalities.[21,40] Apomorphine is used parenterally but must be freshly prepared prior to use. This narcotic derivative has the potential to cause CNS depression, outlasting attempts to reverse its effects by bolus administration of naloxone. Therefore, its use automatically mandates hospital observation. Although apomorphine produces prompt emesis, its use involves complications not easily accepted by most authorities. Mechanically induced emesis by stimulation of the posterior pharynx, although a popular lay public method of inducing emesis, has the potential to produce laceration of the posterior pharynx, and trauma to the oral cavity. It is not recognized as an effective method, relative to the frequency or amount of return.[22,41] It is therefore not recommended as an alternative.[21,22,41] Likewise, the use of copper sulfate is considered toxic and is therefore not recommended.[21,22]

Detergents and detergent combinations, such as liquid emetic agent (LEA), a combination of sodium tripolyphosphate, tetrapotassium pyrophosphate, sodium saccharin, and strawberry flavoring, have been used successfully in inducing emesis but require continued investigation as to their statistical efficacy.[22] Some believe that an appropriate liquid detergent may be used as an alternative when syrup of ipecac is not readily available and rapid induction of emesis is warranted.[22]

Lavage

If a contraindication to the induction of emesis exists, or if the patient will benefit from immediate gastric aspiration and subsequent lavage, gastric lavage procedures should be instituted. Plant and large pill fragments are not readily removed through even the largest of lavage tubes; therefore, gastric lavage may not be as effective as forceful emesis.[21] As expected, gastric lavage is not without its own inherent problems. These problems can be minimized if the contraindications for gastric lavage are respected:

1. Caustics or corrosive ingestions, where the danger of esophageal perforation is real (some authorities recommend insertion of a small, well-lubricated tube into the stomach to remove acid within 30 minutes after ingestion.[42])
2. Uncontrolled convulsions, due to the risk of injury and aspiration
3. Coma and/or absence of protective upper airway reflexes without insertion of an endotracheal tube (cuffed in children greater than 9 years old), to protect against aspiration
4. Significant dysrhythmias (vagal effects with insertion of the tube may precipitate a fatal dysrhythmia)
5. Certain petroleum distillate hydrocarbons, due to the incidence of aspiration pneumonia being higher with lavage than emesis

The best results are obtained with gastric lavage when the largest bore tube that can be reasonably and safely inserted orally in the patient is used. A nasogastric tube is severely limited in value for lavage techniques other than use in certain liquid ingestions. Although a lavacuator hose, or at least 36 to 42 Fr Ewald tube is used in adults, children often cannot tolerate larger than a 26 to 28 Fr orogastric tube. One investigator states that 36 Fr orogastric hoses can be safely passed in children as young as 1 year of age.[43] Therefore, for the logistical reasons of inadequate lavage tube size, emesis is preferred for removal of a solid ingestion in children (i.e., if contraindications to emesis do not exist). After the tube is placed, aspiration of gastric contents is followed by positive identification of tube placement, preceding instillation of any lavage fluid.

If the child is to be endotracheally intubated prior to lavage, the use of a cuffed endotracheal tube in a child under 9 years of age is not recommended (Table 13-7) for fear of tracheal damage. If a cuffed tube is to be used in the older child, it should be deflated immediately following lavage.[24] Without elective endotracheal intubation, the patient should be placed in the left lateral decubitus position, with the head 15 to 20 cm lower than the hips (Trendelenburg position). This will aid in the prevention of aspiration if vomiting does occur without physical protection of the airway.[21,43]

Although the standard lavage fluid in adults is either water or saline, children should only be lavaged with 0.45 percent saline. The use of an alternative fluid composition in children may lead to hypernatremia, hypocalcemia, and/or other severe electrolyte imbalances.[29,43] One tablespoonful

Table 13-7. Pediatric Resuscitation Equipment

Age/ Weight	Endotracheal Tube	Suction Catheter	Foley Catheter	Nasogastric Tube[a]
Newborn (3.5 kg)	3.0–3.5 Uncuffed	8 Fr	5–8 Fr Feeding	5–8 Fr Feeding
6 months (7.0 kg)	3.5–4.5 Uncuffed	8–10 Fr	8 Fr	8 Fr
1–2 yr (10–12 kg)	4.0–4.5 Uncuffed	10 Fr	10 Fr	10 Fr
5 yr (16–18 kg)	5.0–5.5 Uncuffed	14 Fr	10–12 Fr	10–12 Fr
8–10 yr (24–30 kg)	5.5–6.5 Cuffed	14 Fr	12 Fr	14–18 Fr

[a]The use of larger tubes for lavage has been suggested; see text for discussion.

of salt in a glass of water, administered to a 3-year-old child, has the potential to raise the serum sodium concentration by 25 meq/L.[22] By contrast, some authorities still recommend normal saline due to a supposed "decreased risk of electrolyte imbalance."[21] The appearance of hyponatremia and hypokalemia has been reported with tap water lavage.[44]

It has been suggested that massage of the left upper quadrant may aid in dislodging particles from the gastric folds, while facilitating overall return. One drawback to a too-vigorous approach in this manner may be the induction of vomiting. The lavage fluid should also be warmed so as to avoid the production of hypothermia in the child. Whereas adults may tolerate up to 250 to 350 ml lavage aliquots, 15 ml/kg should be used in children.[21,22] If these volumes are exceeded, the stomach contents may be forced through the pylorus into the intestine, or possibly cause vomiting. The lavage should be continued until a consistently clear fluid return is achieved.

The clinician should always be cognizant of the ingestants known to cause gastric concretions, such as ferrous sulfate, meprobamate, glutethimide, salicylates, and large amounts of dry tablets. A concretion may require prolonged forceful lavage to dissolve, break up, or otherwise remove the bolus of material. As a last resort, endoscopic or surgical removal may be required. Clues to the presence of concretions are failure to respond to treatment, continued deterioration, or a measured blood concentration that does not decrease despite adequate GI decontamination modalities.

Intestinal lavage has been advocated by some for management of paraquat, diquat, and ingestion of other toxins.[21,45] This procedure may pose a significant risk of fluid and electrolyte imbalance in children, unless a balanced solution is used.[21,46] Its value in children still remains to be defined.

Activated Charcoal

Activated charcoal is extremely useful in the decontamination of the GI tract. The effectiveness of activated charcoal lies in its ultrasmall particle size and its very large surface area. This permits adsorption of a number of

chemical agents. The standard-grade activated charcoals commonly used today have a surface area of more than 1,000 m^2/g. A new superactivated charcoal boasts 3,000 m^2/g.[47,48] Traditionally, the use of activated charcoal given concurrently with certain antidotes such as ipecac or *n*-acetylcysteine was contraindicated. This is now being challenged.[49] Only two other contraindications to charcoal administration exist at this point. The ingestion of a caustic or corrosive agent, since charcoal does not effectively adsorb these compounds, may cause vomiting, and will most probably hinder any endoscopic attempts to evaluate the esophageal and/or gastric mucosa. Charcoal should also not be repeatedly administered to a patient who has absent bowel sounds or any evidence of ileus.

Activated charcoal cannot adsorb all known toxins. It has limited value with boric acid, caustics, corrosives, cyanide, as well as other elemental metal ingestions. Certain pesticides and other poorly water-soluble compounds may also not be effectively captured by the charcoal complex. The alcohols may be adsorbed to a limited extent by charcoal,[50] but this appears to have little clinical significance, since the systemic absorption of the alcohols is so rapid.

The present controversy, which may very well change the face of our general poison management protocols, is the use, and therefore the effectiveness, of activated charcoal without the prior utilization of syrup or ipecac. A number of studies have recently appeared in the literature attempting to reconcile this issue. Some have alluded to the superiority of activated charcoal alone, without the initial use of ipecac, and have since recommended its immediate use without preceding emesis.[51–58]

The dose of standard-grade activated charcoal is usually 50 to 100 g in adults and 1 to 2 g/kg per dose in children. Some investigators have suggested the use of smaller doses of the superactivated charcoal, since its enhanced surface area markedly improves its binding characteristics.[21,47,48] This could have a definite advantage in children, requiring only approximately one-half the amount of a standard-grade charcoal slurry. In addition, if the patient vomits, what is left in the GI tract will now be potentially twice as effective. The charcoal is mixed and administered as a slurry in at least 100 ml water or 35 to 70 percent sorbitol solution and is given orally or through a gastrically placed tube. The sorbitol has the advantage of improving the palatability of the charcoal while acting as a superior cathartic compared to the magnesium or sodium salts, reducing the time required to produce charcoal-laden stools.[58,59] The use of sorbitol in children is not without concern, for it does possess the potential to induce significant diarrhea and electrolyte imbalance. A cathartic should be used in order to prevent the formation of "charcoal briquets" in the GI tract.[60]

It would be beneficial when dealing particularly with ingestions in the pediatric age group to find an additive that will impart a more palatable nature to the charcoal slurry, thereby improving compliance when administration of the antidote is necessary. Many types of vehicles or additives have been advocated, including milk, sherbet, ice cream, chocolate flavoring, and

mineral oil. All have been shown to have the potential to decrease the absorptive capacity of the charcoal and should therefore be avoided.[22,58,61,62] In contrast to the aforementioned additives, cherry flavoring does not appear to influence the absorptive capacity of charcoal.[22] The ideal additive, still to be determined, would mask the gritty dirtlike texture with or without imparting a flavor and would not reduce the efficacy of the antidote. Some clinicians believe that sorbitol satisfies some of these criteria. In any case, it has been suggested that if a firm approach is taken, the charcoal is more likely to be taken by the child.[21] The charcoal slurry should be placed in an opaque container with a fitted lid to mask its color. The suspension should be sipped through one or two straws, alternating with sips of a clear fluid, such as water. If attempts to get the child to self-administer this antidote within 15 to 20 minutes have failed, a nasogastric tube should be placed, the slurry further diluted, and the charcoal administered through the nasogastric tube.[21] Superactivated charcoal may be used in smaller doses, making administration easier by virtue of the absolute volume to be administered; in fact, it may be more efficacious in children due to a better probability of retaining the slurry when smaller volumes are administered.[21]

Although the use of activated charcoal is generally considered benign, various problems including death have been reported, apparently due to aspiration of charcoal.[63–65] By contrast, some authorities believe that aspiration of charcoal is no worse than aspiration of gastric contents alone and that such cases are due only to the adverse effects of gastric fluid aspiration and not particularly to the charcoal component.[21]

The use of multiple doses of activated charcoal, given every 2 to 6 hours, is now gaining widespread acceptance for such ingestions as phenobarbital, salicylate, theophylline, digoxin, and carbamazepine.[66–74] The use of multiple-dose activated charcoal therapy appears useful not only in cases of long-acting or sustained-release preparations, but with those agents undergoing enterohepatic or enterogastric recirculation as well. It is now clearly recognized that activated charcoal can produce a gastric dialysis[66–76] in addition to serving as a stool marker indicating that no further compound is available for absorption. This GI dialysis concept has proved useful for those agents ingested orally as well as for those administered intravenously. The efficacy of this procedure is purportedly due to the creation of a concentration gradient, enhancing the rate of diffusion from the central compartment into the GI tract, with the charcoal acting as an adsorbent sink. The clinician should not indiscriminantly administer multiple-dose charcoal without a proper evaluation of the risks, clinical symptomatology, and its reported usefulness for a particular compound. Charcoal administration is not without risk. The use and relative efficacy of multiple-dose charcoal in the pediatric literature is limited to case reports.[21] If this modality is deemed necessary, a dose of 1 g/kg standard-grade activated charcoal (or alternately, an

appropriate dose of superactivated charcoal), administered every 4 to 8 hours with saline or sorbitol, seems appropriate for children.[21]

Cathartics

Cathartics are used in an attempt to shorten the transit time of the toxin or toxin–charcoal mixture through the GI tract. Despite the almost universal use of cathartics in the management of poisonings, there is no scientific or clinical proof to support its efficacy.[21,22,77] The exception is the cathartic used to prevent constipation from the administration of charcoal. Adverse effects, such as fluid loss and moderate to severe electrolyte imbalance, appear to be more common in pediatric patients.[21] Fleet's phospho-soda, a favorite with adults, should not be used in children, due to the risk for severe electrolyte complications.[26] Ionic cathartics enjoyed almost widespread notoriety until recently, when the use of sorbitol has been advocated. Sorbitol, as well as the saline cathartics, is not known to significantly affect the adsorptive capacity of activated charcoal.[21,22,26,78,79] It is therefore appropriate for these agents to be mixed with and/or given concurrently with activated charcoal.[80] In addition, the use of 70 percent sorbitol solution has been recommended.[79,81–82] The use of this high concentration in children is now being questioned, since hypernatremic dehydration, possibly associated with a charcoal–70 percent sorbitol mixture, occurred in a 3-month-old infant.[83] It was clear that the concentration of sorbitol used in this case was excessive, although an actual cause-and-effect relationship was not established. Nevertheless, infants and small children should probably receive a more dilute solution, such as a 35 percent concentration, and should be monitored more carefully for dehydration and electrolyte disturbances. Sorbitol is not recommended in a patient under 1 year of age and should be used with caution in the child under 3 years of age.

The dose of sodium or magnesium sulfate in children is 250 mg/kg; 5 ml/kg is the appropriate dose of magnesium citrate. Sorbitol dosing (1 to 1½ g/kg) is controversial in children. It should also be noted that the safety of cathartics, administered every 4 hours with the multiple activated charcoal, has not been established. The use of cathartics is relatively contraindicated in the following circumstances:

1. The actual or suspected presence of an ileus, as indicated by an absence of bowel sounds[77]
2. The presence of an intestinal obstruction or evidence of intestinal perforation
3. The use of magnesium-containing cathartics in renal failure
4. The use of sodium sulfate in congestive heart failure or other circumstances requiring sodium restriction
5. A preexisting electrolyte disturbance
6. A cathartic ingestion/intoxication

SPECIAL PROBLEMS IN THE DEFINITIVE TREATMENT OF PEDIATRIC OVERDOSE PATIENTS

We have seen up to this point that differences do exist in the evaluation, diagnostic considerations, and basic management of children as compared with adults. These subtle differences also carry over to the more definitive secondary management techniques.

Procedures for Enhancement of Elimination

One of the methods used to enhance the elimination of a toxin has already been discussed, that being gastric dialysis. The following discussions apply to other methods by which one may enhance the elimination mechanisms (some in vivo and some extracorporeal) all leading, it is hoped, to a shortened course of toxicity after a poisoning. In general, these modalities are needed in only a small number of cases and should therefore be reserved for those severe and possibly life-threatening and methodologically responsive exposures. Since these modalities do carry some inherent risks of their own, they should not be included in the routine care of the poisoned patient. Again, one must consider the innate differences between adults and children when evaluating the risk versus benefit ratio for each of these modalities.

Alkalinization of the urine, by virtue of its ability to produce an ion-trapping effect, can significantly increase the elimination of phenobarbital, barbital, and salicylates. Prevention of the renal tubular reabsorption of these compounds is accomplished by altering the ratio of ionized to unionized drug, favoring the nonabsorbable ionic form. This is accomplished by raising the urine pH into the range of 7.5 to 8.0, without the blood pH going beyond 7.5. This goal is attained by the use of intravenously administered sodium bicarbonate, 1- to 2-mEq/kg bolus followed by infusion, in itself necessitating the close monitoring of the urinary, as well as the plasma pH. This procedure may also place the patient at risk of hypokalemia and/or hypocalcemia. The use of forced diuresis, where fluid administration may or may not be accompanied by diuretics to stimulate urine flow three to six times that of normal, has been abandoned by some clinicians because of the risks associated with this maneuver.[77] In fact, recent studies suggest that urinary alkalinization alone, without diuresis, may be just as effective as forced diuresis maneuvers.[84] The risks associated with the induction of a high urine flow rate, such as cerebral edema, pulmonary edema, disturbances in acid-base and electrolyte balance, and drug-induced SIADH (e.g., barbiturates, opioids, salicylates, cyclic antidepressants, sulfonylureas, biquanides, carbamazepine) often place the patient at increased risk of morbidity.[85] This is especially true in children, in whom administration of large fluid loads and sodium bicarbonate will induce a more pronounced problem than in their adult counterparts. This problem is compounded

when, by virtue of the specific toxicologic action, certain exposures actually produce cerebral or pulmonary edema or renal failure.

Acid diuresis, accomplished with the use of intravenous ammonium chloride, supposedly enhances the elimination of weak bases such as amphetamines, quinine, strychnine, and phencyclidine. This procedure has since been abandoned, and rightly so, because of its potential to cause severe metabolic acidosis, precipitation of myoglobin in the renal tubules, and severe electrolyte shifts.[85]

Extracorporeal Methods of Toxin Removal

Although fairly useful with certain adult poisonings, extracorporeal means of removing toxic substances may have limited value in children and small infants, depending on the age of the patient and the clinical characteristics of the exposure. These modalities are rarely first-line choices. They are reserved for the severe life-threatening poisonings or overdoses that are amenable to dialysis or hemoperfusion. The ideal characteristics of a removable toxin include low molecular weight, low protein and tissue binding, high water solubility, and a small volume of distribution (less than 1 L/kg).[86] These measures also prove useful for those instances in which maintenance of appropriate acid-base balance becomes difficult or refractory, severe electrolyte imbalances arise, overhydration due to acute renal failure is evident, or the patient develops uremia and its related syndromes. Consultations should be sought with the nephrologist under the following circumstances[77]:

1. Progressive deterioration in patient condition despite intensive supportive treatment
2. Severe intoxication resulting in refractory hypoventilation, hypothermia, and hypotension
3. Complications that portend catastrophic deterioration
4. Impairment of normal drug excretory pathways that would result in a prolonged severe course

Even though peritoneal dialysis is reportedly 1/20th as effective as hemodialysis, it is easier and less hazardous to perform. It is particularly useful in small children, in whom the blood vessel size and small blood volume make hemodialysis more difficult.[85] Peritoneal dialysis has been successfully used for barbiturates, bromides, phenytoin, ethanol, ethchlorvynol, ethylene glycol, inorganic mercury, isopropyl alcohol, lithium, methyprylon, quinidine, salicylate, and theophylline.[85] The use of peritoneal dialysis is contraindicated in a patient who has had recent abdominal surgery or if there is evidence of an intrabdominal infection.

Hemodialysis is considered a more effective means of dialysis, but it requires experience, sophisticated technical skill, adequate anticoagulation, and vascular access. This modality exposes the patient to a risk of hemor-

Table 13-8. Pediatric Dosages of Selected Antidotes

Antidote	Indications	Pediatric Dose
N-Acetylcysteine	Acetaminophen toxicity	Loading: 140 mg/kg Maintenance: 70 mg/kg Q4h for 17 doses Orally as a 5% solution
Atropine	Organophosphates/ Carbamates and other severe cholinergic poisonings	0.05 mg/kg (2 mg maximum) every 2–5 min until cessation of symptoms (e.g., pulmonary secretions)
Cyanide kit	Cyanide poisoning	See below:
Hemoglobin (g)	Sodium nitrite 3% (initial dose; maximum 10 ml)	Sodium thiosulfate (initial dose; 12.5g maximum)
8	0.22 ml/kg (6.6 mg/kg)	1.10 ml/kg
10	0.27 ml/kg (8.7 mg/kg)	1.35 ml/kg
12[a]	0.33 ml/kg (10 mg/kg)	1.65 ml/kg
14	0.39 ml/kg (11.6 mg/kg)	1.95 ml/kg
Deferoxamine	Iron intoxication	*Therapeutic dose:* 90 mg/kg IM or IV to a maximum of 1 g, every 4–12 hr; not to exceed 6 g in 24 hr; IV slow infusion up to 15 mg/kg/hr *Diagnostic trial:* 20 mg/kg IM (up to 1 g)
Diazepam	Seizures resulting from intoxication	0.1–0.3 mg/kg up to 10 mg IV slowly over 2 min
Dimercaprol (BAL)	Intoxications from lead, arsenic, gold, mercury, copper, nickel, antimony	IM use only: 3–5 mg/kg initially, followed by 2.5 mg/kg q4h for 2 days, then q6–12h *Note:* dosage and duration varies with specific intoxicant and severity; see recommendations for each poisoning
Diphenhydramine	Dystonic reactions from phenothiazine and related compounds	1–2 mg/kg IM or IV over 2 min
Ethanol	Methanol, ethylene glycol	*Loading:* 7.6–10 ml/kg of a 10% solution in D_5W over 30 min IV; or 0.8–1.0 ml/kg 95% ETOH diluted to a 20–30% solution for oral administration *Maintenance:* 1.4 ml/kg/hr of a 10% ETOH solution IV (0.15ml/kg/hr PO 95%); add 91 ml/hr IV (9.6 ml/hr PO) if patient on dialysis

(*Continued*)

Table 13-8. (*Continued*)

Antidote	Indications	Pediatric Dose
Glucagon	β-Blocker intoxication	50–150 μg/kg IV over 1 min followed by infusion of 1–5 mg/hr tapered over 5–12 hr *Note:* do not use supplied diluent (contains phenol)
Methylene blue	Methemoglobinemia	0.1–0.2 ml/kg of a 1% solution (1–2 mg/kg) IV over 5 min
Naloxone	Opioid toxicity	0.1 mg/kg IV, IM (Note: many clinicians use normal adult doses in children); maintenance infusion two-thirds the initial reversal dose used, per hour
Physostigmine	Severe anticholinergic poisoning	0.03 mg/kg up to 0.5 mg IV over 2 min repeated to a maximum dose of 2 mg if required *Note:* reserved for severe, life-threatening toxicity when conventional therapy has failed
Pralidoxime (2-PAM)	Organophosphate poisoning	25–50 mg/kg IV over 2 min or IM; repeat every 8–12 hr if needed; if severe can be infused at less than 0.5 g/hr

[a] If hemoglobin unknown, use this dose.

rhage, infection, hypotension, and electrolyte disturbances. The limitation of age or body size vary with the dialysis equipment and local expertise, although it has reportedly been used in newborns.[86]

Hemoperfusion with either charcoal or resin compounds claims the same vascular access problems in children as hemodialysis. Charcoal or resin hemoperfusion may be more effective than hemo- or peritoneal dialysis for certain agents with low water solubility, high molecular weight, and high plasma protein binding.[85] Patient-associated risks are the same as with hemodialysis, with enhanced risk of thrombocytopenia, hypothermia, and hypotension. Whenever these modalities are employed, careful monitoring of the formed blood elements, calcium, glucose, electrolytes, protein, osmolalities, and vital functions should be carried out.

Exchange transfusion is primarily used in young infants and children and is effective with highly protein-bound drugs with low volumes of distribution. It has limited use in older children.

Antidotal Therapy in Children

Another common pitfall in the management of the poisoned pediatric patient is the inappropriate use of antidotal modalities. Table 13-8 outlines the proper indications and dosages in children.

Table 13-9. Indications and Guidelines for Admission

Potential for secondary complications arising from ingestion of a specific substance
Exposure to a highly toxic agent
Symptoms of the toxic exposure:
Because of the nature of the toxin, the patient must be monitored for a period of time even though the symptoms resolve.
Symptoms do not resolve despite therapy.
Suspicion or evidence of nonaccidental ingestion or injury (e.g., child abuse, attempted homicide)

Observation/Admission Guidelines for Children

If adequate facilities are available, observation for 6 to 8 hours in the emergency department will usually be enough time to unmask most delayed-onset toxic reations. Children under 1 year of age are considered at a higher risk of toxicity and often do not show the classic adult symptomatology.[77] Therefore, one must exercise caution with early discharge. Appropriate indications for admission (Table 13-9), other than a patient who is already manifesting signs and symptoms, include exposure to an agent known to be highly toxic, a dose or exposure to a toxin sufficient enough to anticipate the development of toxicity, or a history of an intentional ingestion. The adequacy of the home environment must be assessed before the child's discharge. Older children with suicidal ideations must also be evaluated prior to release.

POISON PREVENTION/AWARENESS

Treatment does not end with the administration of the last therapeutic agent or maneuver. The existing problem must be addressed, whether it is poison-proofing the home, undersupervision of the child, a cry for help, or even child abuse. If warranted, the proper social service authorities must be contacted. If this was truly an accidental ingestion, the clinician must be prepared to teach poison prevention, so a repeat episode can be avoided. Perhaps the biggest pitfall is that this important modality is often overlooked. Unfortunately, established and study-proven techniques for teaching this topic are not available. The physician must tailor each training session to the patient and family situation encountered. Safety packaging has contributed significantly to the decline in pediatric accidental ingestions. The use of these types of packaging for those households with children should be emphasized during these training sessions.

Parents should be instructed to keep products in their original containers and to discard containers safely after they are used up. They should never call medicine "candy" and should refrain from taking medication in

front of children who are great imitators. Old and out-of-date medications should be discarded, as should those no longer used. Medications and household products should be kept out of reach of children, and children should be taught never to eat anything without checking with a parent or responsible caretaker. The appropriate storage and use of syrup of ipecac and reference to the poison control center should always be stressed.

SUMMARY

We have systematically discussed the potential diagnostic and therapeutic errors that may be realized with dealing with pediatric intoxication. Age-related variability in pharmacokinetic parameters encountered in this age group may alter the clinical presentation and predictability of toxic exposures in children. Failure to recognize the subtle differences between adults and children, with respect to age-related physiochemical phenomena, may result in inaccurate assessments. The use of normal adult treatment modalities in children may be incorrect or actually dangerous. The approach to obtaining and relying on the history, as well as assessment of admission criteria, must also be modified relative to pediatric exposures. Finally, the clinician must be prepared to teach poison awareness to their patients and respective parents/guardians so that these preventable accidents can be avoided in the future.

REFERENCES

1. Litovitz TL, Normann SA, Veltri JC: 1985 annual report of the american association of poison control centers national data collection system. Am J Emerg Med 4:427, 1986
2. Morselli PL, Franco-Morselli R, Bossi L: Clinical pharmacokinetics in newborns and infants; age-related differences. Clin Pharmacokinet 5:485, 1980
3. Plakogiannis FM, Cutie AJ: Basic Concepts in Biopharmaceutics: An Introduction. Brooklyn Medical Press, Brooklyn, 1977
4. Mofenson HC, Caraccio TR: General principles of pediatric pharmacology. Resident Staff Physician 30:96, 1984
5. Yaffe SJ, Juchau, MR: Perinatal pharmacology. Annu Rev Pharmacol 14:219, 1974
6. Shirkey HC: Pediatric clinical pharmacology and therapeutics. p. 98. In Shirky HC (ed): Pediatric Therapy. 5th Ed. CV Mosby, St. Louis, 1975
7. Cavell B: Gastric emptying in preterm infants. Acta Paediatr Scand 68:725, 1979
8. Friis-Hansen B: Body water compartments in children: Changes during growth and related changes in body composition. Pediatrics 28:169, 1961
9. Krasner J, Giacoia P, Yaffe ST: Drug-protein binding in the newborn infant. Ann NY Acad Sci 226:101, 1973
10. Wallace S: Factors affecting drug-protein binding in the plasma of newborn infants. Br J Clin Pharmacol 3:510, 1976

11. Wilson JT, Atwood GF, Shand DG: Disposition of propoxyphene and propranolol in children. Clin Pharmacol Ther 19:264, 1976
12. Morselli PL: Clinical pharmacokinetics in neonates. Clin Pharmacokinet 1:81, 1976
13. Fazen LE III, Lovejoy FH, Crone RK: Acute poisoning in a children's hospital; a 2-year experience. Pediatrics 77:144, 1986
14. McIntire MS, Angle CR: The taxonomy of suicide as seen in poison control centers. Pediatr Clin North Am 17:697, 1970
15. Sobel R: Psychiatric implications of accidental childhood poisoning. Pediatr Clin North Am 17:653, 1970
16. Shnaps Y, Frand M, Rotem Y, et al: The chemically abused child. Pediatrics 68:119, 1981
17. Mofenson HC, Greensher J, Caraccio TR: Ingestions considered nontoxic. Emerg Med Clin North Am 2:159, 1984
18. Levin DL, Moriss FC, Moore GC: A Practical Guide to Pediatric Intensive Care. CV Mosby, St. Louis, 1984
19. Reucer JB, Girard DE, Cooney TY: Wernicke's encephalopathy. N Engl J Med 312:1035, 1985
20. Plum FD, Posner JB (eds): The Diagnosis of Stupor and Coma. FA DAvis, Philadelphia, 1980
21. Rodgers GC, Matyunas NJ: Gastrointestinal decontamination for acute poisoning. Pediatr Clin North Am 33:261, 1986
22. Cuprit GC, Temple AR: Gastrointestinal decontamination in the management of the poisoned patient. Emerg Med Clin North Am 2:15, 1984
23. AAPCC: Gastointestinal dilution with water as a first aid procedure in poisoning. Vew Hum Toxicol 25:55, 1983
24. Chin L: Gastrointestinal dilution of poisons with water; an irrational and potentially harmful procedure. Am J Hosp Pharm 28:712, 1971
25. Henderson ML, Picchioni AL, Chin L: Evaluation of oral dilution as a first aid measure in poisoning. J Pharm Sci 55:1311, 1966
26. Goldfrank LR, Flomenbaum NE, Lewin NA (eds): Goldfrank's Toxicologic Emergencies. 3rd Ed. Appleton-Century-Crofts, Norwalk, CT, 1986
27. Neuvonen, PJ, Vartiainen M, Tokola O: Comparison of activated charcoal and ipecac syrup in prevention of drug absorption. Eur J Clin Pharmacol 24:557, 1983
28. Krenzolok EP, Dean BS: Syrup of ipecac in children less than 1 year of age. Clin Toxicol 22:171, 1985
29. Litovits TL, Klein-Schwartz W, Odera GM: Ipecac administration in children younger than 1 year. Pediatr 75:761, 1985
30. Gaudreault P, McCormick MA, Lacouture PG, et al: Poisoning exposures and use of ipecac in children less than 1 year old. Ann Emerg Med 15:808, 1986
31. Mofenson HC, Caraccio TR, Greensher J: Acute poisonings. P. 944. In Rakel R (ed): Conn's Current Therapy—1987. WB Saunders, Philadelphia, 1987
32. Dean BS, Krenzokok EP: Syrup of ipecac: 15 ml vs 30 ml. Clin Toxicol 23:165, 1985
33. Gaudreault P, Lewander WJ, Parent M, et al: Ipecac syrup; lack of dose response effect. Vet Hum Toxicol 26:403, 1984 (abst)
34. Meester WD: Emesis and lavage. Vet Hum Toxicol 22:225, 1980
35. Eisenga BH, Meester WD: Evaluation of the effect of motility on syrup of ipecac-induced emesis. Vet Hum Toxicol 20:462, 1978

36. Manno BR, Manno JE: Toxicology of ipecac; a review. Clin Toxicol 10:221, 1977
37. Gaudreault P, McCormick MA, Lacouture PG, et al: Poisoning exposures in children under 1 year; epidemiology and use of ipecac (abstract). Vet Hum Toxicol 26:411, 1984
38. MacLeod J: Hazards to health; ipecac intoxication use of a cardiac pacemaker in management. N Engl J Med 268:146, 1963
39. Robertson, WO: Syrup of ipecac associated fatality. A case report. Vet Hum Toxicol 21:87, 1979
40. Degenaro F, Nyhan WL: Salt: A dangerous antidote. Pediatr 78: 1048, 1971
41. Dabbous IA, Bergman AB, Robertson WO: The ineffectiveness of mechanically induced vomiting. J Pediatr 66:952, 1965
42. Penner RE; Acid ingestion; toxicology and treatment. Ann Emerg Med 7:374, 1980
43. Rumack BH: Poisoning; prevention of absorption. p. 13. In Bayer MJ, Rumack BH (eds): Poisoning and Overdose, Aspen Systems, Rockville, MD, 1983
44. Peterson CD: Electrolyte depletion following emergency stomach evacuation. Am J Hosp Pharm 36:1366, 1979
45. Okonek S, Hofmann A, Henningsen B: Efficacy of gut lavage, hemodialysis and hemoperfusion in the therapy of paraquat and diquat intoxications. ARch Toxicol 36:43, 1976
46. Dans GR, Santa Ana CA, Morawski SG, et al: Development of a lavage solution associated with minimal water and electrolyte absorption or secretion. Gastroenterology 78:991, 1986
47. Cooney DO: A "superactive" charcoal for antidotal use in poisoning. Clin Toxicol 11:387, 1977
48. Chung DC, Murphy JE, Taylor TW: In-vivo comparison of absorptive capacity of "superactive charcoal" and fructose with activated charcoal and fructose. J Toxicol Clin Toxicol 19:219, 1982
49. Renzi FP, Donovan JW, Morgan L: Concomitant use of activated charcoal and n-acetylcystine. Ann Emerg Med 13:136, 1984
50. Minocha A, Herold DA, Barth JT: Activated charcoal in oral ethanol absorption; lack of effect in humans. Clin Toxicol 24:225, 1986
51. Curtis RE, Barone J, Giacona N: Efficacy of ipecac and activated charcoal/cathartic: Prevention of salicylate absorption in a simulated overdose. Arch Intern Med 144:48, 1984
52. Neuvonen PJ, Olkkola KT: Activated charcoal and syrup of ipecac in the prevention of cimetidine and pindolol absorption in man after administration of metoclopramide as an antiemetic. Clin Toxicol 22:103, 1984
53. Kulig, K, Bar-Or D. Cantrill SV, et al: Management of acutely poisoned patients without gastric emptying. Ann Emerg Med 14:562, 1985
54. Easom JM, Caraccio TR, Lovejoy FH: Evaluation of activated charcoal and magnesium citrate in the prevention of aspirin absorption in humans. Clin Pharm 1:154, 1982
55. Burton BT, Bayer MJ, Barron L, et al: Comparison of activated charcoal and gastric lavage in the prevention of aspirin absorption. J Emerg Med 1:411, 1984
56. Krenzelok EP, Freedman GE, Pasternak S: Preserving the emetic effect of syrup of ipecac with concurrent activated charcoal administration: A preliminary study. Clin Toxicol 24:159, 1986
57. Freedman GE, Pasternak S, Krenzelok EP: A clinical trial using syrup of ipecac and activated charcoal concurrently. Ann Emerg Med 16:164, 1987

58. Cupit GC, Temple AR: Gastointestinal decontamination in the management of the poisoned patient. Emerg Med Clin North Am 2:15, 1984
59. Krenzelok EP: Comparison of cathartics used with activated charcoal Vet Hum Toxicol 26:402, 1984
60. Flores F, Battle WS: Intestinal obstruction secondary to activated charcoal. Contemp Surg 30:57, 1987
61. de Nere R: Antidotal efficacy of activated charcoal in the presence of jam, starch and milk. Am J Hosp Pharm 33:965, 1976
62. Navarro RP, Navarro KP, Krenzelok EP: The relative efficacy and palatability of three activated charcoal mixtures. Vet Hum Toxicol 22: 163, 1980
63. Pollach MM, Dunbar BS, Holdbrook J: Aspiration of activated charcoal. Ann Emerg Med 10:528, 1981
64. Dunbar BS, Pollock PR, Shavari MB: Cardiorespiratory changes after charcoal aspiration. Crit Care Med 9:221, 1981
65. Harsch HH: Aspiration of activated charcoal. N Engl J Med 315:318, 1986
66. Berg MJ, Berlinger WG, Goldberg MJ, et al: Acceleration of the clearance of phenobarbital by oral activated charcoal. N Engl J Med 307:676, 1982
67. Levy G: Gastrointestinal clearance of drugs with activated charcoal. N Engl J Med 307:676, 1982
68. Pond SM, Olson KR, Osterlak JD, et al: Randomized study of the treatment of phenobarbital overdose with repeated doses of activated charcoal. JAMA 253:3104, 1984
69. Radowski L. Park GD. Goldberg MJ: Model of theophylline overdose treatment with oral activated charcoal. Clin Pharmacol Ther 35:402, 1984
70. Weinberger M: Theophylline intoxication. Pediatrics 73:878, 1984
71. Pond SW, Jacobs M, Marks K, et al: Treatment of digitalis overdose with oral activated charcoal. Lancet 2:1177, 1982
72. Neurvonen PJ. Elonen E: The effect of activated charcoal on the absorption and elimination of phenobarbitone, carbamazepine and phenylbutazone in man. Eur J Clin Pharmacol 17:51, 1980
73. Neurvonen PJ: Clinical pharmacokinetics of oral activated charcoal in acute intoxications. Clin Pharmacokinet 7:465, 1982
74. Souich P, Gaille G, Larochelle P: Enhancement of nadolol eliminiation by activated charcoal. Clin Pharmacol Ther 5:585, 1983
75. Goldberg MJ, Berlinger WH: Treatment of phenobarbital overdose with activated charcoal. JAMA 247:2400, 1982
76. Reigert JR. Trammel LH Jr, Lindsey JM: Repetitive doses with activated charcoal in dapsone poisoning in a child. J Toxicol 19:1061, 1982
77. Guzzasrdi LJ: Role of the emergency physician in treatment of the poisoned patient. Emerg Med Clin North Am 2:3, 1984
78. Pirchioni AL, Chin L, Gillespie T: Evaluation of activated charcoal-sorbitol suspension as an antidote. J Toxicol Clin Toxicol 19:433, 1982
79. Mayersohn M, Perrier D, Pirchioni AL: Evaluation of sorbitol-charcoal mixture as an antidote for oral aspirin overdose. Clin Toxicol 11:561, 1977
80. Krenzelok EP, Keller R, Stewart RE: Gastrointestinal transit times of cathartics combined with charcoal. Ann Emerg Med 14:1152, 1985
81. Riegel JM, Becker CE: Use of cathartics in toxic ingestions. Ann Emerg Med 10:254, 1981
82. Krenzelok EP: Gastrointestinal transit times of cathartics used with activated charcoal. Clin Pharmacol 4:446, 1985

83. Farley TA: Severe hypernatremic dehydration after use of an activated charcoal-sorbitol suspension. J Pediatr 109:719, 1986
84. Prescott LF, Balahali-Moud M, Critchley JA: Diuresis or urinary alkalinization for salicylate poisoning. Br Med J 285:1383, 1982
85. Pond SM: Diuresis, dialysis, hemoperfusion—Indications and benefits. Emerg Med Clin N Am 2:29, 1984
86. Peterson RG, Peterson LN: Cleansing the blood; hemodialysis, peritoneal dialysis, exchange transfusion, charcoal hemoperfusion, forced diuresis. Pediatr Clin North Am 33:675, 1986
87. Reed CE, Driggs MF, Foote CC: Acute barbiturate intoxication: A study of 300 cases on a physiologic system of classification. Ann Intern Med 37:290, 1952

INDEX

Page numbers followed by *f* refer to figures; those followed by *t* refer to tables.

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