Staff Meeting Bulletin Hospitals of the » » » University of Minnesota

Pneumonia in Infancy

Volume XV Friday, October 15, 1943 Number 3

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
UNIVERSITY OF MINNESOTA

Volume XV | Friday, October 15, 1943 | Number 3

INDEX

Published for the General Staff Meeting each week during the school year, October to June, inclusive.

Financed by the Citizens Aid Society, Alumni and Friends.

William A. O'Brien, M.D.

I. LAST WEEK

Date: October 8, 1943

Place: Recreation Room
Powell Hall

Time: 12:15 to 1:30 p.m.

Program: Congenital Hyperplasia of the Adrenals
T. E. Bratrud
Willis H. Thompson

Discussion: Leo T. Samuels
Gerald T. Evans
Theodore H. Sweetser

Attendance: 102

Alice Carlson,
Record Librarian

- - -

II. MEETINGS

1. ANATOMY SEMINAR

Saturday, October 16, 1943, 11:30 a.m., Room 226, Institute of Anatomy.

"Experimental Study of the Innvervation of the Sphincter of Oddi in the Cat"
E. A. Boyden
Charles Van Buskirk

- - -

2. CENTER FOR CONTINUATION STUDY
October 18-23, 1943

Continuation Course in Kenny Technique for Infantile Paralysis (for physicians). Medical Sciences Building.

- - -

3. PATHOLOGY SEMINAR

Monday, October 18, 1943, 12:30 p.m., Room 104 Anatomy Building.

"A Clinical Study of a Case of Hypertension"

T. A. Peppard

- - -

III. LECTURES

1. JOHN B. & MARY R. MARKLE FOUNDATION LECTURE

Tuesday, October 19, 1943, 2:00 p.m., Medical Sciences Amphitheatre.

"Tropical Medicine"
Major General James C. Magee

- - -

2. THE MINNESOTA PATHOLOGICAL SOCIETY

Tuesday, October 19, 1943, 8:00 p.m., Medical Sciences Amphitheatre.

"Malaria"
L. T. Coggeshall,
Ann Arbor, Michigan.

- - -

IV. PROMOTIONS (Cont.)

Harold A. Whittaker, Clinical Professor of Preventive Medicine and Public Health.
Wallace D. Armstrong, Professor of Physiological Chemistry
Charles E. Connor, Clinical Associate Professor of Ophthalmology and Oto-Laryngology
Joseph T. Cohen, Clinical Associate Professor of Pediatrics
Ruth B. Freeman, Associate Professor of Preventive Medicine and Public Health
George O. Pierce, Associate Professor of Preventive Medicine and Public Health
Thomas Lowry, Clinical Assistant Professor of Medicine

HEADSHIP CHANGES

Dr. J. C. McKinley, Head of Department of Medicine and Director of Division of Nervous and Mental Diseases has been named Head of the newly established Department of Neuropsychiatry, which includes Divisions of Adult Psychiatry and Child Psychiatry.

Dr. Cecil J. Watson has been named Head of the Department of Medicine and Director of the Division of Internal Medicine.

Dr. Raymond N. Bieter, Professor of Pharmacology has been appointed to the Headship of the Department of Pharmacology to succeed the late Dr. Arthur D. Hirschfelder.

V. SOME PROBLEMS OF PNEUMONIA IN INFANCY

John M. Adams

Introduction

According to Wilburt C. Davison, "prevention is the most important phase of pediatrics. Three fourths of the quarter of a million annual deaths of American children can and should be prevented. Twenty-one per cent of these deaths are due to curable diseases, while 56 per cent are caused by preventable conditions."[1] As an example, the causes of pediatric deaths which can be reduced by better antepartum, intrapartum and neonatal care are prematurity, birth injuries, still births, conditions of early infancy such as atelectasis, asphyxia, suffocation, impetigo and syphilis. These conditions represent 60 per cent of deaths under the age of one year.

What can be done to prevent pneumonia in the infant? To begin to find the answer to this problem we have turned to the pathogenesis of pneumonia and newer knowledge concerning the inception of this disease. Also a study of the anatomy, physiology and pathology of the pulmonary system has revealed some interesting problems pertaining to pneumonia in infancy.

Pathogenesis

We have generally assumed that the infant breathes the germs of pneumonia into its lungs and then may fall victim to the invading organisms. There is, however, a good deal of evidence accumulating to the contrary. Kneeland[2] demonstrated that infants begin to harbor pathogens in their upper air passages at two to three months of age without symptoms. Ho[3] further showed that the pathogens present at the onset of the common cold multiply rapidly in the nose and throat. Smillie[4] in studies concerned with the epidemiology of pneumonia successfully established the fact that infants can harbor type XIV pneumococci in their upper respiratory tract for many weeks without symptoms until attacked by an upper respiratory infection which then renders the infant host susceptible to the harbored organisms, and serious disease may result. These studies indicate that some additional factors other than the presence of pneumococci must operate in producing pneumonia.

Experimental studies by Robertson[5] on dogs lists certain conditions as "essential for the production of the pneumonia lesion: first, the implantation of the pneumococci in the terminal airways; second, a fluid but viscous medium which prevents their rapid expulsion from this region of the lung; and third, the presence of local irritation." Local irritation appears more significant than obstruction in determining whether or not infection occurs.

The inhalation of bacteria into the pulmonary system is probably not the usual method of contracting pneumonia, particularly if the host is in good health. It is commonly recognized that doctors and nurses seldom develop contact infection from cases of pneumonia. Robertson[5] repeated work of Stillmans[6] in which animals were sprayed with very find droplets of virulent pneumococcus cultures, and he was unable to infect mice by this method. Hanburger and Robertson recovered pneumonococci from the peripheral lung tissues of dogs within five minutes after subjecting them to an intrabronchial spray of pneumococcus culture. They showed that the principal mechanisms for expulsion of foreign matter was the ciliary action which is capable of moving material as much as 1 cm. per minute in the bronchi and 3 cm. per minute in the trachea. Further mechanisms of removal are cough, and possible peristaltic movements of the bronchi. In the alveolar spaces, the large phagocytes are largely responsible for removal of foreign material. Robertson states that these mechanisms are so effective that the lungs of normal human beings are for the most part kept sterile.

The most likely manner in which bacteria reach the air spaces is by way of infected fluid exudate which passes the epiglotic barrier and flows or is aspirated into the lung. The epiglotic

barrier has been shown to be incomplete in preventing fluid material from getting into the lung. Chilling was found to cause incomplete closure of the epiglotis in animals.[7] Walsh and Cannon[8] demonstrated that oil in the nasal passages quickly found its way to the depths of the lung and if irritating in character produced a definite lesion. The infant's tendency to aspirate infected material from the upper air passages plays a large role in the inception of pneumonia at this age.

Air Borne Infection

The pathogenesis of pneumonia must necessarily include a brief discussion of aerial transmission of infection. There is no doubt that bacteria are disseminated and spread by air currents. That droplets expelled from the respiratory tract evaporate and ultimately settle in the dust of the room was demonstrated by Wells.[9] Cruickshank[10] demonstrated cross infection in burns in the hospital especially by hemolytic streptococcus. Studies on the pneumococcus, however, have shown little evidence that animals can be infected by the inhalation of pneumococcus - containing droplets.[11]

Virus-containing atmospheres on the other hand, particularly influenza, have been demonstrated to be highly infective for mice. Robertson[11] states that air-borne infection of both bacteria and virus origin depends on the concentration of the infectious agent in the atmosphere. Experimental studies of Henle, Sommer, and Stokes[12] demonstrated that heavy concentrations of air-borne streptococci were necessary to kill mice; and with low concentrations all mice survived, but that a carrier state had been induced in animals exposed under control conditions, and not in those protected by ultra violet light barriers.

Many methods for the control of possible air-borne infections are the subject of a great deal of investigation at present, and entail a long discussion in themselves. Filtration has been shown to be very effective but still too complicated and impractical for use at present. Ultra violet light and chemical sterilization are still in the stage of experimental study. Isolation, adequate masking, and dust control are probably the best practical methods available in most of our hospitals. Loosli[13] and associates have recently reported on the influence of humidity on the survival of the influenza virus in air. They showed that the infectivity rate for mice was much higher in a room of low relative humidity (23 per cent) than in a room of high relative humidity (89 per cent).

Anatomy and Physiology

Briefly, a few points concerning the anatomy and physiology of the infant lung warrant emphasis. The air passages and alveolar spaces of the infant lung are relatively larger than in older children and adults, but are absolutely smaller, adding to the problems of obstruction and elimination of infected exudate. The lining of the bronchial tubes is mucous membrane, the epithelium of which is cylindrical and ciliated. This beats toward the mouth and aids expulsion of foreign particles, bacteria and mucous toward the exterior of the body. A study of the smaller bronchioles of infants reveals the absence of cilia in these air passages. In the adult lung cilia are present in cuboidal epithelium in the smaller bronchioles of less than a millimeter in diameter. We have studied bronchioles of the infant's lung of this caliber, and even areas of columnar epithelium and have found no evidence of cilia. This suggests that once bacteria and infectious agents reach these areas, their removal becomes very difficult. In addition, the physiologic mechanisms of elimination such as cough, ciliary and muscular actions are not fully developed.

A great deal of emphasis has been placed on the fact that the right bronchus leaves the trachea at less of an angle than the left, thus accounting for a higher incidence of foreign bodies and descending infections on the right side. An equally important relationship lies in the fact that the trachea and bronchi have a decided backward slope from the

oral and pharyngeal passages. This angle is 20 to 25 degrees away from the ventral surface of the body. When the infant lies on its back fluid exudate is able to gravitate into the lung with relative ease. Conversely when lying on its abdomen and chest, it is quite impossible for fluid to flow into the lung, and any exudate present in the air passages tends to run out through the nose and mouth.

We have attempted to take advantage of this anatomical fact to prevent infected fluid exudate from reaching the lower respiratory passages by placing infants on their abdomens, and also by raising the foot of the crib. The position compliments the immature development of the mechanisms of elimination and aids the infant in getting rid of aspirated material not only directly following birth, but during the period of early infancy when the incidence of aspiration pneumonia is very high. In the presence of upper respiratory infection, it seems possible that a good deal can be accomplished toward the prevention of pneumonia by position alone. Infants tolerate this position very well and seem to prefer it. Many mothers tell me their babies will not sleep any other way. The danger of choking on obstructing mucus is diminished as is the need for expelling this material by coughing. It is often an effective way of treating this annoying symptom. The mattress under the infant should be firm and flat, allowing free movement of the head to one side or the other.

In our newborn nursery at present, we are keeping half of the babies on their abdomens. There seems to be much less trouble with choking on mucous in these infants. I would like briefly to review the following case which we have observed recently:

Baby Boy H. was born on July 18, 1943 normally. The mother had pre-eclamptic toxemia. There was some difficulty in resuscitation at birth. The baby weighed 3750 gms. at birth (8 1/4 lbs.).

During the first night one cyanotic spell and considerable mucus was noted. Color was again good until 2 p.m. the next afternoon when cyanosis and mucus was again observed. Oxygen was administered and the color returned. Attacks of cyanosis continued associated with feedings. Examination revealed no dullness but some decrease in breath sounds. Heart sounds were normal. Respiratory difficulty continued until the fourth day of life when the infant was placed on the abdomen. The temperature at all times remained normal.

A roentgenogram of the chest taken on the second day of life showed soft mottling especially from the right hilum towards the periphery. Films taken one week later revealed considerable clearing of the process.

Diagnosis: Aspiration Pneumonia.

I think this case illustrates the value of postural drainage in the treatment of pneumonia, but I believe it also emphasizes its possible importance in prevention. The asphyxia present at birth undoubtedly played a role in the development of pneumonia. MacGregor[14] in a study of pneumonia in the newborn emphasizes one fact, the outstanding importance of asphyxia. She states: "It is asphyxia that causes excessive aspiration of amniotic sac contents or vaginal secretion and therefore underlies nearly all cases of true congenital pneumonia. It is asphyxia that produces the pulmonary congestion and edema.....It is asphyxia that causes the depression of the respiratory centre to which persistent atelectasis with all its attendant dangers is most often due."

The most frequent predisposing and precipitating infections of pneumonia are the common cold, influenza, measles and whooping cough. These infections are responsible for local irritation and congestion which appear to be essential factors in the pathogenesis of most of the pneumonias of early life.

The pathogenic factor of inherited or neonatal immunity has been shown to be important experimentally and clinically. Woolpert, Dettwiler, and co-workers[15,16] were able to infect the lungs of embryo guinea pigs with the influenza virus more readily than the full term offspring. In a previous study we referred to the increased susceptibility of the prematurely

born human infant to the virus of primary virus pneumonitis with an 85% mortality among these infants as compared with an 8% fatality of full term babies.[17] In a study of interstitial pneumonia, Giesenbauer[18] reported post mortem observations in 46 cases, 33 of which were prenaturely born.

In chronic cystic fibrosis of the pancreas, death often results from secondary pneumonia or bronchiectasis. Careful study by Anderson[19] has shown a relatively high incidence, 23% of severe vitamin A deficiency in these infants. Pathologically there is a metaplasia of the epithelial linings of the pulmonary system and other organs associated with xerophthalmia. It is possible that these lung changes prepare the ground for the invasion of secondary pyogenic organisms. Blackfan and Wolbach[20] state that "the early effect of the deficiency (Vitamin A) upon the respiratory mucosa is a satisfactory explanation of the frequency, severity, and persistence of the pneumonias that have been in most instances responsible for death."

There is evidence accumulating in our literature which strongly suggests that many infectious diseases which we would like to consider as having a single etiological agent as their cause are in reality the result of two or more organisms. This seems particularly evident in pneumonia and has quite conclusively been demonstrated to be the case in swine influenza. Shope[30] has shown that this disease is caused by the "bacterium Hemophilus influenzae suis and swine influenza virus acting in concert." The H. influenzae suis is not pathogenic for swine when administered in pure culture by way of the respiratory tract, and the virus component causes a transient mild illness, clinically quite distinct from swine influenza. When administered together, however, a clinically severe illness identical with swine influenza in the field is produced.

McCordock and Muckenfuss[31] produced in animals the complete picture of interstitial bronchopneumonia as seen in man by injecting vaccine virus with a suspension of various types of bacteria. McCordock suggests that interstitial bronchopneumonia results from the combined action of a virus and bacteria. Their further studies on the pathology of whooping cough showed the presence of intranuclear inclusions in 45% of the cases studied at autopsy and in only 2 cases out of 90 control autopsies. Goodpasture[27] and his co-workers also demonstrated intranuclear inclusions in cases of pneumonia following measles and whooping cough.

Ordinary primary pneumococcus pneumonia does not usually affect the framework of the lung while interstitial pneumonia involves the bronchial walls as well as the aveolar and interlobular septa. Most cases of interstitial pneumonia occur as complications of measles, pertussis and epidemic influenza according to the latest edition of Holt's "Diseases of Infancy and Childhood." Two of these 3 diseases are known to be caused by viruses, and pathologic evidence exists which would indicate that pertussis may be caused by a virus as well as the Bordet-Gengou bacillus. Rather than considering the secondary organisms as complicating factors, I would like to suggest that they take an active part in producing the disease picture and the resulting pathologic changes.

Smillie[4] as mentioned previously, has shown that infants can harbor pathogenic organisms (type 14 pneumococcus) for some time without evidence of disease until some additional infection (upper respiratory) makes its appearance causing the original organism to produce a severe disease. This appears to be the concerted action of two or more agents producing a disease picture or syndrome which probably would not have occurred had the host been subjected to only one of the agents.

Professor W. G. MacCallum of the Johns Hopkins University writing in Science (June 28, 1940) made the following statement, "In spite of the general understanding that scarlet fever is caused by a streptococcus, it is my belief that in this we have a typical virus infection with the characteristic second-

ary invasion of these bacteria. Of course, the immunological reactions which are regarded as distinctive of scarlet fever are readily explained as the effect of the secondary infection."

Thrush Pneumonia:

We have recently observed an epidemic of what we have chosen to call "Thrush pneumonia." It is extremely rare for the monilia albicans to produce pneumonia and many very large series of cases of thrush are reported with no deaths, directly attributable to pneumonia. Ludlam and Henderson[34] recently reported on 163 cases of thrush, 20 of whom died of various causes. Ebbs[35] reported a series of 22 cases of fatal thrush esophagitis. Little emphasis is accorded to pneumonia except as a terminal event.

Out of 8 cases of thrush in a maternity home, 5 were admitted to the University Hospitals with outspoken signs of respiratory distress and a diagnosis of pneumonia was confirmed in all cases by x-ray examination. Three additional cases of thrush occurred at the University Hospitals following the admission of these previous cases. Two of these cases showed the same characteristics of abundant mucus, cough, dyspnea and cyanosis. All cases had the typical white patches on the tongue, cheeks and palate. The large amounts of mucus present in all cases was an unusual feature of thrush, most authors reporting cases call attention to the dry character of the mucus membranes of the mouth and throat. Dyspnea was present in 7 of the 8 cases studied at the University Hospitals, and cyanosis was recorded in 6 patients. Many of the cases had an elevated white blood count, the average of each patient's highest count was 16,275 cells per cubic millimeter. Temperature elevations were recorded in all except one case at the University Hospitals. Spikes of temperature to 101° and 102°F. were characteristic, while in 2 cases the temperature rose to 104°F. on 1 occasion in each patient. The x-ray findings in the lungs showed a diffuse type of central infiltration in 7 cases. The other case was not examined by x-ray.

Bacteriological studies were made by laboratory of the State Board of Health on the cases from the Maternity Home. The material in each instance was taken after treatment had been started and no monilia were found in the cultures. One nurse was found to have monilia albicans in her throat culture on several occasions. Cultures taken later at the University Hospitals on 3 cases early in the disease all showed monilia to be present.

- - - -

Table I

	Thrush	Staphylococcic Pneumonia	Present Cases
Epidemiology	Endemic	Sporadic rarely epidemic	Epidemic
Onset	Insidious	Sudden	Moderate
Oral lesions	All cases	None	All cases
Mucous	None (dry)	Slight	Marked or severe
Cough	None	Slight or marked	Marked
Dyspnea and cyanosis	None	Marked or severe	Marked
Temperature elevation	None	105° to 106° F.	101° to 102° F.
Toxicity	None	Severe	Moderate
White blood cells	Normal	High as a rule	Moderate average 16,275
Pneumopyothorax	None	4 out of 6 in one epidemic	None
Empyema	None	All cases as a rule	None
Mortality	Slight	Very high	1 out of 7 cases

Of the eight cases first observed at the Maternity Home, 6 had evidence of impetigo about the time of the onset of the thrush lesions in the mouth. One of the cases from the Maternity Home died and autopsy revealed a widespread pneumonia with multiple small areas of abscess formation. A staphlococcus was the apparent cause of the fatal pneumonia. For lack of further evidence, it is merely suggested that this unusual clinical picture of pneumonitis associated directly with the oral thrush in all cases was caused by a staphylococccus and monilia albicans acting together to produce the syndrome of epidemic thrush pneumonia. See table I for further details.

Sudden Death due to Pneumonia:

An additional perplexing problem to us in pediatrics is the explanation of sudden death in infancy. By this we refer to the child who has previously been considered to be quite well and who is unexpectedly found dead in the crib. Little mention is made in the literature and our standard text books of pneumonia as a cause of sudden death. We have all seen frequent reference to these distressing deaths in our newspapers and recently we have had 4 such deaths in newborn nurseries in Minneapolis. I have had the opportunity to study autopsy material from these cases and in every instance the lung findings presented striking evidence for the fatality. The most characteristic changes are those of widespread congestion with diffuse hemorrhage and scattered areas of mononuclear infiltration. Grossly the lungs appear heavy with lumpy areas of consolidation. The plueral surfaces appeared red and on cut section the surface was dark red with a large amount of bloody frothy mucus extending from the bronchi.

McCordock and Muckenfuss[31] and Sprunt et al [32] have produced a similar picture in animals by the use of vaccinia virus. In discussing the nature of virus pathology, Rivers[29] says, "the fact that inflammation occurs in many virus diseases cannot be denied, and despite the acute nature of some of the diseases, if secondary infections do not intervene, the inflammatory process is usually characterized by an infiltration of mononuclear cells." Sprunts[32] states that "in virus disease the mononuclear reaction occurs in the acute phase of the disease....." I have recently reported 3 cases of sudden death[33] which were associated with an epidemic of virus pneumonitis in infants. The most outstanding and constant histopathologic change in these infants' lungs was the interstitial mononuclear response and this was likewise the predominant cellular reaction found in the cases with primary virus pneumonitis, most of whom died on the 6th and 7th day of illness. All of the latter patients showed, in addition, proliferation and desquamation of bronchial and bronchiolar epithelium and also cytoplasmic inclusion bodies in these cells.

The almost complete lack of bacteria and polymorphonuclear leucocyte response in these cases of sudden death suggests a virus etiology. Additional evidence in favor of this assumption is afforded by the fact that the marked mononuclear infiltrations demonstrated in the microscopic sections were similar to those observed in experimentally produced as well as spontaneously occurring, virus pneumonia.

Studies are in progress now in which we are attempting to isolate a virus from some of the recently observed cases of sudden death.

Classification

A pathogenic and etiologic classification of the pneumonias of infancy follows:

I. ASPIRATION PNEUMONIA
   1. Congenital Pneumonia
   2. Lipoid Pneumonia
   3. Thrush Pneumonia

II. TUBERCULOSIS
   (First infection type of pneumonia)

III. EOSINOPHILIC PNEUMONIA
   (Loeffler's Syndrome)

IV. NON-SPECIFIC INTERSTITIAL PNEUMONIA
   (Pertussis, Measles, Influenza, Atypical Pneumonia)

V. PRIMARY VIRUS PNEUMONITIS

VI. SECONDARY VIRUS PNEUMONIA (Goodpasture)

VII. PRIMARY PYOGENIC PNEUMONIA

VIII. SECONDARY PYOGENIC PNEUMONIA

IX. SYPHILITIC PNEUMONIA (Pneumonia Alba)

Pathology

The predominating pathologic change in most of the pneumonias in early infancy is an interstitial mononuclear reaction. Sprunt[22] points out that an interstitial mononuclear pneumonia is only one phase of the lung reaction to almost all causes of pulmonary disease. The only partial exception is in the primary and secondary pyogenic pneumonias. These pneumonias on occasion will produce a predominantly mononuclear change.[22]

Therefore, in order to compare and differentiate these various forms of pneumonia, the specific histopathologic differences will be considered in relation to the etiology and development of each entity. Fat-laden macrophages and foreign body giant cells set apart the pneumonias resulting from aspiration of oils.[23] In tuberculosis, the epitheloid cell and typical giant cell characterize the entity. Widespread pulmonary infiltration of the eosinophilic cell, coinciding with the high blood eosinophilia is assumed for Leoffler's pneumonia, since there has been no opportunity to study autopsy specimens. The blood eosinophiles are larger than normal, with unusually large granules, which are fewer in number [24,25].

The thickening of the various constituents of the pulmonary system seen in interstitial pneumonia is distinctive. Bronchiolitis and peribronchiolitis, thickening of interlobular and alveolar septa and infiltrations of lymphocytes and plasma cells are conspicuous. Geisenbauer[18] and Roulet[26] have recently described the detailed pathologic changes in a large series of infants. Geisenbauer[18] states that the exudate presents a honeycomb appearance, resembles fibrin, but does not take the same stains. The exfoliated alveolar cells frequently contain fatty and lipoid granular inclusions

In primary virus pneumonitis of infants, we have had a singular opportunity to study the pathologic changes most probably produced by a virus in human lung tissues. Necrosis ulceration and proliferation of bronchial epithelium are conspicuous changes. The exudate is predominantly epithelial and mononuclear, no bacteria and few polymorphonuclear leucocytes are evident. A mononuclear peribronchiolar infiltration adds to the microscopic picture. The specific distinguishing feature in these cases is the presence of characteristic cytoplasmic inclusion bodies in the epithelial cells of the bronchial, bronchiolar and alveolar tissues. These bodies have definite features, varying in size from three to six microns, stain acidophilic with the hematoxylin and eosin stain, are frequently surrounded by a clear zone or halo and sometimes have vacuoles within the substance of the inclusion.

Secondary virus pneumonia was first described by Goodpasture and his coworkers[27] in 1939 as a virus infection of the lungs following measles, and in one instance whooping cough. The unusual pathologic features are the presence of hemorrhage in the lung, isolated or situated about areas of definite inflammatory consolidation, a stringy mucoid exudate, ulcerated areas in the trachea and scattered areas of necrosis in the mucous glands. "The specific feature of the process was the presence of intranuclear inclusions, which were almost entirely restricted to epithelial cells." "These involved cells rapidly underwent necrosis and this was the essential cause of the extensive ulceration."[27]

The pathologic-anatomic changes in the lungs caused by the pyogenic organisms are well known. MacCallum[28] in 1918 and 1919 was able to differentiate the pneumonias by the distinctive pathological anatomy produced by the various pyogenic organisms. This led to the conclusion

by him that epidemic influenza was probably due to a virus and not due to bacterial agents which acted as secondary and tertiary invaders in a host weakened by coincident disease.

In sypilitic pneumonia the lungs are pale and specifically demonstrate extensive hyperplasia of the fibrous tissues of the interlobular and interalveolar tissues. Treponema pallidum are found in the large mononuclear cells.

Summary and Conclusions

1. Pathogenic studies indicate that additional factors other than the presence of pneumococci must operate in producing pneumonia.

2. Important factors appear to be obstruction to normal mechanisms of elimination and local irritation.

3. Aspiration of infected fluid exudate into the lung plays a large role in the inception of pneumonia.

4. The immature anatomy and physiology of the infant lung also are significant factors in the pathogenesis of pneumonia at this age.

5. Postural drainage accomplished in part by placing the infant on the abdomen probably plays an important part in the prevention of infected exudate from reaching the lower pulmonary areas and thus serves as a method for preventing pneumonia.

6. Prevention of asphyxia in the bewborn will aid in the prevention of congenital pneumonia.

7. The most frequent predisposing and precipitating infections of pneumonia are the common cold, influenza, measles and whooping cough. Premature birth and vitamin A deficiency as seen in chronic cystic fibrosis of the pancreas are additional primary factors in pathogenesis.

8. Two or more organisms may act together to produce disease; this seems particularly evident in pneumonia.

9. An epidemic of "thrush pneumonia" has been outlined and the differences have been compared with ordinary thrush and staphylococcic pneumonia (table).

10. Pneumonia is probably the most important single cause of sudden death in infants previously thought to be well.

11. The pathologic changes consist primarily of diffuse congestion, hemorrhage and mononuclear infiltration.

12. A virus etiology is suspected in these cases by the fact that the marked mononuclear infiltrations demonstrated in the microscopic sections were similar to those observed in experimentally produced as well as spontaneously occurring, virus pneumonia.

References

1. Davison, W. C.
Preventive Pediatrics
J.A.M.A. 114, 9:742 (Mar.) '40.

2. Kneeland, Y., Jn. and Dawes, C. F.
Studies on common cold; the relationship of pathogenic bacteria to upper respiratory disease in infants
J.Exper.Med. 55:135, '32.

3. Kneeland, Y, Jn.
Studies on the common cold; upper respiratory flora of infants
J.Exper.Med., 51:617, '30.

4. Smillie, W. G.
The Epidemiology of Pneumonia.
Tr.A.Am.Phys., 56:128, '41.

5. Robertson, O. H.
Newer Knowledge concerning the Inception of Pneumonia and its Bearing on Prevention.
Ann. of Int.Med. 18:1, (Jan.), '43.

7. Nungester, W. J. and Klepser, R. G.
Possible Mechanism of Lowered Resistance to Pneumonia.
Fr. Infect. Dis., 63:94, '38.

8. Walsh, T. E. and Cannon, P. R.
Problem of Intranasal Medication.
Ann. Otol.,Rhin.,& Laryng.,47:579,'38.

9. Wells, W. F.
Am.Jr. Hygiene, 20:611, '34.

10. Cruickshank, R.
Jr. Path. and Bact., 41:367, '35.

11. Robertson, O. H.
Air-Borne Infection.
Science, 97:495, (June) '43.

12. Henle, W., Sommer, H.E. and Stokes, J. Jr.
Studies on Air-Borne Infection in a Hospital Ward.
Jr. of Ped., 21:577, (Nov.) '42.

13. Loosli, C. G., Lemon, H.M., Robertson, O.H. and Appel, E.
Experimental Air-Borne Influenza Infection.
Proc.Soc.Exp.Biol.& Med.,53:205,'43.

14. MacGregor, A. R.
Pneumonia in the Newborn.
Arch. of Dis.Child.,14:323, '39.

15. Woolpert, O. C., Gallagher, F. W., Rubinstein, L. and Hudson, N. P.
Propagation of the Virus of Human Influenza in the Guinea Pig Fetus.
J. of Exp. Med. 68:3,313, '38.

16. Dettwiler, H. A., Judson, N. P., Woolpert, O. C.
The Comparative Susceptibility of Fetal and Postnatal Guinea Pigs to the Virus of Epidemic Influenza.
J. of Exp. Med., 72:6, 623, '40.

17. Adams, J. M., Green, R. G., Evans, C.A. and Beach, N.
Primary Virus Pneumonitis.
J. of Ped., 20:4, 405 (Apr.) '42.

18. Giesenbauer, Wilhelm.
Uber die Sogenannts Interstitielle (plasma cellulare) Pneumonie Fruhegeborener and Schwachlicher Kindex.
Monatsschrift fur Kinderheilkunde, 8:1-138 (Mar.) '31.

19. Anderson, D. H.
Cystic Fibrosis of the Pancreas and its Relation to Celiac Disease.
Am.J.Dis.Che.56:344, '38.

20. Blackfan, K. D. and Wolbach, S. B.
Vitamin A Deficiency in Infants.
Jr. of Ped. 3:679, (Nov.) '33.

21. Johnson, W. C. and Meyer, J. R.
A Study of Pneumonia in the Stillborn and Newborn.
Am.Jr. of Obs. & Gyn. IX, 2,3,(Feb.) '25.

22. Sprunt, D. H.
The Significance of Interstitial Mononuclear Pneumonia.
Southern Med.Jr. 31:4, '38.

23. Ikeda, Kano
Oil Aspiration Pneumonia (Lipoid Pneumonia).
Am.J.Dis.Ch.,49:985, '35.

24. Freund, R., Samuelson, S.
Transitory Infiltration of the Lung with Eosinophilia.
Arch.of Int.Med.,66:1215, '40.

25. Botsztejn, Von Anna
Diepertussoide, eosinophile pneumonie des saugling
Ann.Paedit. 157:28, '41.

26. Roulet, F.
Ueber die interstitielle plasmozellulare, Pneumonie im sauglingsalter.
Schweiz. Med. Schnschr. 71:1313, (Oct. 25), '41.

27. Goodpasture, E. W., Auerbach, S. H., Swanson, H. S., Cotter, E. F.
Virus Pneumonia of Infants Secondary to Epidemic Infections.
Am.Jr.Dis.Cis.Child., 57:5,997,'39.

28. MacCallum, W. G.
Pathological Anatomy of Pneumonia Associated with Influenxa.
Johns Hopkins Hospital Rep. 20,149,'21.

29. Rivers, T. M.
Some General Aspects of Pathological Conditions caused by Filterable Viruses.
Am.J.of Path. 4:2, 91, '28.

30. Shope, R. E.
Swine Influenza, Virus Diseases.
Cornell University Press, Ithaca, N.Y., '43.

31. McCordock, H. A. and Muckenfuss, R. S.
The Similarity of Virus Pneumonia in Animals to Epidemic Influenza and Interstitial Bronchopneumonia in Man.
Am.Jr.Path., IX:221-252, '33.

32. Sprunt, D. H., Martin, D.S., and Williams, J. E.
Interstitial Bronchopneumonia.
Jr. Exp.Med.:62:73, '35 and 62:449, '35.

33. Adams, J. M.
Sudden Death in Infants due to Pneumonia.
Jr. of Ped. 23:189 (Aug.) '43.

34. Ludlam, G. B. and Henderson, J. L.
Neonatal Thrush in a Maternity Hospital.
Lancet 1:64 (Jan.17), '42.

35. Ebbs, J. H.
Oesophagitis in infancy.
Arch. Dis. Child., 13:211 (Sept.) '3

36. Smith, C. M.
Staphylococcal Pneumonia among Infants in a Maternity Hospital.
Lancet 1:1204, '35.

37. MacGregor, A. R.
Staphylococcal Pneumonia.
Arch.Dis.Child. 11:195, '36.

38. Clemons, H.H. and Weens, H.S.
Staphylococcic Pneumonia in Infants.
Jr. of Ped., 20:281, '42.

- - -

VI. GOSSIP

O. P. Jones and his good wife Cathryn Knights (M.D.,'35) wish to be remembered to their Minnesota friends. Good fortune has smiled on them for O.P. has just been made Head of the Department of Anatomy at the University of Buffalo, School of Medicine, replacing Donald Duncan, former Minnesotan who has been made Professor of Anatomy at Louisiana State University, School of Medicine. Katie, not to be outdone by O.P. in grabbing all the honors, will shortly present to her good husband and family a new addition. O.P.'s exhibit of their technique of holding clinical hematologic conferences was shown at the recent meeting of the American Hospital Association in Buffalo. It uses the principle of giving the case history and slides for study and diagnosis followed by discussion....A letter from Charles G. Hayden, U.S.P.H.S., assistant regional medical officer for the third civilian defense region, expresses his perennial twinge of nostalgia for the midwest at this time of the year. Charlie is a football fan from way back. While a student here he went out for the squad just to get the feel of it. His family is well and now includes Bill, Jim and Dick. His wife, Clarise is fit as a fiddle only Charlie is showing signs of wear and tear. He will long be remembered as the editor of our Interns and Resident's Procedure Book. It was an A-1 job....Paul C. Swenson, Minnesota graduate (Duluth) has been appointed Professor of Roentgenology at Jefferson Medical College of Philadelphia. He received his training in New York where he was Associate Professor of Radiology at Columbia University College of Physicians and Surgeons in New York....M. H. "Doc" Manson, Worthington, who received his M.D. here in 1929 has been appointed Medical Director of American Telephone and Telegraph Company, replacing Dr. Cassius H. Watson, retired. Dr. Manson served an internship at the University of Wisconsin and completed his graduate training in surgery in Minnesota. He received M.S. in '32, Ph.D. in '34 following which he was appointed an instructor. He left Minnesota in '37 to conduct hospital surveys for the American College of Surgeons. This was followed by a position with the Commonwealth Fund in development of their rural hospital program. He left there to become medical director of the Bell Telephone Company, New York (research unit) where he has been located up to his recent appointment. While at the university he developed a special interest in bacteriology as it relates to surgery. He was a skillful technical surgeon and had excellent clinical judgment. He was outstanding as a teacher and became one of the best known members of our staff. His great capacity for making friends coupled with extraordinary ability will make him an outstanding leader in the field of industrial medicine and surgery. Few promotions which will bring more real joy to a man's friends than this one...Professor Emeritus Jennings C. Litzenberg will play a prominent role in the Meeting of the Omaha Mid-West Clinical Society, Oct. 25-29, 1943. Other guest stars include Eben J. Carey, Tom D. Spies, Sara M. Jordan, Sanford R. Gifford, Rexford L. Diveley, Frank R. Ober, Harold G. Wolff, L. Emmett Holt, Jr., Edward H. Skinner, Raymond W. McNealy, Robert L. Sanders, Cyrus E. Burford, and a group of medical representatives from the Navy, headed by Captain H. L. Dollard of Great Lakes.....Recent gifts to the Medical School include: A new annual appropriation was made by State Legislature of $15,000 for special research in field of cancer. A continuation of grant of $10,000 a year was made by the Citizens Aid Society in support of cancer research and program of cancer education. Annual gift of $5,500 by the Citizens Aid Society in support of George Chase Christian Professorship in Cancer Research was continued. Grand of $5,000 from Jane Coffin Childs Memorial Fund for Medical Research has been made for support of work of Dr. John J. Bittner and Dr. Robert G. Green and associates in Departments of Physiology and Bacteriology on nature and mode of action of milk influence in mammary cancer Grand of $3,500 a year, for a 2-year period, has been made by Commonwealth Fund of New York toward support of Psychiatric Clinic for Children. Toward support of this Clinic the Stevens Avenue Home of Minneapolis will continue their grant of $10,000 for the year beginning July 1,1943 The Medical School has accepted a grant of $3,000 from Parke, Davis and Company, to establish a Fellowship in Clinical Hematology in the Department of Anatomy, under the supervision of Dr. Hal Downey.