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Laboratory data, scientific facts proven by modern statistical methods, and clinical observations resulting in "feelings" of appropriate therapy are necessary for the physician to provide each patient with the most effective treatment. It is important, however, that the physician realize how much fact and how much feeling influences his therapies. The pediatrician does manage by means of a mixture of "feelings and facts" many of the common childcare problems he sees in his office. This symposium examines a few of these problems: Respiratory distress in the newborn infant, Septicemia in the newborn infant, Congenital heart disease (a review of the major series revealing familial incidence), Otitis media (management of acute infection in the middle ear), Genitourinary infection, enuresis, Asthma (management of the child who wheezes), Goiter (diagnosis and treatment of goiter in children), Abdominal pain (management of the "tummy ache" in childhood), Poisonings (management of the child who is poisoned), and Drug abuse.

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ENURESIS						
ASTHMA (in children)						
GOITER (in children)						
ABDOMINAL PAIN (in children)						
POISONINGS (in children)						
DRUG ABUSE						

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PRESENT CONCEPTS IN INTERNAL MEDICINE

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Study to shew thyself approved unto God,

a workman that needeth not to be ashamed,

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II Timothy 2:15

PRESENT CONCEPTS IN INTERNAL MEDICINE
VOLUME IV *January 1971* Number 1

PEDIATRIC SYMPOSIUM

Management of Common Childcare Problems
Facts and Feelings

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FOREWORD

As one reads the history of medicine he is impressed with the amount of "feeling" and "art" used in treatment of patients and their diseases, and the lack of basic scientific facts to support many medical therapies. The use of blood-letting for pneumonia, foxes' lungs for asthma, apple cider vinegar for obesity, honey for enuresis are only a few of the therapies we smile about now and seldom do we wonder how hilariously ridiculous our therapies may appear a hundred years from now.

In this scientific age when everything should be proven by facts and figures, and the treatment of a patient fitted to a "*p*" value, the importance of clinical observations and feelings have been given second place. Medical meetings and journals are divided into clinical and scientific sections. Does a physician have to be one or the other — *either* a scientist *or* a healer? Is bedside art and observation to be replaced by the science of the laboratory? A thousand times "*NO*" must be our answer. Good critical observations on sick people arranged in a logical sequence create an experiment in management which can be reproduced in the management of a new patient.

Laboratory data, scientific facts proven by modern statistical methods, and clinical observations resulting in "feelings" of appropriate therapy are necessary for the physician to provide each patient with the most effective treatment. It is important, however, that the physician realize how much fact and how much feeling influences his therapies. The pediatrician does manage by means of a mixture of "feelings and facts" many of the common childcare problems he sees in his office. This symposium examines a few of these problems.

The physician is aware also that some terms are interpreted differently by his colleagues; for example "respiratory distress" is a term defined only in the mind of the user. Is it a useful term or should it be discarded? "Sepsis" is another term with obscure meanings to various users. Infection

as a cause of mortality is much less in children than in previous years; however, in the newborn infant there may be no difference.

Among the childcare problems which face the physician in the office and the clinic are congenital heart disease, middle ear infection, genitourinary infection, and enuresis. Genetic counseling is sought by parents more and more frequently. What is the chance of a second child's having congenital heart disease? Will this child's children have congenital heart disease? . . . Infections continue to be common in children. It is almost impossible to determine the correct therapy for middle ear infection if one tries to find the answer solely from reading. To treat this condition there are some facts and much feeling and a lot of guessing. Even the diagnostic criteria vary. . . Chronic renal disease is another subject on which noncontradictory facts are hard to find. It is recognized that chronic renal disease in adults may have started as a genitourinary infection in childhood. It is imperative that we know when to treat a child presenting with symptoms of this infection, with what to treat him, and for how long. . . And then enuresis — Ever since children were made there have been bed-wetters. The reader will be fascinated by the stories of treatment of this problem over the period of 3400 years. Every age has contributed its humorous therapies. It is interesting to note that our electronic age has offered expensive, bell-ringing devices as its contribution (— and they are useless too!).

The pediatrician is also concerned about the problems of the child with asthma, goiter, abdominal pain, poisoning and drug abuse. Asthma has been and is treated with almost any and all drugs the physicians can get into the patient. Why are so many needed? — to treat the patient or to make the doctor and parent feel something is being done? . . . One of the most common endocrinologic disorders in children is goiter. Young girls do not like lumps in their necks. These lumps do not contribute to their beauty. The anxiety of parent and child must not influence physicians to neglect proper diagnosis and treatment. . . A "stomach ache" at some time is a part of growing up. The child with abdominal pain should trigger the physician's mental computer to run rapidly through a differential diagnosis and to decide the therapy to be used immediately. . . The fashion of the age appears to be the experience of ingesting chemicals foreign to the body — sometimes accidentally, sometimes purposefully, and sometimes to follow a doctor's

advice. No matter what the cause, poisoning and drug abuse are probably the most serious and biggest problems in the preadult population.

The pediatric residents and staff have taken an honest look at the "state of the art" of these everyday childcare problems.

LTC JAMES L. STEWART, JR., MC
Guest Editor

". . .But no one today is so vain as to regard himself as infallible. And modern science, which can never be too rigorous in method and acid tests, is tolerant of concrete conclusions, if based upon sound principles and demonstrable fact. There is, therefore, no longer any dogmatic or doctrinal method of healing, but anything is admissible or justifiable if based upon methodically ascertained facts or, failing these, upon conscientious consideration of reality. . .

. . . The future aim of medicine is that of any other science and identical with that of medicine at all times: It is the task of seeking and finding the truth, whatever and wherever it is and by whatsoever ways it may be found."

-- WUNDERLICH, Stuttgart, 1859
Translated by A.Allemann, and published in part in Garrison's *History of Medicine, 4th edition*

EDITORIAL

Edward B. Shaw, M.D.
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The pediatric staff contributions to *Present Concepts in Internal Medicine* deal with common problems in childcare medicine; and except for two problems in the newborn nursery, they are concerned with ambulatory care in the outpatient department, emergency room or the physician's office. A large proportion of childhood problems are suitably managed on an ambulatory basis, so that patients no longer are admitted to the hospital as a matter of convenience. Only the very ill, surgical cases, or the most difficult and unusual illnesses require hospitalization.

Childhood illnesses are not trivial, and expert care may avert serious damage in later years. Otitis can lead to chronic difficulties and complications. Urinary tract infection can involve more than temporary treatment and may extend to adult years. Poisoning requires skill in diagnosis and prompt effective treatment.

Ambulatory care has become the heart of modern pediatrics; it offers opportunities for repeated examination and re-evaluation of the child patient which can lead to discovery of conditions requiring study in depth. The pediatrician is the child's physician and this repeated contact with the child leads to trust and cooperation, which is one of the most rewarding components of pediatric practice. While the pediatrician counsels the parents, his most important service is directly to the child.

Only in fairly recent times have pediatricians become involved in problems

of the newborn in the delivery room and the first few days in the nursery; formerly this period was delegated to the obstetrician. Now improved methodology and increased interest and concern have made the pediatrician the senior member of the neonatology team. Enormous effort and expense have been devoted to the care of the elderly who may have only a short reprieve whereas the baby may have a threescore-and-ten survival. Prevention of neonatal death, however, is not the greatest concern; more important is the prevention of damage to the quality of the baby's life for his future years.

There may be question as to whether or not an army hospital is well-designed for residency training in pediatrics. However, the large number of dependents provide an adequate case load, the excellent professional and teaching qualifications of recent and present chiefs of service and carefully selected house staff, make this service one which rivals that of many university departments. Recently, an opportunity has been provided for residency rotation in foreign countries where there is an incomparable opportunity for experience with exotic diseases. This is a training maneuver which only the Armed Services can provide and adds greatly to the strength of this program.

RESPIRATORY DISTRESS IN THE NEWBORN INFANT

LTC Donald W. Thibeault, MC

The term respiratory distress is frequently employed by textbooks, scientific journals, and pediatricians to describe disturbance of respiration in newborn infants. The term is routinely applied to certain disturbances of respiration in neonates. Therefore, it must convey a clinical picture and should be definable. After searching extensively for a number of years, I have not found a precise definition. It is the purpose of this discussion to enumerate the tangible features of respiratory distress and propose some questions which may lead to an objective definition.

Respiratory distress implies to this author a subjective sensation which is a symptom whereas, the clinical usage refers to an observation or sign. Respiratory distress may or may not be distressful to an infant. Respiratory difficulty, labored breathing, or dyspnea are all subjective sensations or symptoms. Respiratory distress is also linked with the idiopathic respiratory distress syndrome, an entity or group of entities which are difficult to define.

Respiratory distress imparts to a physician a mental picture of an infant with a respiratory abnormality. Answers to such questions as (1) Which sign or signs must be apparent so that all physicians would agree that respiratory distress is present? and (2) Would an infant with a respiratory rate of 15/minute or with severe recurrent apneic spells or with cyanosis be considered as having respiratory distress? would help to establish a base for a definition. All reports agree that the presence of certain abnormal movements of the respiratory system is an indication for using the term respiratory distress. Apnea is

excluded because no movements are involved and, importantly and similarly to respiratory rate, it can be accurately quantitated. The majority of rapid respiratory rate (i.e., greater than 50 per minute) will be associated with abnormal respiratory movements. A second sign that universally invokes the term respiratory distress is an abnormal noise emanating from the respiratory system. These noises and movements are signs, and, therefore, objective and together they constitute the term respiratory distress. TABLE I. If these two observations were the only factors involved, the term respiratory distress would have no practical value and probably would have perished long ago because of disuse atrophy. Respiratory distress appears to enjoy longevity because of its qualification - mild, moderate or severe. This objectivity is replaced by subjectivity because no two people will regularly agree on the severity of respiratory distress. The three quantitating subjective factors relate to the work of breathing and the energy the patient appears to expend with each respiration. The only precise method to assess the work of breathing is to measure it with sophisticated pulmonary equipment. However, the majority of physicians would agree that severe retractions are associated with severe respiratory distress.

TABLE I

SIGNS WHICH CONSTITUTE THE TERM RESPIRATORY DISTRESS

Abnormal respiratory movements

- Inspiratory nares dilatation
- Suprasternal notch retractions
- Intercostal, subcostal and retrosternal retractions
- Irregular or jerky inspirations
- Prolonged expiratory phase

Abnormal respiratory sounds

- Inspiratory and expiratory stridor
- Expiratory grunt
- Nasal noises

Can respiratory distress be quantitated or scored clinically similarly to the Apgar score? If so, would it be a valuable tool?

The Apgar scoring system derives its score and singular importance in clinical medicine by assessing many systems including the cardiovascular, pulmonary, neuromuscular; however, it is for the most part related to a single etiological factor - hypoxia in utero. There are obvious exceptions to this, such as central nervous system sedation or damage. Respiratory distress would be scored on the performance of a single system; i.e., the respiratory system. Respiratory distress has multiple etiologies such as heart failure, hypotension, obstructive lung disease, intracranial disease. Thus, the presence of respiratory distress tells nothing about the etiology. The presence of respiratory distress dictates an investigation of respiratory function and the first order of business is to quantitate overall function by measuring the arterial blood gases and the arterial pH. Following this assessment, the system is supported, if needed, and then the etiology of respiratory distress is ferreted out.

Respiratory distress in premature infants has been scored. It is called the "retraction score" /1/ and is based on retractions and grunting respirations. This invites further questions: (1) Could this scoring system or any other scoring system relate to overall pulmonary function? and (2) Is there a good correlation between arterial blood gases and respiratory distress? It is known that retractions are closely related to the work of breathing in certain conditions, such as hyaline membrane disease./2/ The work of breathing at best is only distally related to arterial oxygen tension; therefore, it is most unlikely that a score of respiratory distress could estimate the arterial oxygen tension. Also, an infant may have severe retractions and then if the arterial carbon dioxide tension rises he can become sedated and the retraction actually decrease in severity. Under this set of circumstances, a retraction score will be actually misleading.

The term respiratory distress is partly objective and partly subjective. It adds little to the clinical history. The central drawback is the lack of correlation between pulmonary function and blood gas exchange and the clinical quantitation.

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SEPTICEMIA IN THE NEWBORN INFANT

CPT Stephen L. Done, MC

Septicemia of the newborn may describe three overlapping pathologic states. The first and broadest use includes any toxic products in the blood from any cause, most of which are derived from some type of infection. A second and more restrictive usage refers only to actual bacterial invasion of the blood which would be accompanied by a positive blood culture. The third and most restrictive usage specifies only that group of infections of the blood acquired during or shortly after birth via routes or portals of entry peculiar to the newborn. Bacterial invasion of the blood may occur in the course of any infection in the newborn. Viremias and fungemias, although part of the differential diagnosis of septicemia, will not be specifically considered in this paper.

Incidence and Mortality

Neonatal mortality has decreased significantly since 1935 from 56/1000 livebirths to less than 20/1000 livebirths and 100/1000 premature births. The period 1950-1965 has not realized as great a decline in neonatal mortality as in previous decades./1/ Infection as a cause of death has remained almost static since 1950, when it was found on serial autopsy data to account for between 10-25 percent of deaths./2-4/

The incidence of neonatal septicemia has remained fairly constant over the past 40 years. It has ranged from 1/500 to 1/1600 in livebirths /5,6/ and from 32/1000 to 71/1000 in premature births./2/ The mortality rate of neonatal septicemia as reported by Dunham /7/ in 1933 was 87 percent, by Nyhan and Fousek /8/ in 1958 was 72 percent (mortality due to group A streptococci declined from 59 percent to 34 percent during the period of study); and by Silverman and Homan /9/ in 1949 was 56 percent. McCracken and Shinefield /6/ reported 43.5 percent in 1966 and Gluck, et al/5/ reported 44 percent mortality.

Etiology of Septicemia and Identification of the High Risk Infant

Perhaps the most striking change seen in the disease is the change in etiologic agents over the past 50 years. More and more of the gram negative organisms, the so-called "water bugs" and opportunistic invaders are emerging as the significant organisms in neonatal speticemia./2010/ The common organisms, according to Dunham /7/, were streptococcus and staphylococcus in 67 percent and E. coli in 25 percent of cases reported. Staphylococcus, a cause of much disease during the 1950s /6/ has disappeared as a causative organism in a recent series./10

The emergence of pseudomonas as a cause of neonatal septicemia has been dramatic and reasons for this have been suggested by Hoffman and Finberg./11/ Other opportunists, such as Flavobacterium, Achromobacter and Serratia, have been the causes of microepidemics especially in debilitated infants. There has been a dramatic change from the gram positive organisms to the gram negative organisms as causes of neonatal septicemia.

The reasons for the changing picture of organisms is not apparent but may be in part due to widespread use of antibiotic agents and selection of more resistant organisms, or changes in resident flora. Intrinsic virulence or invasiveness of the bacterial species themselves, changes in the host defenses, or prolongation of life in otherwise debilitated infants who are thus susceptible to opportunist invasion may also have their effect on the changing pattern.

The high mortality rate and usually rapid course of the disease especially in premature infants necessitates early recognition of the potentially infected and the infected infant as well as prompt therapy.

The relationship of obstetrical factors to septemia is a controversial subject./12-16/ In the intrauterine environment the fetus enjoys relative isolation from infection. Most antenatally acquired infections result from hematogenous spread of bacteria, viruses, fungi or protozoa. The protective villous surface of the placenta has been greatly overrated and it is probable that most microorganisms in the maternal circulation can pass the placenta./12,13/ Pryles, et al/14/ was unable to

demonstrate a correlation between positive intrapartum blood cultures and subsequent neonatal septicemia.

Most intrapartum infection is acquired by the ascent of bacteria from the vagina. While the membranes are intact the infant is protected but in cases of premature rupture of the membranes and prolonged latent phase the infant is at increased risk of infection./14/ This type of infection is usually orificial in nature and the lungs and gastrointestinal tract tend to be most commonly involved./12,13/ Septicemia also appears to be correlated with prolonged rupture of the membranes./14/ At even higher risk probably is the infant delivered of a febrile mother especially if her infection is bacterial./14/ Foul smelling purulent amniotic fluid, meconium staining and severe dpression requiring intubation and resuscitation of the infant may also have positive correlation. Infants with respiratory distress, those requiring surgical procedures and those receiving exchange transfusions may be considered at extra risk./5/ The high incidence of septicemia in premature infants, places them at high risk and vigilance is required in the care of these infants./2/

THE CLINICAL PICTURE

Septicemia in the newborn and particularly the premature has a subtle clinical onset and a rapid downhill course. Early diagnosis is of utmost importance and the best single tool of the clinician is a high degree of suspicion aided by a history suggesting an infant at high risk.

One fact which has not been adequately explained is the predominance of males in all series. Other clinical features are highly variable./5/ Attempts have been made to correlate the clinical picture and the offending organism but little success has been achieved. Factors which appear to be correlated are: (1) time of onset of symptoms - within the first 48 hours gram negative organisms are the most common cause whereas after 48 hours nursery acquired organisms, such as staphylococcus and pseudomonas, are more common although the division is not distinct /2,5/; and (2) skin lesions which may suggest the invading organism - erysipelas is seen only in patients with streptococcosis, abscesses suggest staphylococcus, and erythema gangrenosum is associated with infections due to pseudomonas.

The common signs are presented in TABLE I. The earliest signs may be decreased activity, feeding disturbances, vasomotor instability, mild change in body temperature despite constant environmental temperature; also apnea, jaundice, and abdominal distention may be early presenting symptoms. Late signs of toxicity may include pallor, grunting, dyspneic respirations, mottling, cyanosis, vascular collapse, bradycardia, and respiratory arrest.

TABLE I

COMMON SIGNS OF SEPTICEMIA IN THE NEWBORN

General	Prematurity Disturbances of body temperature (particularly premature infants) Decreased activity Feeding Disturbances
Respiratory system	Retractions Tachypnea Periodic breathing Apnea Cyanosis
Circulatory system	Vasomotor instability Pallor Cold clammy skin Signs of shock
Gastrointestinal system	Abdominal distention Vomiting Diarrhea Hepatomegaly
Central Nervous system	Irritability, *or* sedation Convulsions Full fontanelle
Hematopoetic system	Jaundice Splenomegaly Purpura Bleeding

Associated focal infections may be present which either antedate or postdate the septicemia: (1) meningitis - may occur in 12-40 percent of infants with septicemia; (2) thoracic involvement - may occur in some forms; i.e., pneumonia, lung abscess, empyema in as many as 33 percent of patients; (3) urinary tract infections (including pyelonephritis) in 7-49 percent of patients; and a variety of others - omphalitis; conjunctivitis; abscesses; impetigo; erysipelas; otitis media; septic arthritis; osteomyelitis; peritonitis; vaginitis; parotitis; pericarditis; infected omphalocele, encephalocele.

There is no single sign or symptom or pattern of symptoms that will in all cases be found in the infant with septicemia; -- herein lies the difficulty of diagnosis.

Because of the rapid course to termination that is often seen it is necessary most of the time to initiate therapy at the slightest sign of septicemia. Before any therapy is begun a thorough search for the causative organism must be made lest a difficult clinical entity become hopelessly confused because of improper therapy.

A complete review of the natal history and the mother's postpartum course including any organisms cultured from her is invaluable. A review of the infant's course and exposure to contaminants may elucidate other possible sources of infection.

In addition to attempting to verify the signs of septicemia, a search for localized areas of infection should be made and suitable cultures taken. In obtaining cultures (since therapy will often be initiated before culture results are back) attempts should be made to obtain body fluids which permit staining and visualization of the organism. Meningitis and pyelonephritis are common localizations of infection and cerebrospinal fluid (CSF) and urine lend themselves well to characterization of infecting organisms. Seldom are any signs of meningitis given in the newborn, i.e., full fontanelle or rigid neck, but their absence should not deter one from performing a lumbar puncture./17/

Roentgenographic studies of the chest and abdomen may give helpful clues suggesting localization of infection. Nasopharyngeal cultures has been said to give better correlation to possible organisms causing pneumonia than throat cultures.

Of course the sine qui non of septicemia is bacterial growth in the blood. Frequently, cord blood cultures may give false positive results and better samples may be taken aseptically from a peripheral vein or artery. Cultures of CSF and urine should always be made. CSF sugar will be low in bacterial infections but its usefulness is decreased because of hypoglycemia in the newborn. Therefore blood sugar studies should also be done. A complete blood count (CBC) may have some value in the diagnosis. Hemoglobin and hematocrit values are often decreased. Peripheral blood leukocyte counts below 5,000 cc suggests a poor prognosis./6,8/ Leukocytosis greater than 30,000 supports a diagnosis of infection.

The value of umbilical cord histology has been controversial /12,14,15/ but probably helps in identifying the infant at risk. The presence of chorioamnionitis and positive cord blood culture and a febrile mother may suggest risk but one can still await symptoms to begin therapy. One may culture the child at birth.

Infants were once thought to be immunologically incompetent but this probably not true. Several investigators /5,9/ show that premature infants have impairment of phagocytosis. The 20-week fetus can make IgG and IgM in response to infection /18/ and IgM may be elevated in the cord blood in congenitally acquired infection./19/ Placental transport of IgG gives the infant a full complement of these antibodies which are present in the maternal circulation but no transport of IgM or IgA exists. Levels in the cord blood for these immunoglobulins are low or absent. Since infants are able to make IgM in response to infection, serial determinations have been followed in an attempt to recognize the infected infant before he is clinically apparent. Such efforts have not been helpful because symptoms are usually present before IgM begins to rise./18,20,21/

MANAGEMENT

When the presumptive diagnosis of septicemia is made the decision to treat is usually made without an etiologic diagnosis or at best only a gram negative or gram positive organism as identified on smear. Septicemia acquired within the first 48-72 hours of life is usually an intrapartum acquired infection

and includes maternal flora of the genitourinary and intestinal tracts such as E. coli, Enterococcus, and Streptococcus. Infections acquired beyond the immediate postpartum period are usually nursery acquired and predominant nursery organisms are likely offenders such as E. coli including enteropathic, S. aureus, Pseudomonas, and various opportunists.

Because of rapid course, high mortality and decreased host defenses, bactericidal drugs rather than bacteriostatic agents should be used and parenteral routes are generally preferable to oral routes./22/

In septicemia of early onset (before 72 hours) and of clinically undefined etiology broad spectrum antibiotics covering the most likely organisms should be used. Usually kanamycin /23/ in a dose 15 mg/kg/day intramuscularly in two divided doses, and penicillin /24/ 100,000 units/kg/day intramuscularly or intravenously in two divided doses, or ampicillin 100-200 mg/kg/day intravenously or intramuscularly in two divided doses will give the necessary coverage. Ampicillin and penicillin are given every 12 hours because of slightly decreased renal clearance initially. After the infant is five days of age, they are given every 6-8 hours. Kanamycin is effective against E. coli, Klebsiella-Aerobacter and other susceptible gram negative organisms while penicillin is effective against all gram positive organisms. Ampicillin gives coverage against Salmonella, H. influenzae and Proteus mirabilis.

For septicemia of later onset where penicillin resistant staphyococcus is suspected one may elect to use the same agents or choose methicillin 200 mg/kg/day intravenously or intramuscularly in two divided doses or on a 6-8 hours schedule if the infant is over five days old. If pseudomonas is suspected then polymyxin-B sulfate 4 mg/kg/day intramuscularly or intravenously in two divided doses or colistimethate sodium 8 mg/kg/day intramuscularly in two divided doses can be given./24/ If pseudomonas meningitis is present then intrathecal administration of polymyxin-B 1.0 mg in 1.0 ml of normal saline is given daily./25/

The polymyxins and kanamycin are both neurotoxic and nephrotoxic and care should be used in their administration. The toxicity of kanamycin is cumulative and 500 mg/kg total is probably the maximum tolerable dose.* Gentamycin and carbenicillin are effective against Pseudomonas and have been used in the

*Grossman, M. Personal communication.

newborn but dosages are not yet clearly worked out.

General supportive care and careful monitoring of vital signs and blood sugar are invaluable in the treatment of the seriously ill infant. Usually oral feedings are interdicted while vital signs are unstable. Intravenous fluid and electrolyte therapy is necessary.

Complications, such as the appearance of shock and vascular disturbances, may arise and demand therapy. Apparent Shock. Endotoxemia has not been documented in the neonate but clinical shock may occur suddenly in the course of gram negative septicemia. Circulatory support in the form of 10-20 cc of whole blood/kg of body weight should be given. Oxygen and ventilatory support, sodium bicarbonate 2-3 mEq/kg and glucose 1-2 ml of 50 percent/kg should be used as needed for respiratory support and hypoxemia and acidosis. Corticosteroids or additional circulatory support with continuous intravenous infusion of isoproterenol although useful in adults are not of proven worth. Vascular disturbances. Disseminated intervascular coagulation has been seen in a number of fulminant bacterial and nonbacterial infections in the newborn /26-28/ and results in an in vivo activation of the clotting mechanism with a resultant hemorrhagic diathesis./29/ Diagnosis is by demonstration of decreased consumable coagulation factors V, VIII, prothrombin and fibrinogen and also demonstration of fibrin split products in the blood. Its incidence as a complication of septicemia in the newborn is unknown. Hypotension, shock and stasis have some effect on the development of this state and it is probably improved by improvement of these factors. Heparin 1.0 mg/kg every four hours prevents further consumption of coagulation factors and improves the clotting defect but does not effect the mortality./30/

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CONGENITAL HEART DISEASE
A Review of the Major Series Revealing Familial Incidence

MAJ Frederick W. James, MC

Our understanding of the genetic patterns underlying isolated cardiac manifestations in "normal" subjects is limited. Our understanding of cardiac malformations in known heritable disorders is more advanced. The information of the cardiac malformations associated with many recognized heritable disorders has been summarized by McKusick./1/ Several reports are available regarding incidence of congenital heart disease in families. The majority of these reports are retrospective. Consequently, their results must be considered as gross estimates. Familial incidence have been obtained by two major approaches: (1) recording incidence of congenital heart disease in live-born siblings and (2) getting a detail analysis of individual families.

This paper will review briefly the results of major series involving familial incidence of congenital heart diseases and will try to provide useful information which the physician may use to counsel the family. With the advancement in treatment and management of these problems, family counseling has become an important consideration for the cardiologist and pediatrician.

Incidence in Siblings, Parents and Offspring of Probands

The incidence of congenital heart disease in the general population is 0.6-0.8 percent. Groups of 431 probands (McKeown et al/2/), 1227 (Campbell et al/3/), 1,188 (Lamy et al/4/) and 1,000 (Neil/5/) have a 1.4-2.2 percent incidence of congenital heart disease in liveborn siblings. This incidence is approximately three times greater than the incidence in the general population. Campbell /3/ observed complete concordance in 56 percent, partial (ventricular septal defect (VSD) in one sibling, and tetralogy of Fallot in another) in 22 percent and discordance in 22 percent. The parents of probands had an

incidence less than the general population according to the data collected in these series.

Regarding the offspring of probands, Neil and Swanson /6/ reported their study of 704 probands over the age of eighteen. Three-hundred and thirty-six of these were married and 508 pregnancies resulted from the marriages. A 20 percent incidence was observed in the first liveborn offspring. There appeared to be no significant difference in incidence between infants of either affected mothers and normal fathers or vice versa.

Nora et al. /7/ reported five affected children among 190 children of 73 parents with atrial septal defect (2.6 percent incidence in the offspring) and 6 affected children among 162 children of 57 parents with ventricular septal defect (3.7 percent). If one or both parents have congenital heart disease, the frequency of congenital heart disease in the first-born is at least three times greater than the general population.

Incidence of Specific Cardiac Defects

There is more information available on acyanotic lesions than cyanotic. Table I presents the risk of congenital heart disease with complete concordance in the firstborn child of at least one affected parent.

TABLE I*

RISK ON CONGENITAL HEART DISEASE WITH COMPLETE CONCORDANCE†

SEPTAL DEFECT	INCIDENCE General Population	INCIDENCE Firstborn	FREQUENCY
	Per One Thousand Births		
Ventricular	1.35 /8/ 1.75 /9/	37.0 /7/	21+ times
Atrial	0.7 /10/	26.0 /7/	37 times

*Additional data on incidence of specific cardiac malformations can be found in Neill's chapter *in* Moss and Adams' book *Heart Disease in Infants and Children and Adolescent.* /5/

†Firstborn child of at least one affected parent.

Family Studies

There are several family studies reported with two or more family members with congenital heart disease. Kahler et al/11/ reported three families in which two or more members had atrial septal defects of secundum and sinus venosus types and atrioventricular (AV) conduction disturbances were associated with the defects - a rare association. One member of a family had the AV conduction disturbance without evidence of cardiac disease. Therefore, it is suggested that familial atrial septal defect should be considered in a patient with secundum or sinus venosus type defects and AV conduction disturbance. Carleton et al/12/ reported on the data he had collected (1941-1958) from 41 families with two or more members affected with the same congenital heart disease. In 17 members, the lesion was a persistent ductus arteriosus.

Conduction abnormalities are reported in families. Familial congenital AV dissociation with ventricular capture in several members of one family was observed by Khorsandian et al./13/ There is extensive literature available on the familial occurrences of idiopathic hypertrophic subaortic stenosis. This lesion is believed to be transmitted as an autosomal dominance with a high degree of penetrance. Males are more severely affected than female. Monteleon and Fagan /14/ studied three generations of a family with four males member having mitral insufficiency and aortic insufficiency and two of the four had associated tricuspid insufficiency. A fifth male had only mitral insufficiency. The female family members were not affected. The mode of inheritance is suggested to be sex-linked.

There are many reports describing congenital heart disease in individual families. The inheritance patterns remain uncertain, but it is clear that the geneological picture is multifactorial inheritance rather than simple Mendelian inheritance.

SUMMARY

Congenital heart disease occurs in approximately 0.6-0.8 percent of general population. An increased occurrence is noted in families with at least one affected member. The risk of occurrence for the first offspring of affected mother or father is at least 2.0 percent. This risk is approximately three times the risk for the general population.

Probands studied by several investigators reveal an occurrence of cardiac defects in 1.4-2.2 percent of the live-born siblings. These probands have fewer pregnancies, more miscarriages, and deliver more stillbirths than a random group in the general population. In addition the offspring of the probands may not manifest the defect in infancy. These factors must be recognized, and when viewed in this perspective, the percentages of incidence probably are low. It is agreed that congenital heart disease does recur in families. More attention should be directed towards obtaining a careful family history which can provide information that is helpful towards diagnosis and family counseling. More prospective studies with long-term follow-up are needed so as to provide more accurate statistical information on incidences of congenital heart disease in families that have one or more affected members.

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A symposium in *Pediatric Cardiology* is programmed for Friday and Saturday, 12-13 March 1971, Letterman General Hospital. Additional information and reservations may be made by calling 415-561-4268 (Pediatric Service) or 415-561-4275/4276 (Department of Medicine).

OTITIS MEDIA
Management of Acute Infection of the Middle Ear

MAJ Henry C. Reister III, MC

Acute infection of the middle ear is common in the infant and preschool child./1,2/ There is a predominance of this condition occurring in this age group, as compared with other age groups, and the reasons are probably multifactorial. A generalized susceptibility to infections may result from limited immunological experience. Small children may also be disposed to ear infections because of greater patency, shorter length, and relatively horizontal course of the eustachian tube./3/ Moreover, they spend a relatively greater portion of day in a recumbent posture. Some children are easily distinguished as more disposed to otitis than their peers, and in this setting some attention should be given to a variety of additional factors. Environmental humidity below optimal levels may set the stage for superinfection. Similarly, environmental irritants, such as dust, lint, industrial, vehicular, or tobacco fumes, could play a role. Allergy may be predisposing in those children with chronic, intermittent, or seasonally impaired eustachian flow secondary to nasal membranous congestion. Lymphoid hyperplasia, most common in the toddler and schoolage child, may play some role. Rarely one may encounter a child whose infections are due to compromised immunological or cellular defenses. The incidence of infections declines sharply late in the first decade./4/

The attack rate and actual total incidence of acute middle ear infection is not known. One study suggests that one in 15 children below school age will have at least one attack per year. There are no data to show that infections are more or less common at this time in comparison with any past period. It does appear that the complication rate in terms of such surgical problems as mastoiditis and brain abscess has declined in recent decades.

Much of what is written about acute middle ear infections has had serious scientific limitations because it has been based on more or less subjective observations. Diagnostic criteria, as well as criteria for assessing success in treatment, have depended mostly upon a clinicians appreciation of degrees of

"bulge", degrees of "erythema", degrees of "hyperemia", and degrees of "distortion" of "landmarks" and the "light reflex". In some studies credence factors have been reinforced by having more than one doctor agree on the diagnosis, but the degree to which this adds hard scientific data is moot. In short, there is no study in acute ear infection analagous to quantitative urine cultures in urinary tract infections. Nor is there a "pyotitis" to analogize with "pyuria". Nor a "bacteri-earia" to compare with asymptomatic "bacteriuria".

As observed in the past, a hyperemic phase has been described as the "beginning" in the course of acute middle ear infection./3,6/ This phase was followed by successive ones of exudation, suppuration, and coalescence. More pertinent than a description of each stage is recognition that the symptoms and the visible derangements of the ear vary markedly. There are no data presently available to adjust treatment depending upon the stage of infection when it is encountered - and none to show how the success of therapy relates to the stage at which it began. We can only surmise that each case which is put into a treatment series is somewhere beyond early hyperemia - and thus, identifiable as "infected".

Such recognized "infected" ears have been studied bacteriologically in many ways. Data now available point out some fallacies in previous beliefs and practices. Staphylococci were once accorded a major role in acute ear infections. It appears that this view arose from the now outmoded technique of swabbing material from the canal of the draining ear. It was once thought that throat cultures co-incident with acute ear infection might reveal the pathogen. This is a major fallacy./4,7/ It was hoped that perhaps deep nasopharyngeal (NP) cultures might relate well to the coincidental ear pathogens. Looked at critically the value of a NP culture is slight at best. Feingold, et al, /8/ was able to grow the ear pathogen from less than half of sampled noses, and in less than ten percent was the ear pathogen present in pure culture in the nose.

Presently one should view only a diagnostic aspiration of the middle ear itself as a relevant source of bacteriological data. At present one can assemble reports of thousands of such aspirations./6,9,10/ These reports vary in details but have an essential similarity which serves us well in selecting suitable treatment. Bacterial etiologies have been ascribed to the following percents of cases: 50 percent /7/, 53 percent /8/, 59 percent /11/, 72 percent /6/. There are a group of miscellaneous

organisms in every study. In all studies the most common pathogen is the pneumococcus which contributes between 30 - 50 percent of bacterial cases. Hemophilus influenzae (H. influenzae) contributes about 25 percent of cases in overall studies./11/ When age-adjusted, H. influenzae caused 37.5 percent of cases under age four and only 6.3 percent of cases after age four./8/ Streptococcus caused only 1.9 percent of cases in a series of very young children and has an inclining incidence in school age./12/ Bullous myringitis, often thought to be of viral origin, was associated with bacterial middle ear pathogens in two studied groups (5/9 cases) /8/, (10/10 cases) /12/.

The question remains: what causes the non-bacterial fraction of cases with apparent acute middle ear infection? No answer is available. Studies of aspirated fluids for viral and mycoplasmal agents have been unrewarding to date./13/ Clinical observations have certainly linked otitis and M. pneumoniae, but proof of involvement is absent./14/ Gram staining of aspirated middle ear fluid will correlate with 70 percent of positive cultures and in 25 percent of the culturally-positive exudates no bacteria will be seen./8/

At the present time diagnostic aspiration of the infected middle ear should not be a routine step. However, in any case where response to therapy is doubtful aspiration is indicated as a procedure for identifying such bacteria as resistant staphylococci or pseudomonas which would dictate unusual medications.

During the early hyperemic phase of acute middle ear infection hearing is normal. It declines as the ear goes on to an exudative, suppurative, or coalescent condition. About two-thirds of children with such infections have a loss of 15 decibels or more during the acute phases./15/ In 12 percent this level of disability persists for six months./15/ Neil, et al, /16/ found that the average time to return to fully normal audiometry was 23 months. There is little danger of permanent hearing disability after a single episode, but risk of such disability increases with repeated attacks. Incidence of defective hearing in populations with and without history of past otitis is very similar - running at about one to two percent./16/ Audiometry appears to have no relevant place in initial assessment but may be of value in long range follow-up.

The Clinical Picture

The clinical picture of acute middle ear infection is as variable as the pathological changes in the ear and the range of pathogens. Unfortunately, there are little data to associate a clinical presentation with any specific pathogen. Physicians are left to deal with "acute otitis" as though they were dealing with a single specific entity, which, of course, they are not. Many episodes follow a prodrome of several days' length which can not be differentiated from a simple viral upper respiratory infection. Fever is most often present but has no typical range. In one study mean temperatures of patients with sterile middle ear fluids were higher than those with bacterial pathogens./8/ Pain is generally thought to be present, but often it is not. As many as 75 percent have been found to be without any symptoms referable to the ear (pulling, poking, head banging, discharge, or verbal complaint)./12/ In another study 47 percent were free of ear complaints./1/ Discharge from the ear is not common. It occurred in about 15 percent of children with acute otitis in the pre-antibiotic era./6/ It appears less commonly now but no specific figure can be given. Pyrexia due to acute ear infection has triggered a great many "febrile convulsions". In this connection it is wise to remember that the bacterial spectrum of acute middle ear infection is highly similar to that of meningitis. Moreover, it is clear that otitis may seed the meninges. Therefore, any child presenting with a convulsion and a hot ear should have a lumbar puncture unless the physician has compelling evidence that there is no meningitis. There is no scientific data to link otitis with a specific "parenteral diarrhea". Nonetheless, diarrhea is not infrequently seen concurrently with acute middle ear infection. This could be due to pre-existing or co-existing viral infection.

Leukocytes counts are of no value in establishing the diagnosis and do not correlate with the type of pathogen involved./6/

TREATMENT

The treatment of the child with acute middle ear infection can be considered in terms of the aims of therapy.

Relief of Pain

Pain if present, can be approached by either analgesics or decompression. No data are available to ascertain the effectiveness of any measure in numerical terms. Analgesia may be in the form of aspirin by mouth, application of warm packs to the external ear, or installation of drops into the external ear. Aspirin not only relieves pain but provides relief of fever. External heat - such as hot water bottles or electrical pads - is effective but should be used with due caution to prevent external burns. Installation of drops into the ear has no proved value. Warmed drops as a vehicle for heat may be comforting. Medicated drops have been associated with a higher incidence of complications in at least one study./17/ Since pain is considered to be due primarily to pressure on either the distended drum or the inflamed otic mucoperiosteum or both, drainage is highly desirable. To achieve relief of pressure, the first measure to take is the application of nose drops. Neo-Synephrine nose drops ($\frac{1}{4}$ percent) in a quantity sufficient to bathe generously the nasal mucosa (generally 0.5 to 1.0 ml) should be put in the child's nasal passages when his head is hanging back off the table. This posture should be maintained for 30 to 60 seconds after installation so that the drops may reach the torus area. Application of a dose in the office by the physician serves not only to reduce pain on an immediate basis but to teach the parents the proper method as well. If this dose is effective in relieving pain, it can be repeated on a pro re nata (p.r.n.) basis. In the absence of acute pain nose drops are best avoided because of their tendency to cause a rebound congestion and their known ability to induce "rhinitis medicamentosa". Should the above listed measures fail to relieve pain the clinician's considerations may appropriately turn to myringotomy.

Myringotomy has been used less and less frequently over the past three decades. Critical studies have shown that its only use is in the relief of pain. It does not have any beneficial effect in reducing hearing loss or reducing late surgical complications/1,4,6/ Moreover, it is not without hazards. If required it should be performed by an experienced operator with a mummified infant or anesthetized child.

After relief of pain the parent will be most concerned about fever. This should be treated with aspirin. In addition the child should be spared over-dressing or an excessively hot

environment. There is no data to indicate benefit from sponging and alcohol baths.

Facilitation of Nasal Airway

Facilitation of normal nasal airway condition and function should have attention. The more serous the nasal discharge the more effective ciliary action can be and the more effective any mechanical clearing of the airway can be. Such fluidity of secretions can be enhanced by good hydration and by provisions of a cool vaporizer in the environment. A vaporizer may also serve a beneficial purpose in reducing airborne dust and lint. Cool vaporizers also have an antipyrexial effect. Statistical data to support these contentions can not be found. "Warm" vaporizers may be beneficial but may put the patient and his siblings in jeopardy of a scalding; all parents should be warned of this very real risk in use of heated vaporizers. Use of an oral agent to reduce nasal congestion is most often a part of the regimen suggested for otitis. This has theoretical advantage but in the only large study in which such an agent was tested in otitis it seemed to make no difference in course or outcome./1/ Until more data become available to demonstrate advantage or disadvantage oral decongestants will probably remain a part of the recommended regimen but on a basis of rather imprecise criteria at best.

Follow-up Visit

Follow-up is the next feature the clinician must consider. He must impress the parents at the initial visit with the necessity of having the child's ear looked at by a physician periodically until it is normal. This will usually entail several visits. Perhaps the first follow-up visit should be within 2-3 days. This visit ostensibly is scheduled to find out if the right treatment choices have been made. However, it may lead to false confidence. It is interesting that Bass, et al,/9/ found that ears finally judged treatment failures were not different from those successfully treated when seen at two and seven days. This leads to a conclusion that the most critical reinspection of the ear may be the one after the initial course of treatment is completed. Visits before that time may best be determined by the parents' assessment of the child's response.

Antibiotic Agents

Before the development of antibiotics about 97 percent of patients with acute middle ear infections got well without documented surgical sequelae./5,18/ After about a decade of experience with the use of antibiotics the rate of documented surgical sequelae was reduced twenty-fold (from 3.0 to 0.15 percent)./10,6/ A few studies have been undertaken since 1940 in which untreated controls have been included./20,5/ Recently, studies on comparisons of medications /9/ do not have untreated controls. In general, treatment comparisons "lump" together acute middle ear infections of all etiologies and, by convention, exclude those infections in persons with repetitive disease. It seems clear that we treat 100 percent of cases of acute middle ear infections with antibiotics so as to spare three percent from serious sequelae. Since we have absolutely no way to identify those who are at risk of these sequelae (mastoiditis, meningitis, labyrinthitis, brain abscess) the application of the antibiotics to all victims of otitis seems clearly advantageous.

The data which have emerged on antibiotic use are such that a wide variety of drugs must be considered effective in acute middle ear infection./1,4,6,9/ From birth to age four (at least) coverage should include an agent which counters Hemophilus. At all ages an agent should be able to counter pneumococcus. After the child reaches school age, streptococcus coverage is important. In teenage, mycoplasmal coverage may be of some value. Various drugs have been scored singly and in combination. None have been uniformly successful./11/ Most singles and combinations fall in at least 20 percent of cases./9/ In many studies the difference between the "best" and "worst" regimens is not significant./1/ Some agents offer the theoretical advantage of single drug competence for the whole spectrum./9/ Others offer less coverage but fewer side reactions. Each physician should decide what antibiotic drug and route is best for his own patient in light of his patient's condition, peculiar drug history, age, promise of compliance, and history of the drug.

COMMENT

The measures suggested in this paper will lead to clearing of the infection in the majority of cases. Patients who do not

respond to this management may need a diagnostic aspiration of the middle ear fluid to determine a better selection of antibiotic. Some will simply have failed to take their medicine for the proper time, at the proper dose, at the proper intervals; some may have taken the medicine and vomited it back up. These possibilities deserve consideration in any follow-up where success is elusive.

When failure to clear the ear is persistent or frequent relapses occur surgical and allergic considerations play a larger role in management, but there are no good data to show either the percent of cases in which these are involved or their susceptibility to the consultant's efforts.

SUMMARY

Acute middle ear infection is a common pediatric problem and its management is more "art" than "science". I have shared with you the best of what is known today about the condition and the care of the child. The following points should be remembered...

- → Pathogens involved are most often bacterial. Pneumococci are the most frequent cause in all age groups. In the preschool child, Hemophilus is a close second but plays diminishing role with advancing age. Streptococci are increasingly important with the start of school years.

- → The era of antibiotic usage has seen a twenty-fold decline in surgical complications of otitis media. Children with acute middle ear infection should receive an antibiotic suitable to their age and personal history. A wide variety of agents are useful.

- → Most important is follow-up until the ear is normal.

- → Middle ear aspirations are not routinely advised, but may be helpful in unresponsive cases.

- → Myringotomy has no role except in the relief of pain.

- → Other aspects of management have no statistical support.

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GENITOURINARY INFECTION

CPT Bruce L. Pfuetze, MC

Most authorities believe that chronic renal infection, which leads to early renal failure and premature death, often begins in the pediatric age group./1/ All too often the presence of smoldering renal infection is not diagnosed or is treated inadequately. The pathogenesis of this condition is poorly understood, therefore, prevention is difficult. Despite adequate therapy, recurrences are frequent and may progress to chronic renal damage if not adequately followed up./2/ Recent long-term studies have revealed information which provides the physician with a better understanding of urinary infection. This knowledge should help decrease the occurrence of progressive renal damage due to infection.

For the purpose of this discussion, urinary tract infection is defined as infection of any part of the urinary system. Urethritis and cystitis frequently occur without renal involvement. Pyelonephritis may occur by hematogenous spread, however, most cases of pyelonephritis probably occur secondary to ascending infection. Thus, management of both upper and lower urinary tract infections is important to prevent progressive renal damage. Infection in an uncontaminated specimen is defined as bacteriuria. Pyruia will be present in only 50 percent of urinary tract infections./1/ Pyruia may also be present in the absence of infection./1/ Therefore, pyuria cannot be used to diagnose infection. By using proper collection methods, bacteriuria should be used as the criteria for infection. Suprapubic aspiration or urethral catheterization specimens should be sterile in the absence of infection. These methods are usually not necessary because reliable information can often be obtained from a properly collected mid-stream clean catch specimen./3/ A colony count of greater than 100,000/ml pure culture is indication of infection. If the colony count is mixed or between 1,000/ml and 100,000/ml, the test should be repeated. If one is still unsure after repeat clean catch specimens, he should perform a suprapubic aspiration or catheterization.

Although some physicians might argue that bacteriuria itself should not be equated with infection, most authorities now agree that bacteriuria does indicate infection./4/ There are few people at this time who are willing to follow a group of asymptomatic bacteriuric children and look for progressive renal damage. Renal scarring has been noted to occur radiographically in children with asymptomatic bacteriuria./5/

CLINICAL FINDINGS AND PATHOGENESIS

Clinical findings in pediatric patients with urinary tract infection are variable and often neglected. In the infant gastrointestinal symptoms are common. Pyelonephritis in the newborn is often part of a more generalized picture of infection. In childhood the patient may have fever, irritability, vague abdominal complaints, anorexia, enuresis or other non-descript complaints. The older child more often presents with frequency, dysuria, urgency, fever, back or flank pain;however, he too may have obscure complaints. Kunin /2/ has demonstrated that many of these patients may be asymptomatic. This should point out the importance of considering urinary tract infection in almost any patient where the diagnosis is in question.

Considerable information has been obtained from laboratory studies; however, interpretation is difficult. Clinically, certain variables appear to have a close relationship with urinary tract infections and give support to various theories of the pathogenesis.

The difference in incidence between males and females needs to be considered in any theory of pathogenesis. Under three months of age, the incidence of urinary infection is approximately equal./6p8/ The male is especially predisposed during the first month. Although this may be due to the greater number of severe congenital urinary tract malformations in the male /6p32/ other factors could explain this difference. Male infants have an increased susceptability to infections in general. After the neonatal period, females predominate, which is often attributed to introduction of bacteria into the shorter female urethra. During the ages of highest contamination of the perineum (below age three) many investigators have noted an increased incidence of infection of females./7/ It has also been noted that Escherichia coli (E. coli) occurs as the

single infecting agent in approximately 80 percent of uncomplicated first urinary tract infection in females./8/ Serotype studies also revealed that the same organism was frequently noted in the feces at the time the urinary tract infection was noted. These factors give support to the "dirty perineum theory" - the close proximity of urethra to anus and the shorter urethra in the female predispose the urethra and subsequently the bladder to infection. Studies of periurethral contamination comparing various ages support this view./9/

The role of obstruction and stasis appear to be important in the incidence and perpetuation of infection. Obstruction may occur within the kidney, ureter or urethra. Experimental studies have demonstrated that micro-obstruction of renal tissue greatly increased the incidence of infection. Clinical and experimental studies support the view that obstruction and stasis predispose to urinary tract infection. Frequently, it is difficult to demonstrate obstruction - such as bladder neck obstruction. Smallpeice /10/ states that the majority of cases of urinary tract infection in girls are nonobstructive.

Vesicoureteral reflux according to most of the recent literature is abnormal./6p49/ It has a definite relationship to infecting of the urinary tract. Reflux is present in 30-50 percent of children with urinary tract infections. Most authorities now believe that vesical infection is usually the cause of the reflux./6p15/

There are many other factors which have a close relationship to infection. Calculi may cause obstruction or act as foreign bodies. Although there is a close relationship between calculi and infections, it is often difficult to determine which is the primary event. Any condition which damages the kidney has a strong association with infection - such as diabetes mellitus, hypokalemia, and renal papillary necrosis. The introduction of foreign bodies and instruments is related to an increased incidence of infection. If the urinary tract is normal, however, it is difficult to introduce with catheterization infection which will persist./11/

RECURRENCE

Management of urinary tract infections is complicated by frequent recurrence. According to Kunin /2/ "recurrence is the

single most important obstacle to be overcome if any preventive program is to be successful." He presented his data on a long-term study of the epidemiology and natural history of urinary tract infections./2/ This study was conducted over a 10-year period and includes follow-up of 156 girls with persistent bacteriuria - 126 white and 30 Negro. Most of these patients were treated with short term (two weeks) antibiotic therapy. Recurrences were detected on 321 occasions. E. coli was the most common organism found and persisted as the most frequent - accounting for 72 percent of recurrences. Other common organisms included Klebsiella-enterobacter (16.5 percent), Proteus (5.0 percent), Staphylococcus (5.0 percent), and Pseudomonas (1.2 percent).

Serologic studies of E. coli were done to determine if the recurrence was due to reinfection or persistence (recurrence of a supressed infection). If the same organism was recovered it was assumed that this was due to persistence. It is possible that many of the persistent cases may also have been reinfection with the same organism. Therefore, it may be concluded that recurrence is due to reinfection in the majority of cases treated with short term antibiotic therapy.

The relation of recurrence to time after treatment was evaluated. The first two years was the time at greatest risk after any course. In this study each treatment removed about 20-25 percent of white girls into long-term remission. This was noted to be true regardless of the number of treatments. Negro girls seemed to have less recurrence after each treatment - however, the group studied was much smaller. According to Kunin's study /2/ the number of girls requiring multiple treatments decreased with time and after several years only a small number of bacteriuric girls remained.

From statistical analysis of these data there appears to be a predictable nature of recurrence depending on certain variables. The percent remaining with recurrent infection seemed to be affected by the race or age of the individuals but was independent of vesicoureteral reflux or the socioeconomic status of the patients. This study demonstrated that rates of recurrence among previously bacteriuric girls, even after they had been free of infection for several years, were still much higher than the normal population. These data also point out that the longer a girl was free of infection, the less chance there was for her to have a recurrence. Marriage and pregnancy were much more likely to be associated with bacteriuria

and symptomatic urinary tract infection in girls who had previously been bacteriuric - even if they had been free of infection for years.

Urological Evaluation

Once a child has been proven to be infected it should be decided whether further urological studies are indicated. This is not usually a straight forward situation and frequently depends on whether close follow-up will be possible. Other factors such as age and sex are important. All males in the pediatric age group should have an intravenous pyelogram and a cystourethrogram following their first urinary tract infection. All girls with a second infection should have an intravenous pyelogram and cystourethrogram. Cystoscopy is also frequently indicated. Girls with their first urinary tract infection that present with clinical signs of pyelonephritis (fever, back or flank pain, abdominal discomfort) and urine analysis consistent with upper tract involvement also deserve at least intravenous pyelographic studies. In most cases these studies are done after the patient has been adequately treated. Occasionally further studies may be required by the urologist.

TREATMENT

If microscopically the patient is infected, he should be started on a sulfonamide, such as sulfadiazine or sulfisoxazole; and the urine should be sent for culture and sensitivity studies. Recently the Medical Letter* recommended a sulfonamide and stated that ampicillin and tetracycline had not proven to be superior for initial therapy. The use of sulfonamides initially for the treatment of acute urinary tract infections in the outpatient setting has been recommended by others./13/ If the organism is sensitive to the sulfonamide the urine will be sterile within 48 hours./1p926/ Therefore, the patient's urine should be re-evaluated microscopically three days after initiation of therapy. If there is still evidence of infection, the patient

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*Medical Letter. Vol 12, 12 June 1970.

should be started on the least toxic antibiotic for which the organism is sensitive. Ampicillin and tetracycline (only in children over eight years) are probably the drugs of choice if the organism is sensitive. Furadantin® is also frequently effective, and other than nausea, has relatively few side-effects.

Treatment of acute urinary infections should consist of two to six weeks of antibiotic therapy. Pryles /1p925/ recommends that all initial acute infections be treated with full dosage of an appropriate antimicrobial agent for a minimum of six weeks. This is based on observations that the recurrence rate is less after six weeks of treatment than after four or fewer weeks and the same as that after three months of treatment. Recurrence after two weeks of therapy is due to reinfection in at least 80 percent of cases as shown by Kunin./2/ He prefers to treat for two weeks and follow closely for recurrences. Fifty percent of the patients will have recurrence within the first year and 80 percent will have recurrence some time in the future./2/ Therefore, follow-up is the most important aspect of treatment if one is to prevent renal damage. Cultures should be obtained at two week intervals for several months following treatment. Cultures should then be obtained about every three months for the next year. After this time these patients must still be followed closely with periodic urine cultures because they are at much greater risk than those who have not had previous infections.

Most "treatment failures" are not due to persistence, but rather reinfection. However, those recurrences which are due to persistence are more difficult to cure. In general, this group is more likely to have organized and functional abnormalities of the urinary tract./13/ Further urological evaluation including cinefluorographic studies are indicated./1p926/ Any lesion producing obstruction or severe reflux should be surgically corrected. Frequently after proper corrective surgery these infections will respond to antimicrobial therapy.

Vesicoureteral reflux is common in children with urinary infection but has a natural tendency to improve with conservative treatment. Severe vesicoureteral reflux has been noted in children without urinary tract infection. According to Brumfitt and Reeves /13/ "surgical treatment should be reserved for children with uncontrolled infection associated with reflux, in whom progressing renal damage has been demonstrated." Many

authorities would disagree and insist that one should not wait until progressing renal damage is noted. In either case, provided infection can be controlled by chemotherapy, surgical correction of vesicoureteral reflux is contraindicated./13

There are always stubborn infections which persist despite absence of an anatomic defect. These infections are commonly due to resistant strains of gram-negative organisms. Sulfonamides are often not effective./1p926/ In these cases sensitivity studies should be considered and high dosage treatment given for two to six weeks with frequent culture and sensitivity evaluation. Attention should be given to urinary pH. Most antibiotics are more effective if the urinary pH is adjusted for the particular agent./13/

CONCLUSION

The physician must be aware that prevention of progressive renal damage due to infection is possible. However, strict criteria for diagnosis and proper management are necessary. Follow-up of these patients is by far the most important single factor.

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ENURESIS

CPT Larry Rork, MC

Enuresis is a common problem, one which has been recognized and written about as far back as 1550 B.C. Today, the amount of literature concerning enuresis is staggering. However, the understanding of this problem is just beginning. Proposed treatments for this entity over the last 3400 years, based on no apparent understanding of enuresis, are fascinating to review./1/ The Eber's Papyrus, the first known medical writing to mention enuresis recommended as "a remedy for incontinence of urine: juniper berries, one part; cyprus, one part; beer, one henmu measure."

About 3,000 years later, in 1544 A.D., in the Boke of Children, written by Thomas Phaer, the "father of English pediatrics", we find in the section "Of Pyssing in the Bedde"...

> Many times for debility of virtus retentive of the reines or bladder, as wel olde men as children are often times annoyed, when their urine issueth out either in their sleep or waking against their wyles, having no power to reteine it whan it cometh, therefore yf they will be holpen, fyrst they must avoid all fat meates, til ye vertue be restored againe, and to use this pouder in their meates and drynkes.
>
> Take the wesande (trachea) of a cocke, and plucke it, then brenne it in pouder, and use of it twise or thryse a daye. The stones of an hedge-hogge poudred is of the same vertue.
>
> Item the clawes of a goate, made in pouder dronken, or eaten in pottage.
>
> If the patient be of age, it is good to make fyne plates of leade, with holes in them; and lette them lye often to the naked backe.

Other preparations invoked during the last four centuries have included such things as the cerebrum of a hare which was round and imbibed, the lung of a kid, either eaten or made into plasters, the bladder of a young breeding sow pulverized and imbibed, or the dried comb of a cock ground and scattered on the bed of the wetter without his knowledge. Attempts were made at research to find a more effective treatment, and it is stated by one worker...

> I myself know from experience that the flesh of a ground hog checks the flow of urine so that if it be frequently administered, it prevents the passing of urine. Although in this matter, there seems to be controversy because Avicenna asserts that the flesh of a hedge hog softens the bowels and provokes urine, so too, Rasis. Yet if one considered their dicta that he will understand what is said is true. The experiment is true and has been proven by me.

Among more direct methods of treatment were the obstruction of the urethra by either clamping the penis or sealing it over with collodian, placing a balloon into the vagina and inflating it, ligation of the penis with a string, typing a knotted towel or a steel spur into place in the small of the back to prevent the child from lying on his back, or cauterizing the urethral meatus to make it painful to urinate. It may be that some of these treatments had value, but one would wonder about the morbidity associated with a cure.

In more primitive areas of the modern day work, mystical and magical rites are still practiced. The Bantu tribe of South Africa sacrifices the flesh of the cheeks to allow the "bad blood" to escape and thus, hopefully, stopping enuresis./2/ Exorcism and invocation of supernatural forces is also used by other primitive tribes. In West Africa, the child who continues to wet the bed after age four is first beaten. If this fails to cure the enuresis, ashes are poured over his head, and he is chased into the street by other children who chant and sing after him, "adida ga ga ga (= urine everywhere)". The Navajos, although bothered little by most behavioral abnormalities, are quite upset by bedwetting. In this tribe, the treatment of an enuretic child is a "magical" rite - the child must stand naked with his legs spread over a burning bird's nest. If this rite fails, other measures are instituted.

Enuresis was recognized as a frustrating affliction even by the church in the Middle Ages, and prayers of supplication to St. Katherine of Alexandria, on each November 25, were offered for the benefit of all enuretic persons.

It is unfortunate that the suggested treatments shed little light on the thoughts of their advocates concerning the cause of enuresis. As mysticism and magic faded out of medicine, a good degree of knowledge about many other afflictions was acquired. The scourge of diphtheria and poliomyelitis lost prominence on the list of man's diseases. Heart defects were found to be operable, and skills were developed to reduce the morbidity and mortality of surgery for congenital cardiac diseases. Unfortunately, problems affecting mankind such as the common cold have remained an enigma. In recent years, much has been learned, but the resolution of many of these entities, including enuresis, remains a goal in the future of medicine.

Present concepts surrounding the etiology of enuresis are many./3-10/ These theories are often tested with ill-devised studies, and frequently only result in adding a degree of confusion to the picture./7,9,11/ It is difficult to establish among various workers a consistent definition of enuresis and so it should not be at all surprising that criteria for various studies are subject to a great deal of criticism. This paper, offers a general approach in handling the patient who presents with the complaint of bedwetting.

It is well-known by those working with enuretic individuals that a family history will usually reveal at least one of the parents was enuretic /9/ or other enuretics were in one of the parents' families. This observation led Otto in 1830 to write of "congenital enuresis" /1/, a term which may still be found in the literature./12/ Halgren /13/ observed a higher incidence in frequency of enuretics in both of identical twins as opposed to fraternal twins. Others /2,14/ have also shown a high incidence of enuresis occurring in siblings of enuretics whose parents were enuretic.

Muellner /4/ after studying 1,000 pediatfic patients declared that "primary enuresis is due to the improper development of adult urinary control which leaves the child with a bladder capacity which is too small for its age and stature - a theory which is also held by other workers./2/ The feeling of these individuals is that enuresis can be resolved only when the bladder reaches a maturational level at which time it will hold up to about 12 ounces of urine. Muellner states that, by controlling the child's urinary output during the day to enlarge and distend the bladder, enuresis can be cured. This program,

however, takes 3 to 6 months in the hands of parents who are "intelligent enough to follow the training schema" and children "who are mature enough to cooperate".

Obvious organic pathology includes uropathies /2,3,9/ and central nervous system abnormalities. Urethral obstruction, ectopic ureter opening into the urethra, diverticulum or the urethra and other obvious lesions such as meningomyelocele and epispadius result in enuresis. Infection has been found to be associated with bed-wetting, but is thought to be a cause usually only as a result of cystitis./10/

Also there has been shown a great deal of interest in the relationship of abnormal electroencephalograms (EEG) patterns with enuresis./1-3,7/ Several workers have shown that 39 to 46 percent of the enuretic patients studied have unusual EEG activity. These patterns may vary from a borderline normal-abnormal EEG, to a small percent who demonstrate actual seizure activity. It has been stated that enuresis may represent an immature or primitive form of epilepsy./3/

An observation resulting from REM studies in enuretic and normal subjects has been presented by Di Perri and Laura./15/ These investigators placed indwelling catheters in enuretic and non-enuretic individuals. While the subjects were sleeping, enough water was instilled into the bladder to cause firing of the detrusor reflex, which, under normal conditions would initiate urination. In the normal subject, it was noted that before activation of this reflex, a cerebral arousal response was obtained and the person awakened. However, in the enuretic, sleep levels were only transiently changed, progressing, for example, from a deep phase to a lighter phase of sleep without any clinical sign of awakening when the detrusor reflex was activated. The deep sleep /2,11,12/ noted by parents in bed-wetters may be in some part due to this unusual sleep manifestation.

In recent years, the psychophysiologic theories of enuresis have become quite popular./1,2,7,9,16/ Volumes of papers during the last four decades have been offered in an attempt to explain bedwetting. Breger /7/ in his paper discussing the psychobiological approach to enuresis concluded that there were three factors to consider as causes of enuresis - organic, heriditary-constitutional, and psychogenic. The first two have been commented upon. Under psychogenic, he included toilet training pathology which resulted from a cultural lack of adequate training and deficit in communication or inappropriate training methods on the part of the parent. Here, two extremes in attitude may be noted: (1) that in which there is no concern of the parent toward toilet training, or

(2) the opposite in which the parent expects early training and assumes a harsh, punitive role and placed excessive and unrealistic demands on the child in training. (It is interesting to speculate on the comment of one lecturer that enuresis is a form of "weeping from the bladder" for want of love.)

TREATMENT

Although each of the previously mentioned factors (genetic, maturational, pathological, habit deficiency, and psychophysiological) play some part in the etiology of enuresis, none can singularly explain it. Each patient has his own combination of factors which makes him an individual case./9,10/ It is obvious that some children with enuresis will require referral to various services for treatment of this disease. A neurological deficit or urinary tract abnormality may eventually require surgical intervention. This necessitates a complete history and a thorough examination for physical abnormalities before progressing on to treatment. Some suggest that the basic evaluation includes a urinalysis and urine culture, an intravenous pyelogram, and in some cases a cystoscopic examination and a voiding cystometrogram./5/ The necessity for each of these procedures must be considered for each patient in accordance with his age, and the physician's clinical judgment./2,7/

Other patients may require varying degrees of psychotherapy. Treatment for enuretics has been primarily directed, in the past, toward counselling and suggestion./1/ Presently, some workers think that short term psychotherapy may not be any more useful than no psychotherapy./9/ It must be kept in mind that enuresis can be a symptom of an emotional disorder, and although it can be treated as an individual disease, it may be in some patients only an outward manifestation of a deep-seated emotional problem. In these cases, long term psychotherapy may result in resolution of enuresis; and the symptom and the illness may be treated concurrently./9/

The current trend in treating enuretics is to use a pharmacological method, but there are also other programs available which have a long standing popularity.

One of the most popular programs is based on conditioning./1,2, 9,12,17/ This type of therapy is available commercially and may be obtained without a physician's recommendation, although it is used by many practitioners. This form of treatment is generally held

in ill repute by some therapists* though others are less critical of its use./9/ Conditioning is achieved by placing an electrical device on the bed, which sounds an alarm when activated by urine. When the alarm sounds, the parent must also awake to see that the child has been aroused, and to assist him to the toilet if necessary. The child is also encouraged to increase his fluid intake during the day and before bedtime in an effort to increase bladder size. Within two to three weeks, bedwetting has either decreased or disappeared in those children who will benefit by it. Over a long period, this program results in a 40 to 90 percent resolution of enuresis./2,9,17/

The immediate drawbacks to the use of this procedure are significant./9/ The primary obstacle is the cost. One company quotes a rate in excess of $300 for a 45-day conditioning period. This fee is reduced for welfare families. Another potential obstacle is the cooperation demanded on the part of the parents and child to obtain results with the program. There have been burns reported following the use of these devices, but generally, those which are on the market today are quite safe./18/

Another form of treatment deals primarily with counselling and suggestion./1,2/ Although this is a time-honored method /1/, it may not in fact be of much use in achieving results directly./9/ It is, however, necessary to approach the patient with a positive attitude. The family's attitude toward the patient should be structured to reduce the stress he is encountering from bedwetting. Those involved in therapy with the enuretic patient should be instructed to adopt a positive, non-punitive attitude. It is also necessary to organize the family to work as a unit when dealing with this problem. Conflict between parents in response to a wet bed and inconsistency in attitudes adds very little to a child's confidence in treatment.

Time is also known have value in the treatment of enuresis./2/ Unfortunately, when a family requests help, it is no satisfaction to them to know that relief will come with puberty or adolescence. Generally, the child has been restricted from peer activities and finds himself socially isolated, and can not afford to wait. However, it should be made clear to parents that bedwetting, except in severe cases with emotional disturbances, will usually stop with time.

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*Personal Communication, LTC Charles K. Cordes, MC, Chief, Child Psychiatry Service, Letterman General Hospital.

The time-honored practice of restricting fluids before bedtime may be instituted, but in view of Muellner's work /4/ it appears that forcing fluids to distend the bladder and encouraging the child to hold his urine may be more effective./4,17/

The child may also be given an alarm clock to set for a certain time after going to bed so that he may awaken and go to the toilet. Generally, this is not successful, but it seems work to in some cases. A program of rewards for "dry nights" may also be tried, although this can be very discouraging to some children who do not respond easily to suggestion. It may also be used unwittingly as a type of punishment on the part of the parent.

The bedwetter can also be asked to assist in the task of cleaning up behind himself, thus relieving some of the pressure on the parent and if he is old enough to change his bed and his clothes, prefers doing so by himself rather than have his parents assist.

As stated before, unfortunately, many patients with enuresis do not respond to short term counseling./9/ Depending on the circumstances involved, it may be necessary to progress to the use of medication. If a child is young enough that bedwetting is not curtailing social activities, counseling may be extended, and "tincture of time" be prescribed. However, in the case of an older child, who is "different" because he can not stay overnight at a friend's house, or will not go on a camping trip because of fear of wetting /2,8,11,19,20,†/ his bed, pharmacological treatment is indicated.

Imipramine hydrochloride (Tofranil®) is currently the popular medication; /8,11,19,20,†/ desipramine /8/ (Norpramin®, Pertofrane®) and amitriptyline (Elavil®) are also used but not as frequently in the United States. Other agents such as belladona, atropine and probanthine which cause urinary retention have also been used./2/ Dexedrine was considered at one time to have some value but it is known now that it has little value. /12/

The mechanism of imipramine in bedwetting is not understood./10,20/ Parents note that the child is easily aroused to go to the toilet, and usually does so by himself for the first time in his life while taking this drug. It may be that this drug acts in some manner to

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†Nocturnal enuresis in children. Medical Letter 11:19-20 (7 Mar) 1969

coordinate cerebral cortex and the bladder./11/ There are many other theories based on its antidepressive character, its action as a stimulant, and its parasympatholytic qualities./11,20/ Regardless of what causes it to work, double blind, controlled studies and individual reports have shown that it does have a significant value in relieving bedwetting./8,11,19,†/ Unfortunately, it does not establish a long term cure, and the relapse rate following discontinuance of the drug is high./11,19/ The drug may be potentially harmful, /19,†/ and close follow-up of patients receiving this medication is advised.

No easy method is available for treating enuresis at this time. A satisfactory understanding of the problem still needs to be achieved. Methods of treatment now involve conditioning, counselling, and psychotherapy, the use of drugs, and time - all have their advantages, and each has its drawback. Those must all be considered in tailoring the program to each patient's individual needs.

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If there is any possible means of increasing the common wisdom and ability of mankind, it must be sought in medicine.

—DESCARTES

ASTHMA
Management of the Child Who Wheezes

MAJ Owen P. O'Meara, MC

> Asthma is a word with various meanings. To the patient it means wheezing respiration no matter what the cause. The allergists thinks first of bronchospasm due to specific hypersensitivities. To the psychiatrist it is merely a psychosomatic disorder indicating psychological maladjustment./1/
>
> H. Corwin Hinshaw

The above statement gives some idea of the diversity of opinion that exists among physicians about the nature of asthma. There is, perhaps, no other disorder in childhood that causes more emotional reactions in patients, parents, and physicians. The disease somehow affects almost everyone in the patient's environment. The purpose of this paper is to give a few suggestions about the day-to-day outpatient management of the asthmatic child. The management of status asthmaticus is another question and will not be considered here.

During the years 1959 to 1961, it was estimated that 74.3 out of every 1,000 children in the United States were troubled with either hayfever or asthma./2/ One-third (approximately 1.5 million) of these children had asthma. The cost of their medical care is especially frightening when one contemplates the added expense if they require inpatient care. I want to stress, therefore, outpatient care for these children and to point out that the reasons for optimal outpatient care are not all financial. There must be a rational and consistent outpatient program for the asthmatic child. He not only has asthma, but his asthma may led to other forms of chronic disease./2/ Poor management early in life may result in his becoming a pulmonary cripple who constantly wonders where the next breath of air is coming from For this unfortunate individual, the definition of asthma is simply "I can not breathe".

All that wheezes is not asthma. . .

Before specific therapy for bronchospasm is undertaken, other causes of wheezing respiration in the child must be ruled out -- bronchiolitis, foreign body aspiration, external compression of a bronchus or airway, cardiac failure, aspiration pneumonitis, bronchiectasis, tracheoesophageal fistula, pneumonia of various causes, and others. All of these disorders have one aspect in common with asthma -- obstruction of the airway. No matter what the cause of obstruction in an airway, the patient's response is much the same. It is, therefore, to the relief or removal of this obstruction (and preferably to its prevention!) that the physician must address himself.

FORMULATION OF A THERAPEUTIC APPROACH

There are a variety of components or factors that merge together and result in what we call asthma or the wheezing syndrome. Because of this variety of factors, there must be a variety of therapeutic approaches as well. We must not lose sight of the basic fact -- the most important pathophysiological mechanism of asthma is the reactive airway. Why some individuals are born with predisposition to reactive airways disease is not clear, but this predisposition can be noted early in an infant's life. Figure 1 presents some of the etiologic factors which appear to lead to the wheezing syndrome./3/

One may see there are different lines of development from predisposition to reactive airway disease to true asthma. The goal of optimal outpatient management, of course, is to put a block at as many locations as possible along this line of pathophysiological evolution.

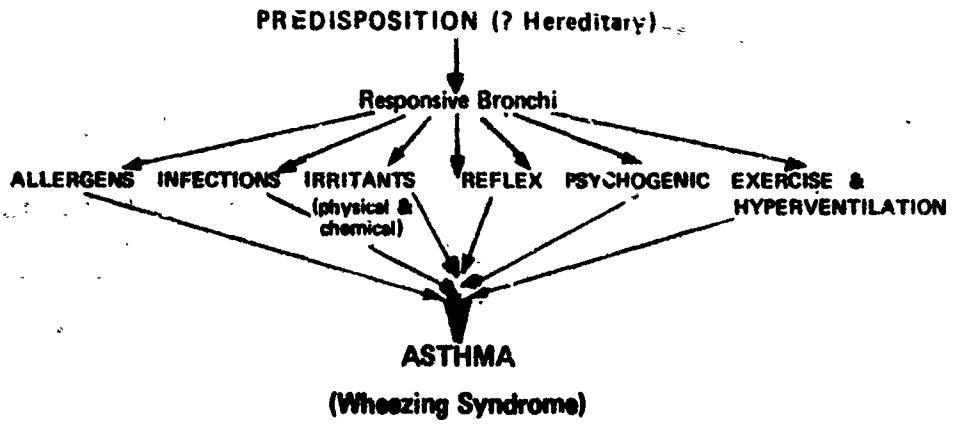

Fig. 1. Some of the etiologic factors which appear to lead to the wheezing syndrome are shown in in this illustration. The most important pathophysiological mechanism of asthma is the reactive airway. ***(Adapted from Ionno and Ward [3])***

PREDISPOSITION ———— REACTIVE AIRWAYS

This step in the chain of events -- predisposition ⟶ reactive airway -- is perhaps the most difficult to block. There are no laboratory tests or pulmonary function tests which give us any clear indication that an individual has hereditary predisposition to reactive airways. A family history of asthma or allergic disorders is all the physician may have as "facts" and all he may be able to do is advise the parents on how to prevent the child's having contact with the specific stimulants that trigger bronchospasm. Sometimes even this simple brand of preventive medicine can not be effectively practiced because of environmental circumstances. It is imperative, however, that the physician begin to sounsel the child's parents as soon as the child presents with symptoms bronchospasm (even if the patient is an infant with the symptoms of bronchiolitis).

The Avoidance of Allergens

It would be naive for one to suggest to a parent that he keep his child away from all the allergens which have been known to result in the stimulation of bronchospasm. I will comment on three common ones -- house dust, pets, geographical location. House dust is one of the more serious stimulants to allergic disorders. A physician may be able to give the parents a brochure which will help them to make their home as dust-free as possible. Often, however, the family is financially unable to do a completely adequate job of this. Then, if there must be an alternative, the physician should spend time instructing the family how to keep dust-free a limited area of the house -- the part where the child spends the greatest amount of his time which is probably his bedroom. Another problem which invariably arises with regard to allergic children is the subject of pets, and this can become a highly emotional topic. If a child shows definite allergic symptoms when playing with a certain type of animal then that type of contact should be avoided but he should not be deprived of having a pet to which he shows no signs of being allergic. Geographical locations -- where one lives -- can make life unpleasant for the individual with allergies, particularly those places with a high ratio of polutants and pollens in the air. How a family deals with this problem is more a matter of how convenient it is for a family to move than anything else. Most often this type of escape is simply not practical.

The Avoidance of Infections

It is virtually impossible for a schoolage child to avoid contact with other children who have upper respiratory infections. One can only emphasize that when the child with reactive airway disease does come in contact with these children and becomes infected himself, then he must have rest and proper medication promptly.

The Avoidance of Physical and Chemical Irritants

In the metropolitan area of this country it is virtually impossible to avoid contaminated air, but the pollutants and irritants expelled into the child's environment by other members of the family can be controlled. They should be encouraged not to smoke in the presence of asthmatic children, and to abandon hobbies and activities which lend themselves to the production of dust, smoke, or noxious fumes. There is no sanity in recommending that a family move half way across the country to avoid a polluted atmosphere when the father is continuing to use a paint sprayer in the basement of the house!

The Avoidance of Hyperventilation and Excessive Exercise

The effects of hyperventilation or exercise have been shown on the reactivity of the airways./4-6/ A child who knows he can trigger bronchospasm simply by breathing fast has a very powerful weapon in seeing that he gets what he wants. As far as exercise is concerned, I would recommend that it be limited only when it appears detrimental to the child Telling the child that he may not participate in sports or other activities involving expenditure of energy can be disastrous.

Pharmacologic Aids of Management

Usually the environmental controls are met with limited success and the use of pharmacologic aids is warranted. The most easily accessible of all medications is water -- plain tap water, soft drinks, juices of any kind,weak tea or any other dilute liquid of a water base. The encouragement of large quantities of water intake is perhaps the one most important thing the parent can do for the child when the child first becomes symptomatic. Water is the best prophylactic agent against the obstructive nature of viscous bronchial secretions.

Medications that relax bronchial smooth muscle, leading to bronchodilatations, can usually be relied on. The most time-tested of these is epinephrine hydrochloride which is usually given

subcutaneously in the form of 1:1000 solution, in a dose range starting at 0.01 cc/kg body weight. This dose should never exceed 0.5 cc in any single dose in any child. It can usually be repeated every 30 minutes, twice or three times. If one can not see a response after two or three administrations of epinephrine, it is fruitless to proceed with this drug. Also, it may be an indication that the child is in a state of acidosis which makes the drug ineffective or that the child's primary problem is infection.

The next most commonly used bronchodilating agent is probably isoproterenol hydrochloride (Isuprel®). This is usually given to children with asthma in the form of a nebulized spray (concentration of 1:400). Two to three deep breaths of this spray can be taken every three to four hours. Isuprel® should be used only when the use of epinephrine has been discontinued. The use of them together may cause complications. (I would like to interject a word of caution about Isuprel®. A child, especially the adolescent child, should not be given a nebulizer containing Isuprel® to take home and to use at will. Children of this age have a tendency to over use this form of medication. Medihalers and nebulizers should be under strict control of the parent who has been instructed by the physician.)

There are some bronchodilators used in the management of childhood asthma which have a combination of ingredients in the product. The three most accepted products combine theophylline, ephedrine and a tranquilizing drug such as phenobarbital, although one brand uses atarax. One brand also adds potassium iodide as an expectorant. In my experience, the addition of potassium iodide has limited value,(and besides, I have found water to be the best expectorant available!). The use of one of these products is most effective if it is begun at the time the child's symptoms begin. The doses are determined by the amount of theophylline in the medication, and the dosage should not exceed 10 mg/kg/24 hr. It is difficult to state whether the use of atarax is superior to phenobarbital. Atarax does appear to be indicated as an alternative when one sees a child's response to phenobarbital is increased activity and increased irritability. At the present time there are no good control studies to indicate that either of these agents is superior to the other. Atarax is expensive and this may be a limiting factor in the frequency with which it is used.

Use of Antibiotics

Antibiotics should be started when the child with wheezing shows the least sign of infection and when the wheezing is unresponsive to bronchodilator therapy. I favor the use of erythromycin or penicillin in these instances. Erythromycin may be the more logical choice because of the similarities to penicillin with regards to spectrum and also because of its possible value against some virus particles. I do not believe in the prophylactic value of antibiotics in the management of asthma.

Use of Steroids

It is difficult to give an answer to the question -- should chronic steroid administration be instituted in the treatment of asthma. I believe they should be used only as a "last resort" and there is no objective way of determining when that time comes. I would suggest, however, that a child who requires hospitalization more than five or six times a year and who has permanent changes on pulmonary function testing may be a candidate. We must always remember -- each child with asthma is an individual problem.

Other Methods of Therapy

Physical Therapy in Outpatient Management

When dealing with any patient with obstructive airway disease, postural drainage can be of tremendous value in clearing the secretions. The parents should be schooled by a qualified physical therapist in the use of the techniques for postural drainage. The child should also be taught to control his breathing, especially his breathing rate during periods of excitement. Hyperventilation has been implicated in triggering bronchospasm.

Less Conventional Modes of Therapy

In 1966 a paper appeared in the literature indicating that the change in potential of atmospheric ionic particles by a charge generator may have a role in therapy of reactive airway disease./7/ I have not seen any follow-up to this initial work. Eventually this and other less conventional modes of therapy, including post-hypnotic suggestion, may be added to the therapeutic regimens in the management of this disease.

"When all else fails call a doctor. . ."

Seeing the physician regularly for direction, prescription and supportive counseling is necessary for the asthmatic child and his parents. Then there are times when it is necessary to take the child to the hospital for inpatient care, such as, (1) when the child fails to take fluids, (2) when he does not respond to hydration and conventional bronchodilator therapy, (3) when there is evidence of carbon dioxide retention or desaturation of arterial blood, (4) when there are signs or symptoms of systemic acidosis, or (5) when there are poor breath sounds regardless of whether or not wheezing exists.

Determining whether or not the child should be admitted to the hospital for a period of inpatient care in addition to emergency procedures administered when he arrives depends a great deal on how much the physician knows about the individual child and his disease. When the asthmatic child is an inpatient he requires variations and additions to the therapeutic management proposed in this paper. The describing of such inpatient care invites exploration into other dimensions of the problems of the wheezing child.

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Claims to Cure Asthma

Editor MEDICAL WORLD – I note that in the May issue you say (page 190): "We all know well that asthma once establisht is incurable." Allow me to say, Doctor, that is a mistake. I never fail to cure asthma, no matter how well establisht. I will give one case: A lady in Pittsburg, Pa., 70 years of age, had asthma for 20 years; much of the time had to sit in a chair; could not sleep in bed. I prescribed the following:

Nitrite of amyl	
Fluid ext. lobelia	
Fluid ext. skunk cabbage..... āā	3 drams
Fluid ext. blood root.....	20 drops
Simple syrup.....	8 ounces

Dose, one teaspoonful before meals and on retiring.

She took three bottle and was cured. I heard from her 20 years after, still living and no return of the asthma. The above has never failed me in any case of asthma. Have lately discharged two cases, both cured. These two cases took only two bottles each. I hope this prescription may be of like benefit in the hands of others.

Wyoming, Del. DR. J. H. THOMAS
June, 1906

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Present Concepts in Internal Medicine, Volume II No 6, June 1969, presented a Symposium on the "Asthma Syndrome".

GOITER

Diagnosis and Treatment of Goiter in Children

MAJ Richard H. Ringel, MC

Diseases of the thyroid are, with the possible exception of diabetes mellitus, the most common endocrine dysfunction in the pediatric age group. Most of them are associated with a goiter and it is the purpose of this paper to review briefly the diagnosis and treatment of goiter in children, excluding the neonatal period. Table I lists the various causes of goiter.

TABLE I

CAUSES OF GOITER

CHRONIC LYMPHOCYTIC THYROIDITIS (Hashimoto's thyroiditis)
ADOLESCENT GOITER
ENZYMATIC DEFECTS (Familial goiter)
THYROTOXICOSIS
IODIDE DEFICIENCY
GOITROGENIC AGENTS
THYROID CARCINOMA
ACUTE THYROIDITIS

The most common cause of goiter in the pediatric age group, and probably the most common cause of acquired hypothyroidism, is chronic lymphocytic thyroiditis, which accounts for up to 40-50 percent of all goiters in this age range. The age of onset is usually between six and sixteen years, with a peak incidence about age 11-12 years. There is a very marked female preponderance - usually 90 percent or more of the cases are found in girls.

The most common presenting symptom or sign is an asymptomatic goiter in a clinically euthyroid child. The goiter is usually firm and frequently has a granular consistency. Rarely is it tender. Nodules are uncommon, but may appear in longstanding goiter or after treatment has begun. Often there is a Delphian node present. When symptoms are present, they are most commonly nervousness and fatigue. There is a positive family history of thyroid disease in up to 50 percent of the cases.

Hypothyroidism will occur in about 25-50 percent of the cases.

There may be other associated pathologic states in these patients, for example Down's syndrome of Turner's syndrome, diabetes mellitus, or hypoparathyroidism, Addison's disease and moniliasis./1/

The protein-bound iodine (PBI) is usually normal, but may be elevated early and decreased late in the disease. A discrepancy between the PBI (normal 4-8 µg/100 cc) and the thyroxine (T_4) (normal 3.0-6.5 µg/100 cc) of greater than 1.5 µg/100 cc is characterisitc of this disease. The difference is thought to be caused by an abnormal iodoprotein, the exact nature of which has not been clearly delineated. The ^{131}I uptake is usually normal, but may be either increased or decreased. When increased, it can be suppressed with triiodothyronine, but when decreased, it can not be elevated with thyroid stimulating hormone (TSH). Antithyroid antibodies (thyroglobulin (CA), microsomal antigen, and a second colloid protein (CA_2)) are elevated in this disease. When all three of the antithyroid antibodies are searched for, they will be present in almost 100 percent of cases.

Adolescent goiter refers to the presence, most commonly in females, of an asymptomatic enlargement of the thyroid in a euthyroid patient. Most of these are probably examples of chronic lymphocytic thyroiditis or a mild enzyme defect.

Familial goiter results from the inheritance of a deficiency or disturbance in the normal production of thyroid hormone. It appears to be inherited in an autosomal recessive pattern; therefore, in contrast to other forms of thyroid disease, the sex ratio is 1:1. Depending on the severity of the defect, the patient may present with either hypothyroidism or a euthyroid state in association with a goiter. The goiter is usually moderate in size, diffuse, and rarely nodular. To date five defects have been recognized.

An inability to trap iodine. There is a decreased PBI and a decreased ^{131}I uptake unresponsive to TSH. Heterozygotes with this deficiency may also have a small goiter.

Inability to incorporate trapped iodine due to peroxidase deficiency. This is probably the most common enzymatic defect. These patients have a decreased PBI and increased ^{131}I uptake, usually reaching a peak at one to two hours. When given perchlorate or thiocyanate, there is a rapid discharge of the labeled iodine from the gland. This defect may be associated with nerve deafness (Pendred's syndrome). When this occurs, the goiter is often smaller and the degree of hypothyroidism less severe (may be euthyroid). Heterozygotes may also have a goiter.

Inability to combine iodotyrosines. This abnormality can be verified only after thyroidectomy. The PBI is usually low and ^{131}I uptake rapid.

Inability to deiodinate iodotyrosines due to dehalogenase deficiency. These patients also have a decreased PBI and increased ^{131}I uptake. Heterozygotes may also have a goiter.

Production of an abnormal iodinated thyroid protein. These patients will have a goiter and a normal or increased PBI, but a marked discrepancy between the PBI and T_4 values. The ^{131}I uptake is elevated.

Among non-euthyroid children thyrotoxicosis (hyperthyroidism) is only one-fifth as frequent as hypothyroidism, but the incidence increases with advancing age. The mean age of onset (12 years) is similar to that of chronic lymphocytic thyroiditis. There is again a female preponderance, being six times as common in girls. Thyrotoxicosis may be found in association with chronic lymphocytic thyroiditis or thyroid carcinoma.

The most common presenting symptoms include nervousness, increased appetite, exophthalmos, a mass or fullness in the neck, weight loss, increased sweating, heat intolerance, palpitations and tremors.

On physical examination most of the children tend to be tall (greater than 75th percentile) and thin, although only about one-third are less than the 50th percentile for weight. They usually have a tachycardia and mild hypertension, characteristically with an increased pulse pressure. Their

skin is warm and moist and their eyes are prominent. They are very hyperactive and restless, and a tremor is often present. The thyroid is moderately enlarged, and a venous hum or bruit is usually heard. Often there is localized adenopathy.

The PBI is elevated as is the ^{131}I uptake. The latter fails to suppress with triiodothyronine. Antithyroid antibodies may be present in low titers.

Iodine deficiency is no longer a common cause of goiter in this country since the addition of iodide to table salt. The PBI is down and ^{131}I uptake rapid. The disorder responds rapidly to iodine.

Another uncommon cause of goiter is the ingestion of goitrogenic agents. Included among these are iodides, cobalt, fluorine, para-aminosalicylic acid, resorcinol, phenylbutazone, sulfonylurea antidiabetic agents, reserpine, chlorpromazine, acetazolamide and 2,3 dimercaptopropanol. Also certain members of the cabbage family (cabbage, turnips, cauliflower, rutabaga) and soybean formulas without added iodine may result in goiter. In all cases removal of the offending agent results in resolution of the signs and symptoms. However, not all patients ingesting these compounds develop goiter, which suggests a genetic predisposition among affected people.

Carcinoma of the thyroid is another uncommon cause of goiter in children. In as high as 70 percent of cases there is a history of irradiation to the chest, face or neck for such conditions as thymic enlargement, hypertrophied tonsils and adenoids, hemangiomas, nevi, acne, eczema, or cervical adenitis. This variety is only twice as common in females as in males. In only one-fourth of the cases is the disease localized to the thyroid at the time of presentation and in three-fourths there is an associated nodule in the draining cervical lymph nodes. The child is generally euthyroid although thyrotoxicosis has rarely been reported secondary to a hyperfunctioning lesion. Carcinoma may be found in association with chronic lymphocytic thyroiditis. Medullary carcinoma of the thyroid may be associated with pheochromocytoma, and multiple mucosal neuromas. There appears to be a familial predisposition to this uncommon form of carcinoma./2/

On examination, the thyroid is often genrally enlarged, but nodules may be felt in 90 percent of cases. Accompanying cervical adenopathy is common.

The PBI is usually normal, uncommonly elevated or rarely decreased. ^{131}I uptake is normal, but scan usually reveals a "cold" nodule. Antithyroid antibodies may be present in low titers.

Acute and subacute thyroiditis are also uncommon in childhood. Both types are usually preceeded by an upper respiratory tract infection. Fever is often present and the goiter is quite tender. The PBI is often elevated and may be accompanied by symptoms of thyrotoxicosis, but the ^{131}I uptake is decreased. Antithyroid antibodies may be present in low titers.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of goiter in childhood requires an accurate history : complete physical examination and appropriate laboratory studies. Symptoms of hypothyroidism occur in cases of familial goiter, iodine deficiency, ingestion of goitrogens and chronic lymphocytic thyroiditis. Symptoms of thyrotoxicosis also occur with chronic lymphocytic thyroiditis, thyroid carcinoma and acute thyroiditis. A euthyroid state in the presence of goiter is associated with chronic lymphocytic thyroiditis, adolescent goiter, familial goiter and carcinoma. A history of a previous upper respiratory tract infection is obtained in acute thyroiditis and occasionally thyrotoxicosis. Previous irradiation to the head and upper body should make one think "carcinoma". A family history of thyroid disease is present in almost all these disease entities, but if the occurrence is in males as well as females, one should consider familial goiter, iodine deficiency, goitrogenic agents or possible carcinoma.

On physical examination a tender goiter is usually present in acute and subacute thyroiditis and rarely chronic lymphocytic thyroiditis. The presence of local adenopathy occurs with chronic lymphocytic thyroiditis, thyrotoxicosis, carcinoma, and acute and subacute thyroiditis. A solitary nodule should make one think of carcinoma, but multiple nodules may be present in thyrotoxicosis, chronic lymphocytic thyroiditis, and carcinoma. In all patients with thyroid nodules, whether single or multiple, a thyroid scan is indicated.

TABLE II contains a list of various laboratory values in most of the above mentioned conditions.

TABLE II

LABORATORY VALUES

DEFECT	PBI	^{131}I UPTAKE	PBI-T_4 >1.5	ANYBODY	OTHER
1. Chronic lymphocytic thyroiditis	N(↑↓)	N(↑↓)	+	++++	^{131}I uptake does not increase with TSH when low, but suppresses with T_3 when elevated. Does not respond promptly to replacement thyroxine.
2. Adolescent goiter	N	N	-	-	----
3. Trapping defect	↓	↓	-	-	↓RAI unresponsive to TSH.
4. Organification defect	↓	↑	-	-	Rapid loss of radioactive iodine from the gland following the administration of perchlorate or thiocyanate. May be associated with nerve deafness.
5. Coupling defect	↓	↑	-	-	----
6. Deiodinase defect	↓	↑	-	-	----
7. Abnormal thyroprotein	N↑	↑	+	-	----
8. Thyrotoxicosis	↑	↑	-	+	Hyperthyroidism- ↑RAI uptake fails to suppress with T_3
9. Iodide deficiency	↓	↑	-	-	Responds to iodine
10. Carcinoma	N(↑)	N	-	+	Cold nodule on scan
11. Acute and subacute thyroiditis	↑	↓	-	+	Goiter is tender.

T_4 = Thyroxine
T_3 = Triiodothyronine

In a clinically euthyroid patient probably all that is necessary is a PBI, T_4, and antithyroid antibodies. From a practical standpoint an ^{131}I uptake is not necessary in hypothyroidism either. With clinical hyperthyroidism a radioactive iodine (RAI) uptake is indicated to help differentiate between thyrotoxicosis, chronic lymphocytic thyroiditis, acute and subacute thyroiditis, or carcinoma. It is usually elevated in thyrotoxicosis, decreased in acute and subacute thyroiditis, and normal in chronic lymphocytic thyroiditis and carcinoma. If the diagnosis is still unclear, a trial of triiodothyronine for eight days is indicated and then the RAI uptake repeated. In thyrotoxicosis, the uptake will not be suppressed as it will in chronic lymphocytic thyroiditis.

Another point in the differential diagnosis is the response of a goiter to suppressive doses of thyroxine. Adolescent goiter and enzymatic defects will respond promptly, within two to four weeks, with a significant decrease in size. A diagnostic criterion for chronic lymphocytic thyroiditis is a failure to respond so rapidly./3/ However, if after a three month period of therapy with suppressive doses of thyroid hormone, and there is no reduction in size of the goiter, a biopsy should be performed.

TREATMENT

The modes of therapy for childhood goiter include thyroid hormone, antithyroid drugs, surgery, antibiotics or removal of the goitrogenic agent. The treatment for chronic lymphocytic thyroiditis is dessicated thyroid in full suppressive doses for approximately two years. For adolescent goiter the dosage is the same, although the duration of time may be shorter. Therapy is indicated in this condition only if the goiter is troublesome from a cosmetic standpoint. The dosage of thyroid is the same for familial goiter, but the duration is for life. Acute thyroiditis responds dramatically to antibiotics, usually penicillin, since the beta hemolytic streptococcus is thought to be the etiologic agent in most instances. In subacute thyroiditis, corticosteroids may be helpful. Carcinoma of the thyroid should be treated surgically. Often postoperative treatment with therapeutic doses of radioiodine is used, sometimes in conjunction with TSH stimulation.

The definitive treatment for thyrotoxicosis is controversial, although the initial therapy is agreed upon. Initially an attempt should be made to bring these patients to a euthyroid state with antithyroid drugs - propylthicuracil or methimazole. On this regimen thyrotoxic signs will usually disappear within a week to ten days and the PBI will return to normal in about a month. Once the patient has achieved a euthyroid state a decision must be made - should surgery be performed on the patient? or should she be continued with the antithyroid drugs at maintenance levels for about two years? This remains a controversial subject.

PROGNOSIS

Adolescent goiter and enzymatic defects respond excellently to therapy. Chronic lymphocytic thyroiditis usually takes longer to respond and at times a small goiter may remain. Twenty-five to fifty percent of patients with this disorder will develop hypothyroidism and require replacement therapy for life. Iodide deficiency responds rapidly to replacement. Removal of goitrogenic agents leads to prompt disappearance of the goiter. Acute and subacute thyroiditis are usually self-limited diseases without sequelae. Thyroid carcinoma generally has a favorable prognosis and maybe compatible with a normal life span. Thyrotoxicosis has a variable prognosis because of either the possible development of hypothyroidism following thyroidectomy or antithyroid drugs, or the occurrence of relapse. In summary, the prognosis for children with goiter appears to be good.

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Bronchocele, Goitre, or Big Neck,. . .

The *treatment* consists of local and constitutional measures. . .

. . . . . .

A change of residence to a dry, pleasant spot, is always advisable; the liberal use of Iodurettted Salt with the food will be found very beneficial, – about twenty grains of Iodid of Potassium or Iodid of Sodium, to a thousand grains of common Table Salt. Rain-water, or spring water that has been boiled and then allowed to cool, will afford the best fluid for drink among those who reside in goitrous districts. In other respects the the hygienic measures may be similar to those named under Scrofula. Eggs, oysters, fresh meats, milk, onions, cabbages, carrots, turnips, chicken, beef, and oyster soups, are important articles in the diet of a goitrous patient. Beans, peas, and all dry amylaceous food should be prohibited. Albuminous diet is highly proper.

–JOHN KING, M.D.*

From *Chronic Diseases*, a book authored by King, a doctor in South Carolina, about 1850. Publisher unknown (title page was missing from volume). pp 1607. This portion appears on pages 417, 418.

ABDOMINAL PAIN
Management of the "tummy ache" in Childhood

CPT Torrey L. Mitchell, MC

> Almost all the deaths and most of the serious complications of acute abdominal conditions in childhood occur in those patients whose operation has been delayed by late diagnosis. It is the awareness of the bad effect of delay that presents the family doctor with dilemma. Many children have abdominal pain or vomiting at some time in their lives, and in the vast majority it is not due to some serious disease requiring surgery. How is he to distinguish the serious from the trivial? If he is uncertain and decides to see the child again in six or twelve hours, is he risking the serious consequences of failing to recognize a true emergency? If he sends the child into hospital at once, is he cluttering up hospital beds with children who will come to no harm and would do better at home?
>
> Zachary/1/

This is the dilemma facing the child's doctor. The child with a "tummy ache" brings to mind a multitude of diagnoses which must be differentiated. The intricacies of the illness can only be unraveled through a careful and detailed history and physical examination.

HISTORY

It is helpful to know the patient's age. The incidence of several conditions is limited to certain age ranges. Intussusception, for example, generally occurs in infants under two. Menstrual cramping would not be considered before puberty. The precise time and manner of onset is essential to know. If the sudden onset of symptoms can be pin-pointed to say "ten past two", we are more likely to think of intestinal obstructions such as intussusception or an incarcerated hernia. Was abdominal pain the first symptom? A child who has had vomiting, headache, cough or fever for twelve or more hours before the onset of abdominal pain is more likely to have disease elsewhere - such as in the lungs or meninges. We need to know whether the pain is continuous or intermittent. Pain that comes and goes over a prolonged period is unlikely to result from an acute surgical condition. Infants and toddlers unfortunately cannot adequately convey the nature of their pains and are frequently stoic and so for the physician to obtain an accurate history is difficult. When there is a language limitation because of the child's age, it is sometimes useful to ask "Is it like an ordinary tummy-ache, or is it different?" Many children clearly understand this distinction. One should be particularly alert to significant disease if the child believes his pain is unusual in character or intensity./2,3pp13-21/

Severity of pain is especially difficult to assess, particularly when given the range of irritable to stoic youngsters. To gain some knowledge of the patient's everyday activities is a useful approach. If the child has gone off to bed in the middle of "Disneyland" (a favorite program), one can surmise his discomfort is serious and his complaint of pain merits more attention. If the patient is an older, more vocal child, one should ask the usual questions defining the character of the pain, the site of the pain at onset, and where it has localized or radiated./1/

Vomiting is an important associated symptom which should be characterized completely. What is its time relationship to the pain? In appendicitis or distal intestinal obstruction, pain may precede vomiting by several hours. Sudden irritation of the peritoneum (such as, passage of a ureteral stone or perforation of an ulcer) may bring on vomiting soon after the

pain. Vomiting preceding pain may arise from infection localized elsewhere, such as in the central nervous system. How frequent is the vomiting? This commonly varies directly with the acuteness of the precipitating illness. The nature of the vomitus must be known. The infant with duodenal atresia will vomit greenish, bile-stained fluid, whereas the child who has hypertrophic pyloric stenosis will have a vomitus which does not contain bile. Distal intestinal obstruction may produce foul, fecal-stained emesis. Acute neurogenic shock, as in torsion of a testicle, can cause the child to retch violently but vomit little./3/

Bowel movements should be investigated in a similar manner. The passage of blood and mucus suggests intussusception in the infant. The passage of frequent small movements is common at the onset of acute appendicitis in children. Notable variance from a child's established bowel pattern is cause for medical concern./3/

Past history and family/social history may provide insight into a child's complaint. The history of childhood nephrosis should immediately lead the clinician to suspect pneumococcal peritonitis.* The recent return of a family from the Far-East brings to mind parasitic infections or cholera. Is the pain recurrent? What has its pattern been in the past? Recurrent abdominal pain in the school-age child is frequently emotional in origin and should prompt a careful search for stresses in the family./4/

THE PHYSICAL EXAMINATION

The basics of physical diagnosis apply as well to the child as to the adult; and therefore shall not be dwelt upon here. There are limitations encountered in attempting to give the physical examination as well in trying to get the history of abdominal pain from the preschool child. The infant is unable to cooperate and may be crying throughout the examination. It may be almost impossible to assess the severity and localization of the pain. It is helpful to palpate deliberately both

*Acute urinary tract infection is one of the most common illnesses misdiagnosed in children. The review of the systems should be thorough and complete so as to rule in or out the large number of nongastrointestinal and extra-abdominal diseases mimicking the acute abdominal conditions.

sides of the abdomen for comparison. The intensity of the cry and even its quality may change when a really tender area is pressed. It may be reasonably satisfactory to examine the child sitting up - if the doctor has the experience to interpret the altered anatomy in the upright position. One must always remember to watch a child's facial expression, rather than his abdomen, so as to detect signs of tenderness./1,2/

The rectal examination is important, but should not follow next in sequence. The child as a whole must be examined in a manner appropriate for his age (such as, generally proceeding from the feet to the head for younger children). Particular attention should be paid to signs in the chest that suggest pneumonia, enlarged tonsils which may be associated with mesenteric adenitis, and tenderness in the flanks suggestive of renal infection. The rectal examination is essential, but should be pursued with caution lest the rectal orifice be torn in a small infant. Simultaneously, the hernia orifices should be inspected. Auscultation of the abdomen is important if bowel sounds are plentiful and other signs have pointed to peritonitis or ileus. Paucity of bowel sounds is seldom diagnostic, because the frequency of sounds is normally variable.

Diagnosis

Medical diseases as causes of abdominal pain outnumber conditions requiring surgery. The differential diagnosis is outlined in Table I.

TABLE I

CAUSES OF ABDOMINAL PAIN: DIFFERENTIAL DIAGNOSIS

Medical	INFECTIOUS COLLAGEN-VASCULAR HEMATOLOGIC	METABOLIC NEUROLOGIC DRUG/TOXINS
Surgical	GASTROINTESTINAL GENITOURINARY TRAUMATIC	

Medical Diseases

INFECTIOUS

The obvious initial consideration is gastroenteritis - viral, bacterial, or parasitic. Viral gastroenteritis should largely be a diagnosis of exclusion, but if other members of the family are similarly afflicted it should be suspected. Salmonellosis and shigellosis present as unusually explosive diarrhea and abdominal cramping. Confirmation of the diagnosis rests on stool culture; however, shigellosis can be readily suspected in a particularly toxic child having a remarkable left shift with upwards of 30 percent band cells and a normal leukocyte count./5/ Typhoid fever produces abdominal pain and distension secondary to enlargement and necrosis of lymphoid tissue in the bowel and mesentery. Amebiasis should be suspected if the child has traveled abroad. Stool examinations confirm the diagnosis of amebiasis or worm infectation. Worms seldom, if ever, cause abdominal pain except in the case of intestinal obstruction with ascaris roundworms or in the rare case of appendicitis caused by pinworms./6/

Pneumonia, bronchitis, and pertussis may cause abdominal pain simply from the muscular strain of coughing. Pneumonia itself can mimic all types of the acute abdominal conditions. The confusion arises because the diaphragmatic pleura is irritated with radiation of pain into the abdomen. Right lower lobe pneumonitis commonly produces right lower quadrant abdominal pain. Similarly, a left lower lobe infiltrate will simulate splenic pain and the patient will have tenderness in the left upper quadrant. It is important to recall that these symptoms normally arise before the child has any respiratory symptoms. Careful examination, however, will usually reveal decreased breath sounds in the suspected area. Pneumonitis should be demonstrable on roentgenograms./3pp182-191/

Pyelonephritis presents typically as intense abdominal pain with high, spiking fever. It is sometimes 2-3 days before flank pain becomes localized. Dysuria is frequently lacking. A microscopic urinalysis should be among the first diagnostic tests performed. Urinary tract infection, in general, ranks high on the list of causes of belly pain./7pp82-91/

Some children experience right upper quadrant pain at the onset of infectious hepatitis - before the onset of jaundice. Bilirubin should be detectable in the urine soon thereafter, and the natural course of the disease should make the diagnosis apparent.

Mesenteric adenitis is difficult to distinguish from appendicitis, except that the fever and white cell count tend to run higher than with appendicitis. When the child has mesenteric adenitis, he may also have tonsillitis or cervical lymphadenitis. The pain is often in the right lower quadrant where the ileocolic mesenteric lymph nodes are largest. The child will not appear as ill as the severity of his pain would suggest./7pp106-116/

A number of common viral diseases of childhood cause belly pain. Herpes zoster's sharp, stinging localized pain is present before the rash is visible./3pp182-191/ Measles has been known to cause varying degrees of appendicitis or colitis./8/ Mumps is the most common cause of pancreatitis in children./9/

COLLAGEN-VASCULAR

In one-third of patients with acute rheumatic fever, acute abdominal pain is an early and sometimes initial symptom of collagen-vascular disease. The pain is usually localized to the epigastrium, but may be diffuse; it may be severe, simulating appendicitis, but is readily relieved by aspirin. The abdominal pain may be the only symptom; thus, the destruction of rheumatic carditis goes undetected./10/

Henoch-Schoenlein (anaphylactoid) purpura's vasculitis results in gastrointestinal bleeding and cramping abdominal pain. This has been known to precipitate intussusception./11/

Rheumatoid arthritis uncommonly causes a mild form of mesenteric adenitis. Ankylosing spondylitis and scoliosis has been known to cause abdominal discomfort due to compression of spinal nerve roots./3/ Systemic lupus erythematosis, polyarteritis, scleroderma, and dermatomyositis all produce pain in the belly because of diffuse vasculitis - especially of the mesenteric vessels./12/

The collagen diseases have in common the use of anti-inflammatory drugs in their treatment. Steroids, salicylates, butazolidine, etc. are collectively known for their ulcerogenic effects.

HEMATOLOGIC

Sickle cell anemia is the prototype of congenital hemolytic anemias. This diagnosis should be promptly considered in a young negro child. He may be experiencing his first "pain crisis". Rigidity of the abdominal wall may or may not be present. Usually there will be aching pain of some joint or extremity. The finding of anemia and a positive sickle-preparation are confirmatory. These youngsters still are just as susceptible to appendicitis as other children and they are also prone to having pneumococcal pneumonia./13/ Other hemoglobinopathies and congenital spherocytosis may occasionally produce mild pain crises and susceptibility to cholecystitis./14/ Hemophilia may result in a hematoma retroperitoneally or within the intestinal wall. Leukemic infiltrates cause enlargement and capsular distension of spleen, liver and kidney. Lymphoma produces enlargement of lymphoid tissue of bowel and mesentery. Solid tumors are painful due to expansion plus hemorrhage and necrosis within the mass.

METABOLIC

Diabetic ketoacidosis and symptomatic hypoglycemia both are associated with abdominal pain. After a child's insulin requirement has risen during some stress, his first symptom may be a gnawing epigastric pain before the appearance of typical ketoacidosis. With an insulin reaction, abdominal pain may accompany headache, sweating and lethargy as initial symptoms. Hypercalcemia associated with hyperparathyroidism causes nausea and vomiting. If there is abdominal pain also, one should suspect duodenal ulcer, pancreatitis, or ureteral stones. Acute intermittent porphyria is a disease which rarely becomes manifest before puberty. It is characterized by severe stabbing abdominal pain. Episodes are typically recurrent and associated with various neurologic and psychiatric symptoms. Barbituates are among several drugs which may

precipitate an attack. The Watson-Schwartz urinary test for porphobilinogen is diagnostic./16,17/ Familial hyperlipidemia is a condition producing very high cholesterol levels. Death from coronary atherosclerosis can occur in early childhood. Abdominal pain arises from secondary vascular insufficiency.

NEUROLOGIC

Lesions of any kind in the central nervous system may produce referred abdominal pain. Abdominal epilepsy and abdominal migraine are entities of questionable validity. Several authors /17,18/ have shown that the incidence of electroencephalographic abnormalities in suspect children is the same as in control groups of normal children. If abdominal epilepsy is to be accepted as a diagnostic designation, the criterion for its application in a given case should be quite restrictive. The following clinical pattern would probably be acceptable to most clinical observers: (1) recurrent episodes of abdominal pain, with or without associated headache, but without twitching or convulsive movements, (2) somulence as a postictal manifestation, (3) an abnormal electroencephalogram, and (4) relief of attacks with anticonvulsive therapy./2,18/ Psychologic aberrations are the most important source of chronic, recurrent abdominal pain in school-age children./4,19,20/ This is to be distinguished from other causes of recurrent abdominal pain, e.g. hydronephrosis, peptic ulcer, food intolerance, constipation, parasitic infestation, and lead poisoning./2/

DRUGS AND TOXINS

Lead poisoning is relatively common in older cities where children are seen eating flaking lead-base paint from walls and window sills. History of pica can usually be obtained from parents. Signs and symptoms consist of abdominal pain, anemia, varied neurologic changes, "lead-line" between teeth and gums, stippling of red cells, radiologic lead-lines at the ends of bones, chips of heavy metal on abdominal films, elevated urinary coproporphyrins, and serum lead levels exceeding 60 µg/100 cc./21/

Poisoning from mercury, arsenic, spider-bite, or staphylococcal toxins in spoiled food results in severe abdominal pain./21/ A number of drugs such as oral iron and tetracyclines notoriously produce abdominal discomfort as side-effects.

Surgical Lesions

GASTROINTESTINAL

The signs and symptoms of appendicitis are usually familiar and identifiable. Some fallacies in diagnosis are noteworthy and listed by Illingworth./2/

> Sixty percent of the cases are atypical in location; one-third of these are retrocecal and therefore present only mild abdominal tenderness.
>
> In only 30 percent of the cases does the pain progress typically from umbilicus to the right lower quadrant.
>
> Urinary symptoms and pyuria may result solely from the proximity of ureter and inflammed appendix.
>
> Rupture of the appendix has an especially high mortality in very young children. At this age, open drainage without removal of the appendix is very dangerous since the body is unable to "wall-off" the infection. It must be removed.

Ulcerative colitis causes cramping lower abdominal pain associated with persistent diarrhea containing blood and mucus./23,24/ Regional enteritis is similar to appendicitis. It is diagnosed radiologically via small bowel follow-through studies.

GENITOURINARY

Sharp or blunt trauma to the kidney is the most common urologic cause requiring of surgical procedure within the abdomen. Renal calculi cause sudden, knife-like repetitive pain radiating to the testicles, groin, or thighs and is associated with vomiting, fever, and gross hematuria. Perinephric abscess produces high fever and chills. A mass is often palpable with obliteration of the psoas shadow radiologically on one side. Renal vein thrombosis is most common in the newborn infant. Associated findings are vomiting, diarrhea, fever, abdominal pain, dehydration, gross hematuria, proteinuria, and an enlarged palpable kidney. Polycystic kidneys may rupture or undergo rapid expansion due to hemorrhage. /7pp82-91/ Other considerations include neoplasms, pelvic inflammatory disease, torsion of a ovarian cyst or tumor, ectopic pregnancy, or intrauterine pregnancy in labor./7pp92-103/

Finally, any history of previous abdominal surgery should alert the physician to the possibility of intestinal obstruction secondary to peritoneal band formation.

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Pain in the stomach may vary in intensity from a sense of soreness, or, tenderness upon pressure upon the epigastrium, to most severe agony and suffering. . . .

. . . . . .

. . .–If it be of a *spasmodic* character, a few drop of Chloroform and Laudanum in an infusion of Scullcap, will be of service; or the Compound Tincture of Virginia Snakeroot; or the Compound Tincture of Lobelia and Capsicum. Relaxation should be effected by placing cloths moistened with hot water (but not hot enough to scald) over the gastric region, changing them frequently; and the bowels should be speedily evacuated by a warm active laxative injection. Equal parts of Tinctures of Gelseminum, Dioscorea, and Skunk Cabbage, will also prove useful, in doses of a teaspoonful every twenty, thirty, or fifty minutes. In many instances, Chloroform or, a mixture of Chloroform and Opium, or, a Belladonna Plaster, applied over the epigastrium, will promptly relieve pain; the chloroform should be prevented from evaporating by covering it with a dry cloth or oil silk. Ointment of Veratria, or, of Aconitia, rubbed over the pit of the stomach until a pricking sensation is produced, and repeated every two or three days, will often prove invaluable in obstinate and severe cases. These external measures, when applied alternately to the epigastrium, and to the sensitive region of the spinal column, will be very effectual in many difficult cases.

– JOHN KING, M.D.*

From *Chronic Diseases*, a book authored by King, a doctor in South Carolina, about 1850. Publisher unknown (title page was missing from volume). pp 1507. This portion appears on pages 750, 752-753.

POISONINGS
Management of the Child Who is Poisoned

CPT Melvin Hoffman, MC

Approximately 500 children less than five years of age die yearly as a result of poisoning accidents. The total number of ingestions varies from 500,000 to two million annually in the United States./1/ Ninety percent of the accidents involve items which are found about the home -- Medicines account for one-half the ingestions, household products the other half. Aspirin is by far the most commonly ingested medicine (50 percent of all medications ingested) and "baby" aspirin predominates (ratio 7:1) over adult-sized tablets. The household products most involved are cleaning and polishing agents, pesticides, pertroleum products, and cosmetics. These deaths are preventable.

The poisoning event can be described as an interaction between an agent (the hazardous substance), a susceptible host (the child), and an unstable environment./2,3/ A program for prevention of poisoning accidents can be directed at these three aspects.

To insure a safe environment, potential poisons should simply be made unattainable by children; for example, medicine cabinets should have child-protective latches Certain precautions concerning the agent should be observed, e.g. (1) all medications or household products not needed should be removed, (2) physicians should not prescribe excess medication, (3) after use, all toxic material must remain in their original containers and should be returned to their proper place of storage. A more important aspect in preventing poisonings is the use of child-resistant containers, specifically the "palm-in-turn" containers; - this one factor according to some studies /2,4/, has decreased the number of poisonings.

Epidemiologic analyses /5-7/ have revealed several characteristics typical of poisoned children and their families. Ninety percent of poisonings involve children less than five years of age, 50 percent of whom are two to three years of age. Younger children tend to ingest household products while

older children ingest medicines. The children are frequently impulsive, overactive, and possess negative attitudes. The parent-child relationships are often disturbed. Problems, such as parental discord, a working mother, or an absent father, may aggrevate the situation. Once a child has taken a poison, he is nine times more likely to repeat the act the following year than the average child. These characteristics help to identify high-risk families that can possibly be helped by early counseling.

The diagnosis of poisoning is principally made from the history. The informant is usually a parent, sibling, friend, or neighbor. Whenever an unusual set of signs or symptoms exist, a diagnosis of poisoning should be sought. Such findings as unexplained rapid breathing, stupor, coma, delirium, and convulsions suggest ingestion. Signs and symptoms may be either non-specific or specific, such as fever, flushing, and mydriasis which accompanies ingestion of toxins containing anticholinergies. Arena /8/ provides an excellent categorization of signs and symptoms related to toxins. When the poison or the amount ingested is unknown, one should order a laboratory analysis, but often analyses require hours or days and this delay should not restrict supportive therapy. Since most poisonings are associated with only a few drugs and toxins, simple presumptive diagnostic procedures must suffice until confirmation is provided by more detailed studies./9/

The management of poisoning consists of removal of the poison, use of an antidote where indicated, and supportive therapy. When exposed body surfaces such as the skin or conjunctivae are contaminated, they should be irrigated generously to remove the toxin. Water is the preferred irrigant, but when it is not available, soft drinks, beer or fresh voided urine may be used.

The most efficient method of removing an ingested poison from the stomach is pharmacologically induced vomiting. Induced vomiting is superior to gastric lavage /10-12/ and mechanically induced vomiting./10,13/ Ironically, two out of three ingestions reported to the American poison control centers are managed by lavage./14/ The two agents used to induce emesis are syrup of ipecac and apomorphine. In general, a patient should not be made to vomit if he is unconscious, convulsing, or he has ingested a corrosive, strychnine, or

hydrocarbon. In children ages one to four years, 15 cc of syrup of ipecac is given orally. If vomiting does not occur within 15 minutes, the dose is repeated. Apomorphine (0.1 mg/kg of body weight)is administered subcutaneously or intramuscularly. Vomiting occurs in 90 to 100 percent of cases within 15 minutes with apomorphine, and in 80-90 percent of cases within 30 minutes with syrup of ipecac./15,16/ If vomiting does not occur within the above time limits, gastric lavage should be started. With both drugs, vomiting is enhanced by administration of 200 cc of water either orally or by tube. Prolonged vomiting or excessive drowsiness due to apomorphine may be treated with levallorphan tartrate (0.02 mg/kg) or nalorphine hydrochloride (0.1 mg/kg) intramuscularly. At the present time, apomorphine is considered contraindicated in cases where moderate to severe central nervous system depression exists.

Activated charcoal is an effective, safe preparation that may be used to remove poison from the gastrointestinal tract. Neglected in the past, this agent has now become a valuable addition to the therapy for poisoning. Several preparations differ in their absorptive capacity -- Merck & Co., Inc.'s activated charcoal, Norit A (American Norit Company) and Nuchar C (Virginia Pulp and Paper Company appear to be the best commercial preparations./17/ The complex formed by the activated charcoal and poison is virtually completely stable /18,19/ and does not significantly separate farther down the gastrointestinal tract as previously reported./14/ The dose of activated charcoal must be at least ten times the amount of the ingested poison, /17/ - up to 100 grams is well tolerated./20/ If food has been ingested within the last 6 hours, a larger dose of activated charcoal is necessary. Many compounds (salicylates, barbiturates, glutethimide, dextroamphetamine, chlorpheniramine, dilantin, chlorpromazine, meprobamate, iodine, phenol, and methyl salicylate) are effectively absorbed by activated charcoal. /20,21/ Removal of toxins less efficiently absorbed is enhanced by larger doses of activated charcoal. Activated charcoal is ineffective against mineral acids, strong bases, and sodium metasilicate./20/ In propoxephene ingestion, the use of activated charcoal may be extremely valuable./22/ The combination of activated charcoal and apomorphine is more effective than either agent alone - at least in salicylate intoxication./19/ Activated charcoal should not be used within 30 minutes of administration of ipecac syrup, because the ipecac is adsorbed and vomiting may be prevented.

Once the poison has been absorbed, treatment consists of enhanced elimination, specific antidotes or antagonists, and supportive care with prevention of complications. Elimination may be enhanced by increasing the metabolism, such as using thiosulfate in treatment of cyanide poisoning or increasing renal clearance. Increased renal excretion is accomplished through forced diuresis and alteration of the urine pH where applicable. Forced diuresis is indicated in severe poisoning with barbiturates, amphetamines, salicylates and other toxins primarily excreted by the kidney. One must use osmotic agents (mannitol or urea), or diuretics in addition to copious amounts of fluids to effect a diuresis. A sustained urine flow of 0.1 ml/min/kg body weight is the goal./23/ The central venous pressure should be monitored to prevent fluid overload, and electrolytes should be closely followed to prevent derangement. The urine pH should be alkaline when treating barbiturate or salicylate toxicity, and it should be acid when treating amphetamine or strychnine. An acid urine is produced with intravenous ascorbic acid, ammonium chloride, or arginine hydrochloride./24/ Alkalinization is produced by THAM or infusion of sodium bicarbonate 2 mEq/kg for the first hour and 2-4 mEq/lg over the next 6-12 hours./23/

A more sophisticated method of enhancing elimination is peritoneal dialysis or hemodialysis. These procedures should be reserved for the severest of poisonings where a potential fatal dose has been taken, or there is progressive deterioration with conservative therapy. Should renal failure coexist, then dialysis is mandatory. Hemodialysis is four times as efficient as peritoneal dialysis. Dialysis has been most successfully used in the treatment of barbiturate overdosage and is indicated when the blood levels are above 3.5 mg/100 cc (short acting) or 8.0 mg/100 cc (long acting)./25/ A review of the use of dialysis in poisoning with an extensive bibliography is furnished by Maher and Schreiner./25/

A few poisons can be treated directly by means of an antidote or antagonist. A list of antidotes is given in Table I. An example of an antagonist is the use of ethanol for treatment of methanol intoxication. Ethanol inhibits the dehydrogenation of methanol to its toxic products by successfully competing for the same enzyme system. Another example of antagonism would be the use of sedatives for the treatment of overdosage with analeptic or dysleptic drugs./24/ However, drugs which are stimulants should not be used in the therapy of sedative overdosage./23,24,26/

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TABLE I*

ANTIDOTES FOR SOME TYPES OF POISONS

POISONS	ANTIDOTES
Organic phosphate insecticides	Protopam chloride Atropine sulfate
Inorganic cations	
Mercury	British anti-lewisite (BAL)
Arsenic	BAL
Lead	Versene, BAL
Iron	Desferrioxamine
Cyanides	Nitrite, thiosulfite
Narcotics	Nalorphine levalorphan tartrate
Warfarin	Vitamin-K
Dicumarol	Vitamin-K
Methemoglobin	Methylene blue

*Compiled from Kaye /27/.

Supportive care is probably the most important aspect of the management of poisoning. Unless specific indications exist, one should not, as a rule, use drugs as therapy for ingestion of other drugs or chemicals. The most frequent complication of poisoning is aspiration. Steps must be taken to prevent this complication. Other aspects of supportive care include maintaining respiration, acid-base balance, electrolyte balance and blood pressure. The management of the poisoned child requires removal of the poison using the principles outlined in this paper, using specific treatments and antidotes when indicated, and sound supportive care.

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Dear DOCTOR TAYLOR: – Not so long ago Dr. L., of a neighboring village, was treating a nondescript case the chief characteristic of which was occipital distress. Under his ministrations the case was steadily growing more grave–it was getting positivly *[sic] graveward.* . . He had been giving the patient five or six powerful drugs in huge doses. . . . He was simply adding drug poison to the already exisiting morbid condition, and thus diminishing his patient's chance for recovery.

It was scarcely courteous in me, but I askt him why he didn't give nature a *little* chance.

. . . . . .

This over-drugging habit–the necessary outcome of a form of superstition–what shall we do about it?. . . I think that every experienced and thoughtful physician will admit that hypermideication is the greatest fault of the general medical profession. He can easily do this if he will but remember two things: First, that it is ten times as easy to give too much of the *right* drug as it is to give too little of it; and second, that–owing to our ignorance–we are ten times more liable to give the *wrong* than the right drug. . .

Cleves, O. W. C. COOPER*

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DRUG ABUSE

MAJ Joe M. Sanders, Jr., MC

In the past decade an epidemic has emerged and spread unchecked throughout our society. This epidemic takes many forms and goes by many names, but the form which is causing the most concern is the one involving young people and is most commonly referred to as "drug abuse". This illness, like those in which some micro-organism is the etiologic agent, does not represent some new phenomenon, but rather the emergence of a situation which has existed through the years in a more dormant state. As with any epidemic, the predisposing factors exist in varying degrees of suppression, patiently awaiting the time when there is a relaxation and a realignment of those forces which have kept it in check. When its time comes, as it inevitably does, the epidemic raises its ugly head and races rampant through an unsuspecting population. Such is the case today - the use of illegal drugs by young people has reached epidemic proportions.

We have had the problem of drug abuse in this country for many many years. Long before the New World was discovered by the European explorers, the Indians of what is now Mexico and the American Southwest were ingesting the hairy buttons which grow on top of a particular cactus plant in order to produce visual and auditory hallucinations./1/ The drug they were using was peycte. This crude drug and one of its alkaloids, mescaline, is still legally employed in the ritual services of the Native American Church.

The hemp plant Cannabis sativa is a freely growing weed throughout much of the United States. Western folklore is rich with accounts of cattle, and the cowboys who herded them, being affected by "laco weed". In the 19th Century this weed was the source of a drug legally prescribed in the Western world as a cure for a variety of ailments ranging from rheumatism to painful menstruation./2/ This drug is known today as marihuana (marijuana) or "pot", and is now illegal.

If Columbus himself did not introduce to the New World the process of fermentation and distillation of fruits and grains to produce ethanol, then surely one of his early successors did! Alcohol is today, just as it always has been, the primary drug abuse problem in the country. In 1968 there were some 64,000 recorded narcotic addicts in the United States as opposed to approximately 5 million known alcoholics./3/ Yet, alcoholism has never really been considered a drug abuse problem. Perhaps this reflects the opinion of anthropologists - each society tolerates a single intoxicant, and alcohol is the one permitted in the culture of Western man.

What is Considered Drug Abuse?

Basically there are two forms of drug abuse - legal and illegal. Of the legal forms the most common is alcoholism. A second form of legal drug abuse is the vending of various medicinal agents sold either over the counter or upon presentation of a doctor's prescription. When one picks up a newspaper or turns on the television, he is literally bombarded by a barrage of advertisements of drugs which will cure "headache number 27", which will "relieve the blahs", which will "soothe tired, aching muscles", or which will produce "safe and gentle sleep, sleep, sleep". These drugs are readily available to anyone who can afford the purchase price, and there are no restrictions on the quantity which can be obtained. It is reasonable to speculate that there is not a medicine cabinet in the average American household which does not contain at lease one vial of medicine obtained as the result of a physician's prescription. There are antibiotics and antihistamines, digitalis and diuretics, a wide variety of tranquilizers, appetite suppressants, sleeping remedies, mood elevators and on and on through the entire spectrum of drugs developed to treat non-organic maladies. People can become just as "addicted" to these legal prescription and non-prescription drugs as they can to drugs sold on the illegal market. Is the housewife who takes a diet pill to get her going in the morning, several tranquilizers to get her through the day, and a sleeping pill to sleep at night - all drugs prescribed by her doctor - any less a drug abuser than the adolescent who takes LSD to escape the troubles of his world? Is the teenager who smokes pot to "turn on" really any different from the business executive who has to consume several martinis in order to unwind at the end of the day? These are, of course, philosophical questions,

and are beyond the scope of this paper. Nevertheless, they do serve to emphasize that drug abuse does take many forms. The one that concerns us today is the utilization of illegally obtained drugs by young people. When one speaks of "drug abuse" he is generally referring to the teenager or young adult who is "smoking pot", "dropping acid", or "shooting speed". Indeed, this is drug abuse, and it has definitely reached epidemic proportions in our society. This epidemic is not confined to the ghettos or to the lower strata of our socio-economic structure. It reaches beyond and potentially involves every household regardless of social class, of religious beliefs, or ethnic background. The remainder of this paper, then, will deal with the problem of adolescents and their abuse of illegal drugs.

In general, there are three groups of drug users among the adolescent population. First, there are the "experimenters". This group, fortunately, encompasses the majority of young people who take illegal drugs. It includes the kids who smoke pot, either at a social gathering involving a group of peers or alone behind the security of a locked bathroom door. It includes the high school and junior high school students who ingest one or several "uppers" or "downers" given or sold to them at a nominal fee by one of their classmates. It includes the college student who ventures to the weekend rock festival and "drops some acid" in order to better "perceive the vibes". The reasons these young people take drugs are varied. Most do so out of curiosity. The mass media is flooded with articles about drugs and their effects, and this is a prime topic of conversation on campuses from the grammar school level through the graduate college level. This gives rise to a second major cause, the pressure to do the "IN-thing" with peer groups. The third major reason for young people to use drugs is rebellion, to "blow the minds" of adults. These young people usually do not develop dependence upon drugs. This represents, then, drug abuse in its most popular but mildest form. Even this, however, can have reprocussions. Overdosage, ingestion of drugs of dubious quality and composition, and the psychological effects of a "bad trip" are among the hazards; and one cannot overlook the fact that experimentation can lead to actual dependence.

The second group of adolescent drug abusers are the "oblivion seekers". This group includes those young people who turn on with drugs in order to turn off the world. They take drugs to escape from reality, to avoid the day-to-day stresses imposed upon them by society. It is from this group that the problem of drug abuse among young people had its

inception. In 1965 and 1966 the Haight-Ashbury and other meccas of the hippie world suddenly sprang into existence./4/ Here young people began to congregate to express their dissatisfaction over the social mores which had been imposed upon them. They demonstrated their position by adopting a new style of dress, a new style of music, and a virtually new language; by protesting against those evils - ranging from war to pollution - for which they blamed the Establishment; and by trying to exist in a world of total unconcern for the Almighty Dollar. They turned away from reality by creating their own world of psychedelic sights and sounds, and they used drugs as their main avenue of escape. They turned on with marihuana and hashish, with peyote and mescaline, with LSD and STP, and with the various amphetamine preparation. This "hippie world" attracted adolescents from the "straight world" by the thousands. Some came and stayed. Others communicated back and forth between the two worlds on weekends and vacations. This latter group took back to the straight world the ideology, the dress, the music, and the drugs. This resulted in the emergence, directly and indirectly, of the previously mentioned category of adolescent drug abuses known as the "experimenters".

This impact of the hippie culture on the drug scene can be extended to include not the creation but certainly the expansion of the ranks of the third category of drug abuse among adolescents, the "hardcore addict". The hippie world of the mid-1960s degenerated into three fractions. Many left and returned to the straight life, retaining various aspects of the new culture they had experienced including drugs (thus, further fostering the "experimenter" group). Others chose to retain the newly adopted life style, but found it necessary to leave the meccas and form their own individual groups known as communes (this constitutes the majority of the remaining "oblivion seekers", although many of these individuals no longer require drugs as an escape mechanism). The unfortunate third fraction chose to remain in the meccas, and were encompassed in the changing drug scene which has deteriorated from one of psychedelic drug usage to frank narcotic addiction. This latter fraction, then, joins the ranks of the hardcore addicts. This group had existed in our country long before the establishment of the hippie movement. It is composed primarily of psychiatrically unstable individuals, of the socially and economically oppressed who deem themselves incapable of existence in any other state, and of those individuals who drifted unchecked through the less offensive forms of drug abuse until they finally became addicted to the opiates. This group of drug abusers become not only a menace to themselves

but also a burden to society, for their habit is expensive. It costs the addict from $15.00 to $100.00 a day to support his habit.[14] To raise that kind of money he must resort to a life of crime ranging from dealing in the illegal drug market, to theft, to prostitution. This certainly is the most offensive category of drug abuse, and the one which most desperately needs to be controlled and rehabilitated.

MANAGEMENT

When one considers the problem of management of drug abuse, two areas must be investigated. The more important aspect is prevention; however, we must certainly be prepared to provide emergency treatment of the complications arising from drug abuse. Medical history recalls many epidemics which man has conquered. These were usually squelched by either eradicating the causative organism or by developing a method of immunizing the population against the harmful effects of the organism. It is obvious that we will never have a vaccine which will prevent drug abuse, and it is equally obvious that we will never eliminate drugs - even the illegal drugs - from our society. Prevention is still the treatment of choice in the drug abuse problem, but prevention will not come by sealing off the border to Mexico, or by tightening the security grips on those ports through which passes the trade from the Middle East and the Far East, or by mass arrests of known drug users and even known drug pushers. These methods have been attempted, and they have failed. The only way to prevent drug abuse is through education. It is imperative that we teach young people of the potential dangers of experimenting with drugs. However, it is equally imperative that we educate ourselves in order to better understand why young people resort to drugs. We must recognize that drug usage is really only a symptom of some underlying emotional or physical abnormality. We must provide our youth with other avenues for help in dealing with their problems in order that they not turn to drugs as an avenue for escape. Perhaps if we critically examined ourselves and the world we have created for our young people, we might even take time to listen to some of the suggestions offered by young people to make our world a better place for everyone to live in. In any event, the real solution to the drug abuse problem lies in prevention, and this can only be achieved through programs of education and understanding.

The other aspect of drug abuse management is the specific treatment of emergencies resulting from drug abuse. It would be impossible to outline a course of management for the various adverse reactions to drugs which one might encounter in an emergency room because they would be infinite in number. Instead, I would like to discuss a few basic principles which are applicable in most cases.

First of all, one might adopt as his motto, "Don't just do something, stand there". This is particularly true when one has no idea which drug has been administered. There are no specific antidotes, and to treat one drug reaction with another drug is often simply adding more fuel to the chemical fire. There is one exception to this rule. Nalorphine (Nalline®) and levallorphan (Lorfan®) are specific antagonists to the opiate alkaloids and related synthetic compounds (morphine, codeine, heroin, meperidine, and methadone); however, if these antagonists are given to an individual who has not been dosed with one of the morphine type drugs then they themselves can produce actions which resemble those of morphine. As a general rule, when a person comes in with a suspected drug reaction, do not first think of antidotes and antagonists; but rather think in terms of close observation and support of vital signs.

Although there are many adverse reactions to drugs which require emergency treatment, the two general problems most commonly encountered are (1) those associated with overdose of drugs in the amphetamine, narcotic, and barbituate/tranquilizer categories, or (2) psychological effects of a "bad trip" associated with the hallucinogenic categories of drugs. In dealing with overdosage we are particularly concerned with supporting vital signs and eliminating the drug from the body. We must attempt to determine what drug has been taken by interviewing the patient or those who have brought him to the hospital, but we can not wait until laboratory analysis gives us this information which may take several days. The patient should be examined for clues as to which drugs might have been taken and the possible route of administration (needle marks and thrombosed veins are the hallmarks of the "mainliners"; inflamed and irritated nasal mucous membranes indicate inhalation of narcotics); his state of consciousness and reflex reactivity should be evaluated and his vital signs closely monitored. Then we must assimilate all this information in an effort to determine which drug has been taken and how much. Once we know this, then we can institute specific therapy. (Further information can be obtained by making reference to standard toxicology texts or by contacting available Poison Control Centers.)

The other category of drug abuse complication that requires emergency medical treatment is the "bad trip" experienced by the hallucinogen user. Little is known as to the etiology of these adverse reactions, and therefore there is no specific management. In general, these patients are best handled by maintaining close and unstressful contact with them; they are in essence "talked out" of their "bad trip". Such patients usually present either in a panic state or in a state of acute depression. If the former is the case and the patient terribly aggitated, then small doses of mild tranquilizers or short-acting barbiturate may be necessary to calm him down. Once this is accomplished then he should be treated just like the patient who is acutely depressed. He should be placed in a quiet, unstressful, non-punitive environment and treated with large doses of "Tincture of Time" and Tender Loving Care ("TLC"). Above all, close human contact must be maintained with the patient until he recovers. This contact can be accomplished by anyone (doctor, nurse, orderly) and really requires no special skill or experience if the attendant is calm and understanding, and reassures the patient that everything will be all right.

COMMENT

We are experiencing an epidemic in this country and it concerns primarily young people and their abuse of illegal drugs. Drug abuse is really only a symptom of some underlying disease. Our main role as physicians should be directed at preventing the further spread of this ugly disease. We must accomplish this by, first of all, determining why young people are using drugs and then educate society as to their needs. We must also be equipped to handle the emergency situation which results from adverse reactions to drug abuse (e.g.: overdose or "bad trip").

TABLE I*

DRUGS, SYMPTOMS, AND TREATMENT

PHYSICAL SIGNS	GENERAL DESCRIPTION	COURSE	TREATMENT
→ **Amphetamines:** methamphetamine; mephentermine; phenmetrazine; dextroamphetamine			
Emaciation and poor hygiene; needle marks in IV users; tremors at times. No clouding of senses unless other drugs (alcohol and barbiturates) taken concomitantly. Occasional disorientation with phenmetrazine.	Abuse most common among hippies, women trying to lose weight, truck drivers and night workers	Period of lassitude, sleepiness and depression after discontinuation. Clearing occurs in 5 to 10 days.	Psychiatric hospitalization and withholding of drug. Acidification of the urine with ammonium chloride will hasten excretion.
→ **Barbiturates and other sedatives and minor tranquilizers:** pentobarbital, secobarbital, sodium amobarbital, glutethimide, meprobamate, chlordiazepoxide, etc.			
Intoxication: no specific diagnostic signs. Ataxia, nystagmus, dysarthria, and impaired coordination are characteristic; depression; disorientation; concentration impaired.	Overdose usually presents picture of sedation and depressed level of consciousness; sometimes that of intense aggressive behavior as in acute inebriation.	Usually clears within 24 to 48 hours, depending on dose taken.	*For overdose:* supportive care or hemodialysis depending on severity. For inebriation syndrome: hospitalization; if needed, restrain.
→ **Hallucinogens:** lysergic acid diethylamide (LSD), mescaline psilocybin, dimethyltryptamine (DMT), diethyltryptamine (DET)			
Some pupillary dilation, mild tachycardia. Usually hyperalert but patients may show preoccupation with perceptual distortion.	Possible initial paranoia; confusion; fear of permanent change. Homicide and suicide have occurred.	Rapid recovery, usually within 3 days.	Requires only hospitalization, supportive environment, sympathetic nursing, reassurance that symptoms result from drug ingestion. Chlorpromazine in individualized doses.
→ **Heroin**			
Pinpoint pupils (less contraction in seasoned addicts), slow pulse and respiration, needle marks, sometimes pulmonary edema. In overdose: coma, drowsiness.	Rush of euphoria when injected intravenously. Psychic and physical dependence with withdrawal sickness; preoccupation with drug-taking.	Use continued to avoid withdrawal sickness or fear of it as well as to obtain euphoria.	*Overdose:* artificial respiration where necessary; nalorphine hydrochloride, not to exceed 40 mg total dosages; stimulants; stomach lavage if drug taken orally. *Withdrawal:* should be undertaken by specialists at hospital or therapeutic center.

Continued on next page

TABLE I*, *concluded.* DRUGS, SYMPTOMS AND TREATMENT

PHYSICAL SIGNS	GENERAL DESCRIPTION	COURSE	TREATMENT
→ **Marijuana** Euphoria with no physical signs of intoxication; odor of burnt leaves or hemp on breath or clothes. Red conjunctivae with large doses. Initial stimulation followed by mild sedation.	Acute paranoid reactions and depersonalization states may precipitate schizophrenic reactions in latent schizophrenics.	Reactions usually clear as pharmacologic action abates. Heavy use may lead to bronchitis or conjunctivitis.	Acute reactions should be treated in much the same way as acute reactions to hallucinogens.

*Adapted from *The House Physician Reporter*, August-September 1970, page six.

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