ABSTRACT OF CURRENT LITERATURE

ACUTE INFECTION

CONCURRENT IMMUNIZATION AGAINST TETANUS, DIPHTHERIA AND PERTUSSIS: J. J. Miller and T. M. Saito. *Journal of Pediatrics*, **21**: 31, 1942.

The following study differs from those previously reported in that it was a trial of concurrent immunization against pertussis as well as against tetanus and diphtheria. The simultaneous administration of three or more antigens has been adopted in France under the leadership of Ramon. He has advanced evidence indicating that the effect of each antigen is thereby enhanced rather than impeded. Seventy-six children were given phase I. *H. pertussis* vaccine and fluid combined toxoids. One hundred children received vaccine and alum-precipitated combined toxoids. The routine time schedule of injections is shown below. However not all patients followed the schedule exactly.

Concurrent Immunization Schedules

First Series with Fluid Toxoids	*Second Series with Alum Precipitated Toxoids*
1 c.c. of combined diphtheria and tetanus fluid toxoids containing 20 billion H. pertussis.	1 c.c. of combined diphtheria and tetanus alum-precipitated toxoid and in other arm H. pertussis vaccine, 20 billion (1 c.c.).
Two-Week Interval	
1 c.c. of fluid diphtheria toxoid and in other arm H. pertussis vaccine, 30 billion (1·5 c.c.).	H. pertussis vaccine, 30 billion (1·5 c.c.).
Two-Week Interval	
H. pertussis vaccine, 30 billion (1·5 c.c.).	H. pertussis vaccine, 30 billion (1·5 c.c.).
Four-Week Interval	
1 c.c. of fluid diphtheria toxoid and in other arm 1 c.c. of fluid tetanus toxoid.	1 c.c. combined diphtheria and tetanus alum-precipitated toxoid.

FOUR-WEEK INTERVAL

Smallpox vaccination.	Smallpox vaccination.

TWELVE-WEEK INTERVAL

1 c.c. of fluid tetanus toxoid (Schick test).	0·5 c.c. alum-precipitated tetanus toxoid (Schick test).
Total fluid diphtheria toxoid 2·5 c.c.	Total alum-precipitated diphtheria toxoid, 1 c.c.
Total fluid tetanus toxoid, 2·5 c.c.	Total alum-precipitated tetanus toxoid, 1·5 c.c.
Total pertussis vaccine, 80 billion.	Total pertussis vaccine, 80 billion.

Concurrent immunization of 100 children with combined alum-precipitated diphtheria and tetanus toxoids and H. pertussis vaccine yielded satisfactory results as determined respectively by Schick tests, tetanus antitoxin titrations, H. pertussis agglutination tests, and exposures to whooping cough.

Concurrent injections of fluid diphtheria and tetanus toxoids, together with H. pertussis vaccine, yielded relatively poor responses in tetanus antitoxin but satisfactory immune responses with respect to diphtheria and pertussis.

For tetanus immunization, a three-month interval between the first and second injections of combined alum-precipitated toxoids is recommended. A third injection of alum-precipitated tetanus toxoid (without diphtheria toxoid) is indicated three or four months thereafter. Although this dosage may be more than necessary to secure basic immunity, it is desirable in that it results in the maintenance of high levels of antitoxin for a year. Such high levels would be important in instances in which toxoid reinjection at time of injury was neglected or impossible.

PERTUSSIS PROPHYLAXIS: JOHN F. COPPOLINO. *Journal of Pediatrics*, **21**: 348, 1942.

The use of pertussis vaccine as a means of active immunization against whooping cough has been neither fully evaluated nor universally recognised.

The vaccine used was a heavy suspension of hemophilus pertussis (phase I). Three successive subcutaneous doses of 20 billion, 40 billion, and 40 billion bacilli were given at two or three week intervals at approximately the seventh month of age. Agglutination, complement fixation, and opsonocytophagic tests have been used to determine the development of immunity.

A vaccine of pertussis bacilli (phase I) given at two or three week intervals in three successive subcutaneous doses of 20 billion, 40 billion, and 40 billion organisms appears to be of definite value in the prevention of pertussis.

Although no absolute statement can be made, one may say that immunity may be conferred as early as two months following vaccination.

The duration of immunity appears to be from three to four and one-half years or more.

It is recommended that immunization be carried out routinely at approximately the seventh month of age.

CHRONIC INFECTION

PRIMARY TUBERCULOSIS: E. M. JONES, T. N. RAFFERTY and H. S. WILLIS. *American Review of Tuberculosis*, **46**: 392, 1942.

Clinical pulmonary tuberculosis in children runs a more or less predictable course. Following the primary focus in the lung, there is associated infection and enlargement of the tracheobronchial lymph nodes draining the involved area. In the large majority of cases healing takes place over a period of months or years, with gradual resolution of the pulmonary lesion, return of the lymph nodes to normal or nearly normal size and eventual fibrosis or calcification of the infected tissue—the entire process eventuating in what is known as the primary complex. This course represents the largest group of cases encountered in children.

In a small percentage of cases, due possibly to excessive dose, accidental rupture of a caseous node into a bronchus or blood vessel, "poor resistance," or, possibly, certain intangibles about which we know little, the infection may manifest itself as an extremely severe and even rapidly fatal process. This type and course is also too well-known to require elaboration.

A third group includes the rather considerable number of cases of primary tuberculosis which progress and as "reinfection" tuberculosis, run a checkered and often down-hill course. Extensive pulmonary tuberculosis and tuberculous pneumonia may feature in this group.

In the fourth and last group fall the cases that have been called epituberculosis and many other cases of bronchial obstruction that masquerade as extensive pulmonary tuberculosis or tuberculous pneumonia.

Evidence has been presented in this discussion in support of the hypothesis that the syndrome is essentially a lobar or lobular atelectasis, occurring as a result of interference with pulmonary æration and drainage; this interference arises during the active phase of the primary complex and most frequently is a direct result of extension of the tuberculous process into the bronchial wall from infected peribronchial lymph nodes. It may consist of simple mechanical obstruction by an enlarged lymph node or tuberculoma; or it may be due to interference with ciliary and peristaltic motions. Repeated correlations of bronchoscopic and roentgenographic evidence, as described, are thought to furnish definite proof of the validity of this hypothesis.

Atelectasis occurs in children with primary tuberculosis much more frequently than is usually thought and its possibility should always be

borne in mind in the child whose pulmonary film shows a dense, homogeneous shadow.

This term epituberculosis has for many years been used to designate a clinical syndrome of which the essential feature is lobar consolidation, benign in nature and occurring in tuberculin positive children. The pathological basis for this phenomenon has never been understood clearly.

BRONCHIAL OBSTRUCTION AND PULMONARY ATELECTASIS: E. M. KENT. *American Review of Tuberculosis*, **46**: 524, 1942.

The incidence of pulmonary atelectasis during the course of childhood tuberculosis has been recognized in recent years, and the literature contains confirmatory evidence of this complication from several sources. There are those who have become convinced that the massive roentgenographic shadow, commonly diagnosed as epituberculous pneumonia, is in reality an atelectasis which may involve an entire lung, one or more lobes or a part of any lobe.

The bronchial obstruction can be of two general types, namely, the result of intrinsic tuberculous processes within the wall of the bronchus and of compression of the wall of the bronchus from without, notably as a result of increase in size of adjacent lymph nodes. Marked enlargement of peribronchial and hilar lymph nodes is common in childhood tuberculosis.

A group of cases of pulmonary atelectasis in patients with childhood tuberculosis comprises the material upon which this study has been based.

The principal objective of this clinical problem has been an effort to determine whether bronchiectasis can be shown to be a sequela of pulmonary atelectasis occurring in the course of childhood type tuberculosis. A secondary objective is to add evidence to the contention that pulmonary atelectasis does occur during the course of childhood tuberculosis and that such cases do represent clinical pictures commonly but erroneously considered to have been instances of epituberculous pneumonia.

The universal factors acting in any instance of bronchial occlusion to produce secondary bronchiectasis are the degree of obstruction, the duration of the occlusion of the bronchus and the presence and the severity of the infection set up in the pulmonary segments compromised by the bronchial closure. The one factor upon which there has been a reasonably satisfactory control is the duration of bronchial obstruction and consequent pulmonary atelectasis. In general those instances in which the time interval has been less than six months have escaped secondary bronchiectasis.

The absence of residual bronchial stenosis of any appreciable degree lends support to the contention that the obstruction of the bronchus is the result of extrinsic compression rather than of intrinsic bronchial disease.

It is worth nothing that the expected shift of the mediastinum and its contents has been demonstrable only when an entire lung became atelectatic. Obstruction of a primary bronchus with lobar collapse has not brought about this change.

The cases of lobar atelectasis were not particularly ill during the course of the bronchial occlusion. This indicates that there has been relatively little infection in the affected areas of the lung. Such observations suggest that these patients might easily pass without detection under less favourable circumstances for diagnosis.

Wheezing, rhonchi and musical rales were present in several of these cases and were suggestive of an asthmatic syndrome. This is a possible pitfall in diagnosis.

The question arises whether cases of this sort contribute appreciably to the incidence of infected bronchiectasis. This series of cases presents a much larger percentage of upper lobe bronchiectasis than is seen in any general study of the problem of infected bronchiectasis and a natural conclusion is that this syndrome plays little part in the whole picture. The fact that not one of these children provides symptoms of infection, as yet, is of some confirmatory value in assessing the importance of the syndrome.

TUBERCULIN SENSITIVITY IN CHILDREN WITH BONE TUBERCULOSIS: P. J. HOWARD, J. A. JOHNSTON and C. L. MITCHELL. *American Review of Tuberculosis*, **46**: 532, 1942.

Sensitivity to tuberculin was measured by using dilutions of OT and determining thresholds of reaction in 19 patients with bone tuberculosis.

The initial threshold reactions in 4 children, cases 1 to 4, at the start of bone involvement were 0·1 to 0·01 mg. OT. These levels subsequently rose to 0·000,1 to 0·000,000,01 mg. and then receded again to levels of 0·01 and 0·000 mg. with the healing of the process.

A second group of 7 children followed the same rise and fall pattern of allergy but did not appear for treatment at the start of bone involvement.

A group of 5 children, cases 15 to 19, showed a high plateau of sensitivity for from three to six years, associated with poor ability to heal, and one death from constant spread of tuberculous infection.

All children showed a positive test at the start of bone infection, in one case four months before bone destruction could be seen on an X-ray film.

On the basis of this admittedly small series, it is concluded that a rise to high level of sensitivity with a subsequent fall is characteristic of the evolution of the healing process in bone tuberculosis, and that sustained high levels of allergy have characterized lesions which have shown a failure to heal.

AUTHOR'S SUMMARY

DISEASES OF THE EYE

OUTBREAK OF ACUTE CONJUNCTIVITIS OF UNKNOWN ÆTIOLOGY. JACOB H. LANDES. *Journal of Pediatrics*, **21**: 343, 1942.

Epidemics of acute catarrhal conjunctivitis, commonly referred to as "pink eye," are frequent occurrences. Outbreaks usually occur in schools, nurseries, and institutions in the spring and summer months. Most of these epidemics are caused by the pneumococcus and less rarely by the Koch-Weeks bacillus. The infection is spread by direct contact, by fomites, and occasionally through droplet infection from the respiratory tract. Many epidemics of follicular conjunctivitis, the swimming pool variety, have also been reported, the most famous one being that at Baden and referred to by Duke-Elder. That form of follicular conjunctivitis is easily recognized by the discovery of epithelial inclusion bodies. Cases of conjunctivitis in which no organism is present are occasionally seen by ophthalmologists, but outbreaks involving several hundred cases, where no organism is discovered, are rare and infrequent. A description of such an outbreak in which more than 200 children developed a catarrhal conjunctivitis and where the etiology was unknown is hereby reported.

During the first two weeks in May, 1941, thirteen children with flamed eyes who had complained of itching, tearing, and mild photophobia were excluded from school X, a junior high school in the Borough of Brooklyn. The palpebral conjunctivæ were slightly inflamed. Several children also had an inflammation of the scleral conjunctivæ. The symptoms, as a whole, were mild and not particularly disturbing. To the school nurse, the condition seemed to be that of a mild "pink eye" although thirteen children, over a two-week period, constituted more than the number usually seen by the nurse at that time of the year. Still, in view of the large school registration of over 1,600 children, the nurse at school X was not perturbed.

During the next five days twenty-nine additional children with conjunctivitis were excluded, and from four to seven children were excluded each day of the following week. The outbreak reached its peak on June 3 when forty children with conjunctivitis were referred for exclusion. The outbreak terminated on June 13. From May 6 to June 13, 203 children developed conjunctivitis in that school. Based on a school registration of 1,620 as on March 3 of that year, this constituted an attack rate of 12·5 per cent.

Of the forty-seven classes in school X, seven did not have a single case of conjunctivitis. The others had from one to sixteen cases. Fourteen children had recurrent attacks after returning to school, with a variation of from one to sixteen days, the interval of time from the return of these children to the development of the second attack. Two children manifested symptoms of conjunctivitis the day after returning to school, and one child developed symptoms two days later. It is very likely that the latter three children were readmitted without having completely recovered from their original attack.

In eight instances there were two children with conjunctivitis in one family, and in one case there were three children in one family. In the

latter case, two children attended school X and one child attended school Z. In only one case was a history obtained of the development of conjunctivitis in older members of the family. In that household the father, mother, and an older sister developed "eye trouble." In two houses there were two cases of conjunctivitis each and, in one house, three cases of conjunctivitis in children not of the same household.

Conjunctival smears were taken of ten children and forwarded to one laboratory, while smears of ten other children were taken on another day and sent to a different laboratory. All smears were negative for pus cells, bacteria, or endothelial cells. Scrapings of the conjunctivæ were also negative.

This outbreak of acute conjunctivitis presents many unusual etiologic features. The characteristic feature of the outbreak, lasting a little over five weeks and reaching its peak on June 3, doubtless points to an organism as the etiologic agent. The recurrence of the condition in several children after returning to school is another indication of an organism being a factor.

The many smears that we took were all negative for organisms, pus cells, or endothelial cells. Conjunctival scrapings were also negative. If the outbreak was of an infectious nature, the cause beyond doubt was a virus. It is possible that the agent concerned was of such a type that it would not produce any neutrophilic response, similar to the toxin produced by the Morax-Axenfeld organisms or the toxin of *Neissena catarrhalis,* both of which produce little or no neutrophilic response.

The absence of inclusion bodies in epithelial scrapings also does not preclude an infection. According to THYGESON, secretion smears are more satisfactory than epithelial scrapings for the reason that with the exception of Koch-Weeks bacillus and the gonococcus no bacteria are found multiplying on the epithelial cell of the palpebral conjunctivæ.

It is likely that the disease was spread by direct contact and through fomites. The many cases that occurred in several classrooms on one floor, the classrooms being adjacent to a gymnasium, should be taken into consideration. The low pressure of the drinking fountains and the method of handling silverware in the cafeteria are two other possibilities for spreading the infection.

INTERNAL MEDICINE

THE VOMITING DISEASE: J. I. WARING. *Am. J. Diseases of Children,* **64**: 482, 1942.

In the winter of 1941 and again in 1942 there occurred in Charleston, S. C., an epidemic of the disease described by ZAHORSKY as "the winter vomiting disease," or hyperemesis hiemis.

The number of cases was very large, running probably into the thousands, though most of the attacks were of such transient nature that they were never reported to the health department. The disease occurred among patients of all ages, but the incidence was particularly impressive among the younger children of the city. The symptoms were identical with those described by ZAHORSKY—abrupt onset, violent and contniued

vomiting, prostration, dehydration and, after a day or two, diarrhœa more or less persistent. The stools were characteristically light yellow or gray and had, according to numerous volunteered descriptions, a "rotten egg odor." They did not contain any gross signs of mucus, blood or pus. In some instances vomiting was the only symptom. Diarrhœa was usually not of more than two days' duration. Dehydration requiring parenteral fluids for correction was very unusual. In occasional instances the temperature was as high as 104°F. (rectal) but the fever was of short duration. The range of the few white blood cell counts which were made was from 6,450 to 16,000.

The persistent vomiting and the apparent absence of bile from the stools seemed to suggest some disease in the upper portion of the intestinal tract. The numerous children who showed this picture gave no indication of infection of the respiratory tract. A few physicians spoke of a granular appearance of the pharynx, but I did not observe this condition among a large number of patients.

Treatment of this disease was not noticeably effective except when fluids were given parenterally. Restriction of the diet to fruit juices, carbonated drinks, crushed ice and water seemed desirable. Magnesium hydroxide in small repeated doses perhaps was helpful, and enemas sometimes seemed beneficial. Attempts at administration of sulfanilamide or one of its derivatives or of a sedative usually resulted in aggravation of the vomiting.

SEVERE VOMITING IN INFANCY: E. FREUDENBERG. *Schweiz. Med. Wschr.*, **72**: 405, 1942.

Severe vomiting occurs not infrequently in conditions other than spastic pyloric stenosis of infants, and the differential diagnosis may be very difficult, requiring special knowledge and careful roentgenologic examinations. It is very important to determine accurately, as early as possible, how much of the food is actually vomited and this is best done by weighing prepared diapers, collecting the vomit in them, and weighing them again; in this way the severity of vomiting may be judged before loss of weight results. Many writers stress the distinction of spastic and atonic vomiting, but it should be emphasized that the former, although predominant in pyloric stenosis, may occasionally occur in other conditions.

During the first few days of life, pyloric stenosis hardly occurs and malformations should first of all be taken into diagnostic consideration. Atresia of the œsophagus may be diagnosed by probing, and the diagnosis will be definitely established by roentgenologic examination; the prognosis is unfavourable as the infants do not survive the required extensive operations. Other malformations include suprapapillary and infrapapillary stenosis of the duodenum, and other frequently multiple stenosis and atresias; surgical treatment is indicated in all these cases, but will hardly ever be successful. Of much more practical importance than severe vomiting of new-borns caused by these relatively rare anatomic malformations, is the type of severe vomiting caused by functional factors; it occurs between the second and fourth day of life and is characterized by the reflux of bile. Attempts at feeding increase the

irritation and result in increased retching. As a rule, prompt cure will be achieved by lavage of the stomach with warm physiologic saline solution or with warm mineral water. In case of failure of this simple measure, fluids should be administered rectally, subcutaneously, or intravenously: saline or Ringer's solution, mixed with equal parts of a 5 per cent glucose solution, may be used for this purpose.

After the new-born period which includes approximately the first 10 days of life comes the period during which pyloric stenosis develops. The author first discusses causes of severe vomiting other than pyloric stenosis. Too large feedings or too short intervals between them may cause severe vomiting, but it will hardly ever be as intense as in pyloric stenosis; therapeutically, reasonable regulation of the nutrition will be all that is required. Suprisingly, not only overfed but also underfed infants may vomit, particularly infants with nervous irritability, and aerophagy may be observed concomitantly; the clinical syndrome is characterized by a slow weight increase, pallor, rarity and dark color of the fæces, and susceptibility to infections; wrinkling of the forehead, by some considered pathognomonic of pyloric stenosis, may also be observed. Treatment of neuropathic vomiting depends on the provoking cause. When not enough milk is obtained from the breasts, additional food should be given after each feeding until a normal quantity is reached. Very small amounts of luminal may be given as a sedative. In some cases of so-called habitual vomiting no special cause may be demonstrable, and increased irritability of the vomiting center is probably dealt with, requiring treatment with atropine and food of increased consistency.

Atropine sulfate is prescribed in a 0·1 per cent solution in diluted alcohol, and 1 to 2 drops of this solution are placed on the tongue four times daily immediately after meals; the dose may be increased up to 4 drops four times daily, as infants tolerate atropine well. Redness of the face is a sign of overdosage. After the age of two to three months, the food may be made more consistent by adding flour, but in younger infants this addition may lead to fermentative dyspepsia; in these young infants 2 to 3 teaspoonfuls of the more consistent food should be given before the regular thin food, as this quantity will suffice to prevent vomiting and yet will not cause dyspepsia.

Pyloric stenosis differs from other forms of severe vomiting by the phenomenon of gastric peristalsis which becomes visible through the abdominal wall, provided the stomach is filled to a certain extent and the infant keeps quiet; the peristalsis stops as soon as the infant cries or becomes restless, and repeated examinations may be required before peristalsis can be discovered. Another important sign of pyloric stenosis is a palpable tumor, but palpation may be very difficult. The most important diagnostic method is roentgenologic examination which indicates the degree of disturbance of the gastric evacuation. As a rule, evacuation of the stomach in case of pyloric stenosis will not even have begun 20 minutes after administration of mother's milk with 1 tablespoonful of contrast substance, and in severe cases almost no food will have left the stomach after two hours, at which time the stomach would normally be empty; in very severe cases food will remain in the stomach

for up to 24 hours, or no food at all will pass the pylorus. When after two hours more than half of the meal is still found in the stomach, surgical treatment seems indicated.

Medical treatment where indicated consists of feeding numerous small meals of mother's milk, administration of atropine, and skilled nursing care. In very advanced cases, with exsiccosis and collapse, intravenous drip infusions and blood transfusions are required as preparation for operation.

The author then deals with cardiospasm and œsophagospasm; these conditions may simulate pyloric stenosis, but may readily be differentiated from it on the basis of the above-mentioned classic symptoms. Characteristic symptoms of these spastic conditions are the premature onset of the vomiting, often even during the meal, and the fact that the vomit does not give an acid reaction. Roentgenologically the lower third of the œsophagus will be seen to be transformed into a pear-shaped sac. Treatment consists in feeding through an indwelling tube for a period of about four weeks; operation is not indicated.

The author finally deals with rumination; this disturbance involves neuropathic infants during the second half of the first year of life and is usually preceded by habitual vomiting with resulting dystrophy. The infants may enjoy the play of alternating swallowing and ruminating, but a large share of the food is lost in this way, and the dystrophy will become progressively worse. SIEGERT recommended treatment with a gastric tube, provided with a condom; the tube is introduced after each meal, and the condom inflated and clamped so that the infant cannot ruminate. This method is effective, but the author states that other less brutal methods are equally successful. First of all a change from fluid to more consistent food, like vegetables, farina, mashed potatoes, and scraped apples, is made, and second the infant is placed in a half-sitting position. Rumination has psychic cause and must be treated psychologically and pedagogically: it is a neuropathic manifestation caused by boredom and irritative hunger, and occurs only in very intelligent and active infants, while among older children and adults it occurs only in idiots and imbeciles. Such infants require early distraction and appropriate play.

The author finally mentions as causes of secondary non-characteristic vomiting of infancy infectious diseases, dyspepsia, inflammatory abdominal affections, hydrocephalus, meningitis, cerebral tumor, and renal insufficiency.

SUMMARY, *Int. Med. Digest*

GIARDIASIS: SOME CLINICAL AND THERAPEUTIC OBSERVATIONS: A. W. FRANKLIN. *Archives Dis. Childhood*, **17**: 60, 1942.

He summarized the recorded facts about giardia intestinalis, stressing the high incidence of infestation in parasite surveys of children, with a peak at six years, and in dysentery convalescents. Oral atebrine (quinacrine) was a uniformly successful treatment. He described four cases with symptoms thought to be due to giardiasis: one with eleven months of diarrhœa following dysentery, two of delayed mental and

physical growth with abnormal motions, and one of acute afebrile diarrhœa. Common features were the passage of bulky, undigested, mucus-containing stools of most offensive odour, abdominal distension, and partial relief on a low fat, low residue diet. Remarkable clinical improvement allowing a return to normal diet followed atebrine treatment. Giardia cysts had been founded in eighteen children, six without and twelve with gastro-intestinal symptoms. Eight had been treated successfully with oral atebrine, the cysts disappearing from the stools during treatment. One adult, a never failing source of positive stools for ten years, was free of cysts from the fourth day of treatment.

The suggested doses of atebrine (quinacrine) were:—

Age in years	Daily dose in 0·1 Gm. Tablets.	Course in days.	Total dose
½— 2	... ¼ × 2	3—5	0·10—0·25 gm.
2— 6	... ½ × 2	3—5	0·30—0·50 gm.
6— 9	... ½ × 3	4—5	0·60—0·75 gm.
9—12	... 1 × 2	5	1·0 gm.
Adult	... 1 × 3	5	1·5 gm.

GROWTH OF THE LUNG IN HEALTHY AND SICK INFANTS.

STEEFAN ENGEL. *Archives Dis. Childhood,* **17**: 41, 1942.

Combined evaluation of the anatomical and clinical investigations leads to the following data on the growth of the lung in infancy. The right lung of the new-born infant has a volume of 60 to 70 c.c.; this volume is doubled in the first three to four months and quadrupled towards the end of the first twelve months. The left lung is smaller than the right lung; its volume amounts to 75 to 80 per cent of that of the right lung. The decrease in the specific weight indicates that, apart from the growing volume, the relative amount of functional tissue increases as the age and volume increase.

The relationship between wasting of the infant and its respiration has never been considered. The interest of pediatricians has been focussed on the intestinal and metabolic disturbances which are responsible for the deterioration of the general condition.

The impaired growth of the lung explains first the high incidence of collapse and pneumonia in wasting infants; and also suggests a new form of treatment. It might be possible to improve the general condition by influencing the respiration and thus breaking the vicious circle. This might be a considerable help in difficult dietary treatment. This question can, however, be settled only in the wards.

The lung of the normal infant grows fastest in the first few months. It doubles its volume in the first three to four months and quadruples it by the end of the first year.

The growth of the lung reacts in infancy to the slightest change, positive as well as negative, of the general condition, and comes to an almost complete standstill in wasting infants. This implies not only that the lung remains small, but also that the structural differentiation of the tissue is arrested at an early stage.

The arrest of growth and differentiation of the lung explains the high rate of collapse and pneumonia in wasting infants. On the other hand, recognition of the pulmonary impairment suggests that respiratory therapeutics may be an additional aid in the treatment of wasting infants.

ROENTGENOLOGY

ROENTGENOGRAPHICALLY DEMONSTRABLE CAUSES OF CYANOSIS IN THE INFANT AND NEW-BORN: J. F. Bowser. *Journal of the Kansas Medical Society*, **43**: 291, 1942.

The writer brings to one's attention several of the causes for cyanosis in the infant and new-born which can be demonstrated and diagnosed by roentgenographic examination.

The commonest roentgenographically demonstrable cause of cyanosis in the new-born is congenital heart disease. In these cases one sees on "the anteroposterior x-ray film and fluoroscopically a heart larger than normally seen in overall dimension and in transverse diameter. It is often globoid with overprominence of either the right or left silhouette or both. The upper central shadow is usually not wide. Cardiac pulsation as observed fluoroscopically is often of low magnitude.

Clinically a heart murmur may or may not be heard. In one group of congenital heart disease cyanosis is a prominent sign due to the admixture of oxygenated and unoxygenated blood as the result of an arteriovenous shunt. The tetralogy of Fallot is the commonest congenital heart in this group. It consists classically of: (1) pulmonary stenosis, (2) dextro-position of the aorta, (3) interventricular septal defect, and (4) hypertrophy of the right ventricle. In the radiography of the tetralogy the hypertrophy of the right ventricle without the pulmonary conus gives the wooden shoe or *'coeur en sabot'* contour.

It has been stated that a high percentage of cases of congenital heart disease, perhaps around 50 per cent, show no change from the normal contour on the x-ray film, so that many of the cases of congenital heart are not diagnosed in infancy radiographically.

The second most common roentgenographically demonstrable cause of cyanosis is the somewhat controversial enlarged thymus which, when it produces pressure on the trachea and great vessels, causes cyanosis.

The diagnosis of enlarged thymus gland causing symptoms rests on the clinical presence of spells of cyanosis and the radiographic demonstration of a wide upper mediastinal shadow often having a lobulated outline. "It is not always possible to eliminate a congenital heart. The enlarged thymus gland can be reduced in size by the therapeutic application of roentgen ray in suitable small doses."

Congenital diaphragmatic hernia is not a common cause of cyanosis, but the writer observed two cases proved to be congenital hernias through the left diaphragm. "It is easy once one is familiar with the picture of this condition to diagnose it from the x-ray film of the chest and abdomen of the infant, as was done in these cases. Cyanosis was a major sign. Death, when it occurs, is often by asphyxiation. The symptomatology is often entirely related to respiratory system because

of compression of lung tissue and the mediastinum. Congenital diaphragmatic hernia is in the majority of cases on the left, due to a persistent hiatus pleuroperitonealis, the result of failure in fusion of the posterior and lateral segments of the diaphragm. It can occur on the right. Any or all of the abdominal organs may slip through the opening in the diaphragm into the thoracic cage. Early surgical repair is a lifesaving procedure.

Agenesis of the lung is a rare cause of cyanosis in the infant, only 39 being reported in the literature up to 1939. It is difficult of diagnosis from the roentgenographic appearance, the correct diagnosis being usually made at autopsy. Anatomically a rudimentary fragment of lung tissue may be present or there may be complete absence of lung. The bronchus on the one side, if present, is small. Pulmonary vessels to the affected side are absent. The remaining lung is often hypertrophied. The heart and mediastinum may be displaced to the side of the thorax from which the lung is absent, giving the roentgen picture of massive atelectasis of one lung. The small and large intestine or the liver with a high undescended diaphragm may occupy one-half of the thorax.

Spontaneous pneumomediastinum in the new-born must be considered in any case of cyanosis in the new-born as cyanosis is the cardinal sign in this condition. It is a collection of air in the mediastinum, varying in amount and tension, which arrives there by way of the perivascular sheaths of the lung from ruptures of the alveolar bases.

The morbid physiology of pneumomediastinum is a pressure on the great vessels which causes vascular congestion resulting in dyspnœa, cyanosis, and fall in blood pressure. The air may extend from the mediastinum into the neck, retroperitoneally, or into the lung opposite that from whence it came. A pneumomediastinum of severe degree can result in death. Treatment of the condition, if severe, is relief of the air pressure with mediastinotomy.

Large tumor masses in the neck, such as the cystic hygroma or teratoma, can by their presence and proximity to the trachea cause intermittent obstruction of the trachea with periods of cyanosis. In the like manner retropharyngeal and upper medistinal abscesses by their forward displacement of the trachea can be a cause of cyanosis and respiratory difficulty.

Persistent atelectasis or congenital atelectasis in the new-born is that condition in which expansion of the lungs which is normally complete in two to three days of life has not occurred. The most characteristic finding is cyanosis often in attacks. It occurs in infants who are too weak to make the necessary respiratory effort. The x-ray shows irregular linear shadows usually at the bases or a lack of aeration of one or more lobes of the lung.

Cyanosis is often a sign in pneumonia in the infant but, as such, is of minor significance. The radiography is of considerable help in the diagnosis of pneumonia in the infant, both as to the type and extent of involvement.

SURGERY

MINOR SURGERY IN CHILDHOOD: H. W. S. WRIGHT. *Practitioner*, **149**: 179, 1942.

For the purpose of this article, minor surgery is defined as being concerned with operations which may be undertaken in the course of general or out-patient practice. Most of these operations are not peculiar to childhood, but certain aspects of their management become important because of the patient's age.

Minor surgery is only minor in the eyes of the practitioner: in the minds of the child and the parents it is a major event. In this respect parents are perhaps wiser than is usually admitted. "Anxiety" is the most common complication of any operation and the circumstances in which minor operations are generally performed make this complication perhaps more common after minor than after major surgery. Every child should have some form of premedication, coming to the operating table either asleep or sleepy enough to produce complete anæsthesia. To attain this purpose, the barbiturates are probably the best drugs: the average dose of nembutal is 0·6 grain per stone of body-weight, with a maximum of 3 grains. The barbiturate should be supplemented by atropine, 1/100 grain, half an hour before the operation, as barbiturates tend to produce some œdema of the respiratory tract.

TONSILS AND ADENOIDS

Adenoids.—Infected adenoids may be the cause of three important groups of symptoms:—

(1) Recurrent attacks of rhinitis and sinusitis.

(2) Infection and re-infection of the middle ear.

(3) Chronic gastritis and laryngitis, with a constant cough. The swallowing of infected mucus may upset digestion and irritate the larynx.

Removal of Adenoids.—This operation may safely and satisfactorily be performed in the child's own home, if circumstances permit. Full anæsthesia is preferable, after premedication. The operation is done with the child on his back. a sandbag under his shoulders and his head well back. When he is well under the anæsthetic a gag is inserted and the adenoids removed with a blunt curette of suitable size. After curetting, a finger is inserted to make sure that no tags remain behind, and then a swab packed in the nasopharynx controls most of the bleeding, the main bulk of which occurs in the first few minutes. The pack is then removed, and a swab on a sponge-holder placed behind the soft palate effectively prevents any blood from getting into the larynx. The child is turned into "Sim's" position with his head well back, and the sponge-holder is left in place until the cough reflex returns and the danger of aspiration of blood is over; it is then removed. The child is sent back to bed still in Sim's position.

Tonsils.—Tonsillectomy will cure nothing but recurrent attacks of acute tonsillitis, chronic tonsillitis and its results.

(1) On pressure with a probe, pus can be squeezed from the crypts.

(2) Palpation reveals the presence of a persistently enlarged tonsillar gland situated just behind and below the angle of the jaw.

There is one other indication for tonsillectomy—the enlargement of

a group of glands known as the upper jugular chain, with physical signs suggesting tuberculosis. In these cases, tonsillectomy frequently results in their disappearance.

Tonsils should be enucleated, either by guillotine or by dissection: the latter method is preferable, but it is not minor surgery.

ACUTE OTITIS MEDIA

Acute otitis media is a common sequel to nasopharyngeal infection. It is undoubtedly true that the sulphonamides have revolutionized the treatment of acute otitis media, and that many if not most of the cases now completely subside.

Myringotomy.—Myringotomy is indicated when the drum is red and bulging, or when a pulsating bead of pus on a small perforation shows that drainage is inadequate. A general anæsthetic should be given, and with a good light a myringotome should be inserted in the postero-inferior quadrant, and the drum incised in an upward direction.

ACUTE LYMPHADENITIS

This is commonly secondary to infection of tonsils and adenoids, but it may of course have other origins, such as secondary infection of a tuberculous gland. In either case treatment follows the same lines. While the inflammation is confined to the gland or is spreading to the adjacent supporting tissues, the most important factor in the local treatment is rest. The child should be in bed, and movement of the head limited by sandbags fixed on either side. This simple method relieves pain more surely and effectively than any other form of local treatment. Hot fomentations or antiphlogistine may supplement but should not replace fixation. The spread of the infection may be limited, and resolution hastened by the use of sulphonamides, which should, however, be used with discretion and care: a spreading brawny cellulitis is a definite indication for their use.

Incision is indicated only when the infection is defintely localized. The active cellulitis should be nearly over, and a fluctuating swelling should be present.

Operative Procedure.—The opening of an abscess in the neck is no occasion for "cut and run" surgery. The patient should have full anæsthesia, and a good light should be available. The incision should be adequate and the cavity gently wiped free of pus. A small track will be found in the deep fascia leading to a necrotic gland: this should be enlarged by insertion of the points of a pair of artery forceps and opening them gently. The remains of the necrotic gland should then be curetted away with a sharp spoon, used with just enough force to remove soft necrotic material: any obvious sloughs from the subcutaneus tissue should be treated in the same way. There is no danger in doing this if gentleness and precision are employed, because the fibrotic capsule of the gland is not removed.

Every crevice of the wound is then packed with gauze impregnated with B. I. P. P. or flavine in paraffin, and most of the incision sewn up with a few stitches. In most cases the packing may be left in for about ten days. When it is removed, the cavity will be found to be clean and lined with healthy granulation tissue, and it may be repacked once more.

RETROPHARYNGEAL ABSCESS

Retropharyngeal abscesses sometimes require urgent surgery. It should be remembered that these abscesses are of two types: the acute and the tuberculous, and they may be distinguished by their physical signs, which depend on anatomical considerations. The tuberculous type lies behind the deep cervical fascia and is secondary to tuberculosis of the cervical bodies: onset is relatively slow and palpation demonstrates fluctuation in the middle line. The acute type is secondary to naso-pharyngeal sepsis, and lies in front of the deep fascia, between it and the mucous membrane. Fascia and mucosa are closely attached in the midline, so that the acute retropharyngeal abscess is necessarily situated to one side or other of this line. Occasionally a submucous abscess may spread from tuberculous cervical glands into this lateral situation, but the presence of the glands makes diagnosis easy.

Operative Procedure.—An acute abscess should be opened as soon as it is diagnosed. Light anæsthesia should be given, and the child placed with head well back so that all pus runs down into the nose. The mouth should be opened with a gag, and a tongue depressor inserted. A very small incision should be made in the fluctuating area with a sharp knife, so that pus oozes slowly and may be wiped up at once: when most of it has come away the incision may be enlarged. If the head is well back there is little danger of aspiration pneumonia, although this position may cause some temporary obstruction.

CIRCUMCISION

Another minor operation often required, but more often demanded, is circumcision. It is indicated when:—

(1) The foreskin is long and the prepuce narrow, so that it balloons out when the child passes water.

(2) Balanitis is present.

(3) There is constant irritation and redness of the foreskin.

If it is obvious at birth that it will be impossible to retract the foreskin, operation may be performed on the day after birth without an anæsthetic.

MEATAL ULCER

After circumcision, meatal ulcer is far too common, and is a most distressing complaint. There are two views about its origin:—

(1) That it is due to trauma, i.e., the constant rubbing of delicate unprotected epithelium on the clothes.

(2) That it is caused by ammoniacal urine.

Whatever the cause, to cure it napkins should be changed frequently, washed with non-irritant soap and dusted thickly with boric acid powder, and boric acid ointment is smeared on the glands and put just inside the meatus with a probe after micturition. In older children, a small piece of cotton-wool should be put over the glands to prevent rubbing on the clothes, and this is held in position by a bag. All clothes should be loose.

RECTAL POLYP

Diseases of the rectum provide a number of conditions amenable to minor surgery. Rectal polypi are comparatively common in childhood.

Parents usually say the child has piles: inquiry reveals that straining at stool is frequent and that stools contain a large amount of fresh red blood and mucus. Polypi are a common cause of any material amount of fresh blood in children's stools. Gentle digital examination usually allows a small soft tumour to be felt: if nothing is discovered there should be no hesitation in giving an anæsthetic and passing a small proctoscope. The child should be in Sim's position with the buttock well raised by a sandbag.

Removal of Polypi.—If a polypus is found, its pedicle should be transfixed by a tight ligature, and the polypi removed, leaving a long stump to prevent slipping. Most polypi can be brought outside the anus if the sphincter is adequately relaxed by a local anæsthetic: this is better than deep general anæsthesia.

Anal Fissure

This is a distressing ailment, easily cured. It is perhaps the most common cause of severe pain after defæcation. Pain leads to constipation, the relief of which becomes more and more painful, and a few drops of bright red blood are often found in the stool. A small fissured ulcer will be found at the anocutaneous margin, generally in the middle posteriorly. As in children they are nearly always recent and not indurated, they can be made to heal if the superficial part of the external sphincter is paralysed by an injection of proctocaine. General anæsthesia is necessary to make the injection, for which a rather wide-bore needle must be used. The proctocaine is injected subcutaneously around the anus and behind the fissure. Using the same technique as indicated for novocain, 2 c.cm. are distributed into each ischio-rectal fossa. It is important to see that no proctocaine is introduced intradermally, otherwise sloughing will occur. The pain ceases at once, although the injection may have to be repeated at the end of a fortnight. Excision is rarely required in children.

Prolapse of the Rectum

This is another condition that causes parents a great deal of anxiety. In quite a number of cases, no prolapse is found after admission for observation, although in other cases it may be obvious and extensive, and sometimes ulcerated. Operative treatment is rarely called for. Wasting, straining at stool, and lack of muscular tone are important ætiological factors, and these should be treated. The buttocks should be strapped together, and defæcation should take place with the child lying on his side.

TROPICAL DISEASES

TROPICAL DISEASES: W. E. Cooke. *Practitioner*, **149**: 305, 1942.

Malaria

New Drugs.—Successful treatment of malaria with a new drug "sinine" is reported by Liu, Chang, Ch'uan and Tan (1941) 3 gm. of the dried powder was given three times daily after food to adults. Parasites in all three species of malaria disappeared from the blood in eight days and the splenic enlargement was reduced. No relapses occurred.

"Quinoblen" contains chlorohydrate of quinine 15 cgm., arrhenal 2 cgm., methylene blue 2 cgm., normal saline 5 ccm., and is given intravenously twice or three times a day. DALEAS (1940) claims that it is a safe and effective remedy in grave cases of malaria. It causes bluish pigmentation of the skin. The necessity of frequent intravenous injections makes it suitable only for patients in hospital.

Work on the exo-erythrocytic schizogony of malarial parasites appears to support the hypothesis, first clearly enunciated by JAMES, that some hitherto unrecognized stage of development, probably in cells other than the red blood corpuscles, intervened between the injected sporozoites and the first forms to appear in the red blood cells. Careful observation showed that they entered a cell of the reticulo-endothelial system and developed into schizonts, the merozoites arising from which would seem to be capable of entering either reticulo-endothelial cells to give rise to exo-erythrocytic schizonts, or red blood cells to develop into pigmented blood forms. Should this knowledge prove correct it will open new fields for the discovery of a drug which will destroy the parasites before they reach the stage of blood cell invasion.

THE DYSENTERIES

The successful use of sulphanilylguanidine or sulphaguanidine is accumulating. MARSHALL, BRATTON, EDWARDS and WALKER (1941), reporting on a series of patients with Flexner and Sonne infections so treated, state the results were uniformly good when the drug was given early, on or before the third day, whereas if it was delayed to the fourth to the fourteenth day the results were not uniform. In early treated patients, some appeared well after only twenty-four hours' therapy and the diarrhœa was checked in from one to three days.

The dosage recommended was:—Initial dose 0·1 gm. per kgm. by mouth. Maintenance dose 0·05 gm. per kgm. four-hourly until the number of stools daily is less than four, subsequently 0·1 gm. per kgm. eight-hourly for at least three days. The finely powdered drug is given in milk or water, and should not be continued for more than fourteen days because of the possible danger of agranulocytosis.

Nickel Pectinate.—According to BLOCK, TARNOWEKI and GREEN (1939), in acute and chronic bacillary dysentery, pure pectin is ineffectual but nickel pectinate possesses detoxifying, bactericidal and antihæmorrhagic properties. In ninety-five patients, adults and children, treated, definite improvement occurred in every case. The dose of nickel pectinate given was 2 ounces in cereals or milk t.i.d., later increased to 2 ounces every three hours. The inhibitory action of the preparation on bacterial growth is assumed to be due to ionization of the nickel. Nickel pectinate acts also as a catalyst and the catalytic production of antitoxin and agglutination by it is feasible. It may also aid in the assimilation of vitamins.

LEISHMANIASIS

Complement-fixation tests have been carried out with sera of cases of human and canine kala-azar with antigens prepared from spleens or livers of moles or hamsters experimentally infected with *Leishmania donovani*. As a result of these, CHUNG and LU (1941) report that the complement-fixation test was of definite value in diagnosis, and their

results point to canine kala-azar being identical with the human infectio in China.

Further investigation by SMITH, HALDER and AHMED (1940) show that the phenomenon of blocking, comparable to the blocking of the œsophagus of fleas with plague bacilli, can take place in the sand-fly *P. Argentipes*, and that dissection of such blocked flies shows them heavily infected with flagellates.

Efforts at infection of hamsters and mice with such flies was successful in seven animals out of thirteen. Taking into account epidemiologically how closely the sand-fly *P. Argentipes* is connected with the spread of kala-azar, the results of these tests, even without the final tests of transmission to human beings, leave little doubt that *L. Donovani* in in India is transmitted by *P. Argentipes*, and that sand-flies in genera are vectors of leishmania throughout the world.

The Aromatic Diamides.—The drug 4·4-diabidino-stilbene has been more widely used in India, the Sudan, and this country. It may not cause an immediate fall in the temperature or reduction in the size of the spleen. With both this drug and 4 4-diamidino-diphenoxy-pentane, punctate and nodular skin lesions, in which leishmania may be found, may appear during the course of treatment. These skin lesions disappear rapidly after treatment is completed, thereby differing from the dermal leishmanoid of India. The drug is given intravenously and may cause vomiting, flushing, a burning sensation over the chest and abdomen, weakness, faintness, dyspnœa and headache, and a fall of blood pressure. Relief is obtained by the administration of adrenaline. Dosage is 1 mgm. per kgm. of body-weight, and the injections are given daily for eight or ten days.

SPRUE

Recent observations suggest that the sprue syndrome results from damage to the villi of the small intestine. Phosphorylation of fats takes place on the epithelial surface of the villi. Damage to the villi lessens or prevents the absorption of fats, and may also lead to the non-absorption of vitamins, which is more important than the absence of the latter from the diet of these patients.

The treatment of sprue has improved since the administration of nicotonic acid and of riboflavin, as has been advocated by MASON-BAHR (1941). Nicotonic acid in doses of 150 to 300 mgm. daily alleviates the mouth symptoms, and riboflavin in 3 mgm. doses daily relieves the angular stomatitis.

GIARDIASIS

Intestinal infection with this parasite has for its most common symptoms diarrhœa and abdominal pain, but sometimes it is found on routine examination in patients showing no symptoms.

Treatment with mepacrine hydrochloride or praequine in dosage of 0·1 gm. three times a day after food is proving effective in removing the organisms.

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