

27. Lancet 1:137 (Oct. 26), 1823.
28. Lister, J. On a New Method of Treating Compound Fracture, Abscess, etc.: With Observation on the Conditions of Suppuration. Lancet 1:364-373 (May), 1867.
29. Lowbury, E. J. L. Cross-Infection of Wounds with Antibiotic-Resistant Organisms. Brit. M. J. 1:985-990 (Apr. 23), 1955.
30. Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death, Vol. I, Sixth Revision, Adepted 1948. Geneva, Switzerland: World Health Organization, 1948.
31. McDermott, W. The Problem of Staphylococcal Infection. Brit. M. J. 2:837-840 (Oct. 13), 1956.
32. McLetchie, N. C. E. Staphylococcal Pneumonia in Childhood. Canad. M. A. J. 60:352-356 (Apr.), 1949.
33. Meloney, F. Infection in Clean Operative Wounds: A Nine Year Study. Surg. Gynec. & Obst. 60:264-276 (Feb. 15), 1935.
34. Nightingale, F. Notes on Hospitals. Cited by Cowles, E.¹⁵
35. ———. Notes on Nursing: What It Is, and What It is Not. London, 1859. Reproduced by J. B. Lippincott, Philadelphia, 1946.
36. Poole, W. H., and Whittle, C. H. Epidemic Pemphigus of the Newly Born. Lancet 1:1323-1327 (June 8), 1935.
37. Ravenholt, R. T., and LaVeck, C. D. Staphylococcal Disease—An Obstetric, Pediatric, and Community Problem. A.J.P.H. 46:1287-1296 (Oct.), 1956.
38. Ravenholt, R. T.; Wright, P.; and Mulhern, M. E. Epidemiology and Prevention of Nursery-Derived Staphylococcal Disease. New England J. Med. 257: 789-795 (Oct. 25), 1957.
39. Reed, C. B. Impetigo or Pyodermitis Neonatorum. Am. J. Obst. & Gynec. 17:49-58 (Jan.), 1929.
40. Reid, J. The Furunculoid Epidemic. Lancet 1:2-3 (July 7), 1855.
41. Schimmelbusch, C. The Aseptic Treatment of Wounds, Berlin, 1893. Cited by Walter, C. W.⁴⁶
42. Semmelweis, I. The Etiology, the Concept and the Prophylaxis of Childbed Fever. Reprinted in Medical Classics 5:350-773, 1940-1941. Baltimore, Md.: Williams & Wilkins, 1941.
43. Simpson, J. Y. Our Existing System of Hospitalism and its Effects: Part I. Edinburgh M. J. 14:816-830 (Mar.), 1869.
44. Starkey, J. Control of Staphylococcal Infections in Hospitals. Canad. M. A. J. 75:371-380 (Sept. 1), 1956.
45. Tenon, J. R. "Memoirs," 1788. Cited by Garrison, F. H. History of Medicine (2nd ed.). Philadelphia, Pa.: Saunders, 1917, p. 406.
46. Walter, C. W. Aseptic Treatment of Wounds. New York: Macmillan, 1948, p. 372.
47. Wells, W. F. Airborne Contagion and Air Hygiene. Cambridge, Mass.: Harvard University Press, 1955.
48. What Is the Use of a Hospital, Editorial. The London Times, June 28, 1877. Reprinted in the Edinburgh M. J. 23:181-183, 1877.
49. Wysham, D. N.; Mulhern, M. E.; Navarre, J. C.; LaVeck, G. D.; Kennan, A. L.; and Giedt, W. R. Staphylococcal Infections in an Obstetrical Unit I. Epidemiologic Studies of Pyoderma Neonatorum. II. Epidemiologic Studies of Puerperal Mastitis. New England J. Med. 257:295-306 (Aug. 15), 1957.
50. Wysham, D. N., and Kirby, W. M. M. Micrococccic (Staphylococccic) Infections in a General Hospital. J.A.M.A. 164:1733-1739 (Aug. 17), 1957.

Dr. Reimert T. Ravenholt is director, Division of Epidemiology and Communicable Disease Control, Seattle-King County Department of Public Health, Seattle, Wash.; and Otto H. Ravenholt is a senior medical student, University of Minnesota, Minneapolis, Minn.

This paper was presented before a Joint Session of the Engineering and Sanitation, Epidemiology, and Maternal and Child Health Sections of the American Public Health Association at the Eighty-Fifth Annual Meeting in Cleveland, Ohio, November 14, 1957.

Presented with the sponsorship of the Committee on Air Hygiene of the Engineering and Sanitation Section.

OBSERVATIONS RELATIVE TO THE NATURE AND CONTROL OF EPIDEMIC STAPHYLOCOCCAL DISEASE

Frederick H. Wentworth, M.D., F.A.P.H.A.; Adah L. Miller, R.N.; and Bertina B. Wentworth, B.S.

SINCE MARCH, 1956, our study group has had opportunity to make detailed observations of nine outbreaks of staphylococcal disease among newborn infants and has been able to make limited observations of five other epidemic situations. The epidemiologic

pattern in every instance, has been remarkably similar and strongly resembles that presented by several recent publications in the British, Canadian, Australian, and American literature.¹⁻¹⁰ The outbreaks have consisted of cases of pyoderma among newborn infants

during their hospital stay, invariably associated with an increased incidence of breast abscesses among nursing mothers, as well as various types of suppurative disease among infants and family members following the discharge of the infant from the hospital. Furuncles, carbuncles, and a variety of subcutaneous abscesses have predominated in the family group. The same manifestations, plus mastitis, breast abscesses, and staphylococcal pneumonia, have been observed among the infants.

This report presents selected observations pertinent to three specific areas of concern: (1) the hospital-infected infant as a focus of family infection and the extent to which family members are at risk of infection and suppurative disease; (2) the role of infected nursery personnel in the natural history of nursery staphylococcal infections; and (3) problems of applying methods of prevention and control of nursery outbreaks. The data presented in support of our views on the first two points were derived from the study of three epidemics in two different hospitals. Opinions concerning prevention and control are based on our general understanding of published reports and on our own experiences with the 14 epidemics.

The methods used to study epidemics comprised personal observation of the nursery and nursery technics, interview of the hospital personnel, a review of the pertinent hospital records, and repeated cultural surveys of the infants and nursery personnel. Information concerning the infants following discharge and of the mothers and family members was obtained through a visit to the home, either by a member of the study team or, much more frequently, by a generalized public health nurse on the staff of the local health department. Specimens of suppurative material from lesions and nasopharyngeal swab specimens were examined for the presence of coagulase-positive staphylococci.

Small wire cotton swabs were used to obtain the nasopharyngeal cultures. High salt (7.5 per cent) brain-heart-infusion broth and Mannitol salt agar were used for the primary isolation of staphylococci. Tube coagulase tests and disk antibiotic sensitivity tests were done by standard technics. Coagulase-positive staphylococci were bacteriophage typed with 32 phages originally obtained from the laboratory of Dr. John E. Blair of New York City.

Since the addition of bacteriophage typing and antibiotic sensitivity to the usual methods of identification of staphylococci, it has become increasingly apparent that in epidemic situations one identifiable strain of coagulase-positive *Staphylococcus pyogenes* tends to predominate. It also has become evident that certain strains identifiable by these technics are much more frequently isolated from epidemics than are other strains. Although there are definite limitations to the usefulness of these methods of identification, the concept of the "epidemic strain" has been reasonably well established. The incrimination of a given strain as the one responsible for a given epidemic situation is still difficult, but the isolation of a previously established epidemic strain from cases and contacts in an epidemic situation permits observation of the natural history of the disease and application of control measures previously impossible. Shaffer and his associates¹¹ have given recognition to *Staphylococcus pyogenes* phage type 42B/44A/47C/52/80/81, sensitive to novobiocin, bacitracin, chloramphenicol and erythromycin and resistant to penicillin, streptomycin, and the tetracyclines, as an epidemic strain responsible for a large number of outbreaks in this country and abroad. A strain of staphylococcus indistinguishable by available technics from those accumulated by Shaffer has been isolated from the majority of our epidemics and is the or-

ganism responsible for the outbreaks discussed in this paper.

In most studies of nursery outbreaks the epidemic strain of staphylococcus has been isolated not only from the infants with pyoderma, but also from the nasopharynx and skin of a high percentage of apparently well infants in the nursery and following discharge. To investigate the importance of these subclinical infections we studied an epidemic of

pyoderma in a hospital nursery in which 23 of the 90 infants born during March, 1956, had developed skin lesions while in the hospital. A retrospective study of the situation begun in April produced the data summarized in Table 1a, b, and c. The table shows that when nasopharyngeal swab cultures were obtained from 71 of the 90 infants, 10-35 days following discharge, the 48 infants present in the nursery

Table 1—Study of Infection and Lesion Rates Among Infants and Family Members—Lancaster, Ohio, 1956

Table 1a. Lesion Rates and Nasopharyngeal Infection Rates 10-35 Days After Discharge

Lesion Status in Hospital—March, 1956	No. of Infants *	Infection †		Lesion Following Discharge	
		No.	Per cent	No.	Per cent
Hospital lesion	23	9	39
No hospital lesion	48	17	35	20	41

Table 1b. Lesion Rates in Infants According to Infection Status 10-35 Days Following Discharge

Infection Status †	No. of Infants	History of Lesion 10-35 Days Following Discharge	
		No.	Lesion Rate per 100 Studied
Infected	17	14	82%‡
Not infected	31	6	19%‡

Table 1c. Risk of Infection and/or Lesions in Family Members Following Discharge of Infants During Three to Six Months Observation Period

Member Family	Total Studied	Infected †	Infection Rate per 100 Studied	Lesion Rate per 100 Infected
Mothers	26	18	72%	44%
Fathers	24	7	28%	42%
Siblings	37	11	30%	72%

* A total of 90 infants were born in March, 1956, and 71 of these were investigated by home visit following discharge.

† Recovery of *Staphylococcus aureus* phage type 42B/44A/47C/52/80/81 from nasopharyngeal swab cultures.

‡ The difference is significant at $p < 0.01$.

during the outbreak who had not developed skin lesions while in the hospital had developed nasopharyngeal infections with the epidemic strain at about the same rate as the 23 infants who had lesions while they were in the hospital. That these infections were more than transient, passive carrier states is suggested by Table 1b which reveals that approximately 80 per cent of these infected infants developed some type of skin lesion or suppurative disease following hospital discharge, while only 19 per cent of those not infected had similar experiences. This high lesion rate, which may be predicted for infants infected during the epidemic period, and the over-all lesion rate of 41 per cent for infants discharged without a history of hospital disease emphasize the importance of post-hospital discharge observation during epidemic periods. These findings are very similar to those recently published by Wysham and associates¹⁰ following their very excellent studies in Seattle, Wash.

In our experience, and in the experience of others, an outbreak of staphylococcal pyoderma in newborn nurseries is almost invariably associated with an outbreak of maternal postpartum breast abscesses. Where family follow-up studies have been carried out an association of the nursery outbreak with suppurative lesions among other family members has also been demonstrated. An attempt to quantitate the risk of infection and/or suppurative disease among mothers and family members is presented in Table 1c. While the numbers involved are relatively small and the infection and lesion rates are subject to considerable variability on repeated study, the results nevertheless suggest that infants discharged from a nursery with a nasopharyngeal infection with an epidemic strain of *Staphylococcus* act as foci of infection for family members. Approximately three-fourths of the

mothers and one-third of the fathers and siblings developed nasopharyngeal infections with the epidemic strain during the observation period of three to six months. Overt manifestations of disease appeared in about 40 per cent of the infected mothers and fathers and about 70 per cent of infected siblings. Again it should be emphasized that 17 of the 26 infants associated with these family outbreaks did not have lesions while they were in the hospital.

The persistence of the epidemic strain in the family group is graphically demonstrated in Table 2 where the results obtained during a five-month follow-up family study are presented. These three families were selected from a group of 17 families studied in a similar manner. The epidemic strain was isolated from the nasopharynx of a number of the family members and appeared to be transmitted freely within the family group. Skin lesions and subcutaneous abscesses appeared in a number of the infected individuals during the observation period. These data support the concept that the epidemic strain, once introduced into the family, becomes firmly established and may be responsible for cases of suppurative disease over an extended period. The value of precise identification of individual strains in the study of staphylococcal disease is also demonstrated here as it would be difficult, indeed, to associate a nursery outbreak with a furuncle in a father occurring five months later unless the isolates could be reasonably identified as being identical. These families have not been followed intensively beyond the five-month study period, but we know that the epidemic organism has persisted in some of them for as long as 15 months and a variety of disease manifestations were still occurring among the family members at that time. Parenthetically, it may be noted that we have had an opportunity to obtain cultures from a family whose

Table 2—Persistence and Spread of Family Infections

Family No.	Family Members	Age or Birthdate	Dates of Culture				
			4/4	4/28	6/22	8/27	9/11
1	Baby	3/5/56	+L†	—	—	—	—
	Mother	30	—L	+‡	—	—	—
	Father	29	—	—	—	—	—
	A *	9	—	+	—	—	—L
	B	5	—	+L	+	—	—L
	C	4	—	+	—	—	—
	D	2	—	—	+L	+	+
	Baby	3/8/56	+L	+	—	+L	—
2	Mother	37	+BA**	+	—	+	+
	Father	41	—	—	—	—	—
	A	16	—	—	—	+	+
	B	9	—	—	—	+	+
	C	7	—	—	+	—	—
	D	5	—	—	+	—	—
	Baby	3/8/56	+L	—	+	—L	+
5	Mother	22	+	+	—	+L	+
	Father	24	—	—	—	+A	—
	A	1½	L	—	—	—	+

* Letters=siblings.

† L=skin lesion.

‡ + = isolation of *S. aureus* 42B/44A/47C/52/80/81 from NP swab and/or lesion.

** BA=breast abscess.

baby was born during the outbreak reported by Shaffer⁹ as having occurred in October, 1954. In July, 1957, more than two and half years after the epidemic, lesions were still occurring within the family and three of five family members were carrying the epidemic strain.

The results of a study of the association of suppurative disease among family members with a nursery outbreak involving much larger numbers are summarized in Table 3. In this study the families of 1,287 infants born during May, June, July, and August, 1956, in a hospital in Cincinnati, Ohio, were visited one to five months (with a mean

of three and a half months) after the birth of the infant. These families represented 66 per cent of the 1957 infants born in this hospital during the four-month period. A serious outbreak of pyoderma occurred in the nursery of the hospital reaching its peak in June and coming under control during August. Investigation revealed that among the 1,287 families studied there were 277 families in which pustular skin lesions or subcutaneous abscesses occurred in one or more family members during the follow-up period. Table 3 compares the family disease rates per 100 families in which the infant had pyoderma or suppurative disease before

or after hospital discharge with the rates in families where there was no history of illness in the infant. In this study 45.1 per cent of 435 families with an ill infant experienced disease in one or more family members during the study period, while 9.5 per cent of 852 families with well infants had a similar experience. In view of the large number of families involved, the difference between 45 per cent and 9.5 per cent is significant with a probability less than 0.001 and strongly suggests an association between the illness of the family members and the illness of the infant.

Another expression of this association may be seen in Figure 1 which shows the similarity of the epidemic curves of the nursery and family outbreaks plotted according to the month of birth of infant. When viewed in the light of the data in Table 3 it seems reasonable to conclude that the control of the disease in the nursery was accompanied by a control of family disease.

The findings presented thus far have been interpreted as almost conclusively incriminating the hospital nursery as one source of community infection with epidemic strains of staphylococci. Com-

munity here refers to families with recent nursery contact, for there is as yet little evidence available concerning the transmission of epidemic strains from infected families into the community at large. The persistence of the epidemic strains within the family for long periods make it likely, however, that transmission to friends and relatives would eventually occur, and perhaps through infected siblings they may be communicated to school populations. Although the work of several authors suggests the hospital as being the primary source of antibiotic resistant strains of staphylococci, other sources of community infection with epidemic strains need thorough exploration. From one of the epidemics studied by our group, we have accumulated evidence incriminating *Staphylococcus pyogenes* phage type 52A/79, a strain sensitive to all antibiotics in current use, including penicillin, as the epidemic strain. In this instance, the pattern of the nursery epidemic was the same as the one previously described, and infection and suppurative disease were demonstrated among mothers and family members. At the same time, as seen by

Table 3—Studies on Staphylococcal Infections Among Family Members Following an Epidemic of Pustular Dermatitis in Hospital S, Cincinnati, Ohio—May–August, 1956

Suppurative Disease in Families of Infants* with and without Disease (Pustular Dermatitis or Suppurative Disease) During or Following Hospitalization

	Total	Families with Ill Members †	Family Disease Rate per 100 Families
Families with ill infants	435	196	45.1‡
Families without ill infants	852	81	9.5‡
Total families investigated	1,287	277	21.5

* Infants born at Hospital S, Cincinnati, Ohio, during May, June, July, and August, 1956.

† Includes mothers and other family members; excludes infants.

‡ The difference is significant at $p < 0.001$.

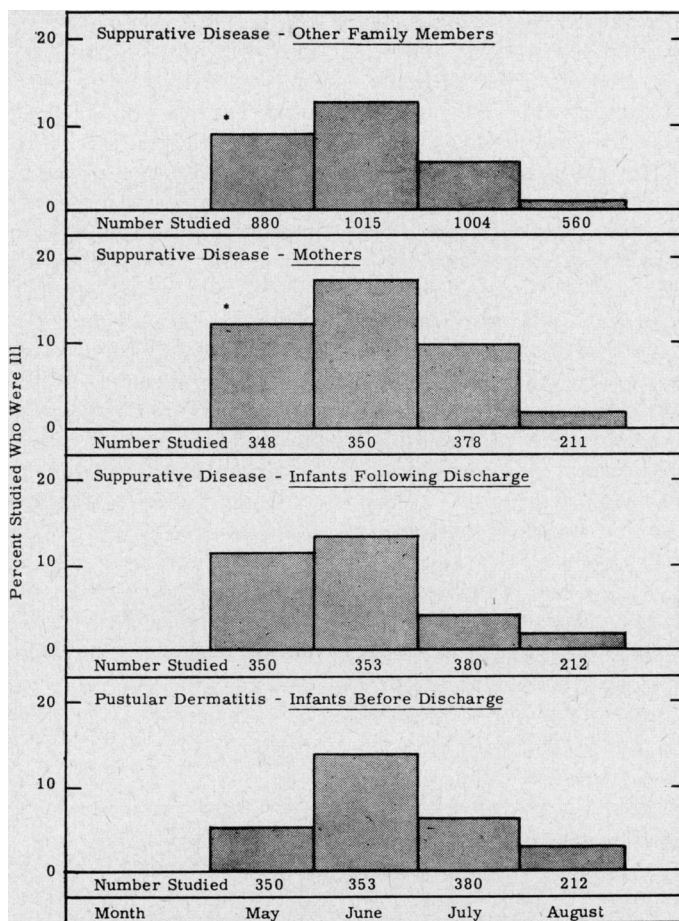


Figure 1—Pustular Dermatitis and Suppurative Disease Among Infants, Mothers, and Other Family Members Following an Epidemic of Pustular Dermatitis in Hospital S., Cincinnati, Ohio, May–August, 1956. By Month of Birth of Infant

the physicians in the community, there seemed to be a general increase of suppurative disease which appeared to be independent of any hospital association. The same strain was isolated from a number of these cases. Whether the hospital can be considered the source of this community outbreak is not clear. We are in no sense attempting to discount the importance of the hospital nursery as a source of community infection, but we hope that this established fact will not prevent the exploration of

other sources of epidemic strains within the larger community.

In addition to establishing the nursery as a source of family infections, the preceding data confirm the findings of other authors concerning the natural history of infection during an epidemic period. Infants exposed to an epidemic strain in a nursery under epidemic conditions are at high risk of developing nasopharyngeal infection. Some of these infants will develop skin lesions during their hospital stay, but even if

they do not the risk of developing them after discharge is still very high. Whether or not they have lesions while in the hospital, infected infants act as foci of infection for the family. These infections tend to spread and persist for long periods within the family group. It is obvious that attention must be given to the infection potential of all infants exposed in the nursery during an epidemic period. Where infected infants being discharged from a nursery are acting as a source of family infection, control of the hospital outbreak may be expected to limit at least that portion of the disease in the community due to contact with the hospital.

In nearly every nursery outbreak, where personnel have been investigated, the epidemic strain has been isolated from the nasopharynx and/or skin of varying percentages of the nursery staff.

The significance of these findings has been differently interpreted by different authors. The high percentage of well infants that are infected; the ease with which the organism has been isolated from the nursery environment, including the nursery air; and the apparent intranursery transmission in the absence of infected personnel has strongly supported the view that the predominate method of spread during the epidemic period is from infant-to-infant or from infant-to-environment-to-infant.

This situation appears to prevail in most of the epidemics we have studied. In a number of instances, however, the epidemic appeared to have been preceded several weeks or months earlier by an occasional case of pyoderma within the nursery or by a sporadic case of suppurative disease among discharged infants or mothers. Similarly,

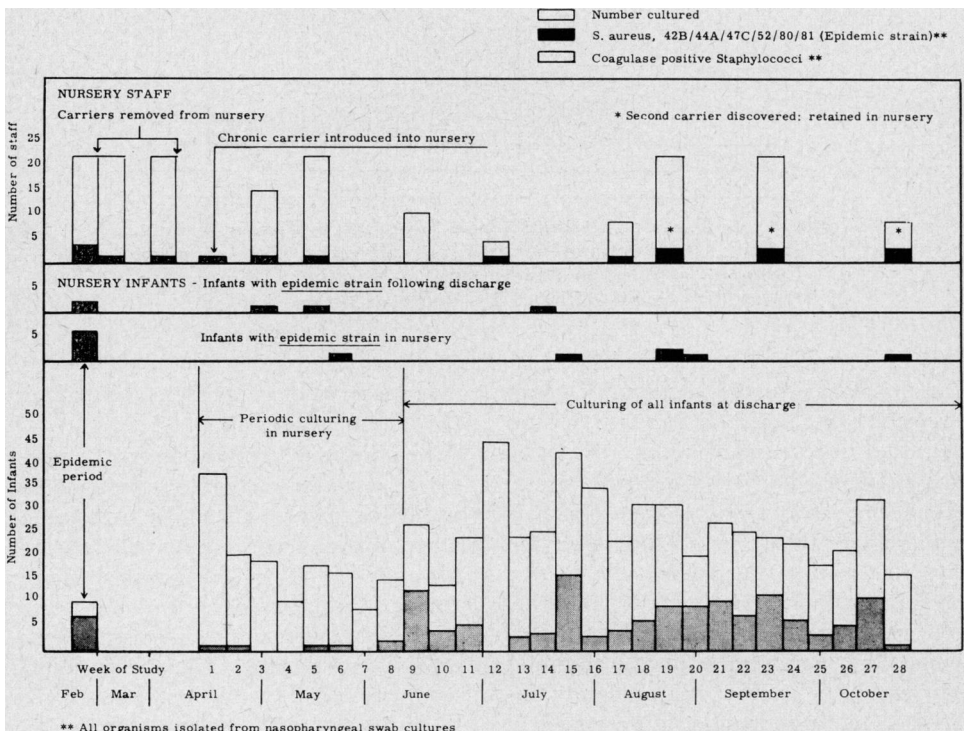


Figure 2—Endemic Staphylococcal Infection in a Newborn Nursery, Ohio, 1957

we have been impressed by the sporadic occurrence of cases during prolonged periods of observation following the apparent control of epidemic situations. Sometimes these cases are followed as long as a year later by a second frank outbreak due to the same epidemic strain. It appeared likely that, while infant-to-infant and infant-to-environment-to-infant transmission were primarily responsible for the epidemics, the infection was being maintained in the hospital by small numbers of chronic carriers and that the sporadic disease was resulting from occasional direct personnel-to-infant transmission.

An opportunity to study this possibility was afforded us and the results are summarized graphically in Figure 2. An outbreak of pyoderma occurred in this nursery in March, 1956, and again in February, 1957. During the February epidemic, noted on the graph, 13 cases of pyoderma among nursery infants occurred and when cultured six of 10 nursery infants were infected with the epidemic strain and four of 22 staff members were similarly infected. In bringing the epidemic under control antibiotic prophylaxis was used on newborn infants and infected personnel were removed from the nursery. Three of the four infected staff members became negative during the ensuing weeks, but one remained persistently infected. In April, 1957, the one remaining carrier was reintroduced into the nursery. The infants were regularly observed and cultures from the nasopharynx of infants and nursery personnel were obtained as indicated in Figure 2. During the last eight weeks of the study all mothers were also cultured at discharge. None of them were carrying the epidemic strain. One round of cultures from 25 sites in the nursery was obtained in April, but this was not repeated and no air samples were taken. The dry-care method of infant care was

used throughout and no antiseptic baths were given at any time.

The completeness of the observations during the ensuing weeks is less than optimal, but it appears to us that the results suggest sporadic infant infection with the epidemic strain at a time when the vast majority of infants were not infected. The lower than expected rate of infection with other strains of coagulase-positive staphylococci during the first few weeks of the study is probably related to our method of periodic culturing. During the latter two-thirds of the study period, however, when cultures were obtained on all infants at discharge, a substantial infection rate with other strains was observed but still only an occasional infection with the epidemic strain was demonstrated. It is important to note that no infants developed recognizable lesions during their hospital stay and that the presence of an epidemic strain in the nursery would have gone undetected had there not been a follow-up of the occasional infants who developed lesions after discharge or a bacteriologic surveillance of the nursery.

Although an epidemic situation has not arisen in this nursery during the 28 weeks of observation, it is likely that an epidemic potential exists nonetheless. The carrier involved in these studies has been examined on several occasions. In each instance we have been unable to demonstrate the epidemic strain in exhaled air, but we have cultured it from the nasopharynx and the skin of her face and hands. If because of an intercurrent upper respiratory infection, such as the common cold or influenza, or, if for any other reason this carrier should begin discharging appreciable numbers of staphylococci into the air for even a short period, or, if through a break in technic she should contaminate some portion of the environment which might serve as a point source for a mass ex-

posure outbreak, the nursery could become saturated with the epidemic strain and the subsequent infant-to-infant and infant-to-environment-to-infant transmission would result in a frank epidemic. It is also likely that infant-to-personnel transmission would occur during the epidemic period resulting in a number of transient carriers among the personnel. As the epidemic proceeded, a mixture of the various modes of transmission would tend to promulgate the infection in a cyclic fashion.

This material has been presented primarily to substantiate our view that the endemic phase of nursery staphylococcal infections may precede or follow the epidemic phase. The epidemiologic pattern in a given situation and rational methods of control will obviously depend upon which of these two phases is predominant. During the endemic period it is likely that no cases of pyoderma will be recognized in the nursery and only an occasional infant or mother will be returned to the hospital with suppurative disease. The time lapse between such patients would prevent their being associated and the total number of cases during any one time period will be below the threshold of anxiety about the situation. Unless a bacteriologic survey of the infants and personnel is made it is likely that an epidemiologic observation of the situation would not be very fruitful. For these reasons it is our opinion that infected nursery personnel play an extremely important role in the natural history of nursery staphylococcal disease and methods of control which are not aimed at discovering carriers and removing them, either through therapy or transfer from the nursery, stand little chance of permanently effecting the course of the disease in the nursery.

Recently, there have appeared in the literature a number of recommended approaches to the control of nursery

outbreaks and for the early detection and prevention of epidemic situations. If accompanied by a search for personnel carriers of the epidemic strain, the time-honored closure of the nursery or the newer application of antibiotic prophylaxis will usually bring the epidemic under control. A variety of methods of increasing intra- and interhospital communication will promote early detection of epidemic or endemic disease in the nursery. A routine follow-up system using forms, telephone surveys, or home visits of discharged mothers can work to the same end. Long-term prevention will depend heavily upon high standards of hospital hygiene and personnel carrier surveillance and perhaps ultimately upon changes in nursery structure and nursery technics.

We have had some experience with most of these methods, but as control procedures are being presented in subsequent papers we will not cover them in detail. However, we wish to make a few comments on this subject. It has been our experience that it is easier to read about recommended control procedures than to put them into practice. We think it fair to state that the practicing epidemiologist usually finds himself giving unrequested consultation to a hospital administrative staff reluctant to admit that a problem exists, a medical staff unaware of the extent or the ramifications of the situation, a frightened nursery staff liberally seeded with feelings of guilt concerning their role in the epidemic and faced with grossly inadequate laboratory facilities for bacteriologic surveillance of the situation. It is not unusual for several weeks to be consumed in gaining the confidence of the hospital personnel, arranging for laboratory facilities in order to accomplish even the simpler identifying procedures, gaining an understanding of what is going on, and arriving at and selling a rational method of control.

Even in the simplest situations where endemic disease exists and one chronic carrier is established among the staff, her removal from the nursery will be resisted if she happens to be the supervisor of the nursery, or in a smaller hospital the only nursery trained member of the staff, until the administrative and nursing staff have accepted the importance of such a move. Even if the carrier can be removed from the nursery, the question of the position in the hospital to which she may be safely transferred is perplexing. In more complex situations the problems are correspondingly multiplied.

In addition to these problems one is usually faced with inadequacies in our own field. The problem of providing laboratory assistance to hospitals in the quantity necessary to be of real help is a formidable one. Preparation of our own staff and inservice training of local health department personnel in technics not generally part of their routine is time consuming. In our case considerable experience with several epidemics was needed before we had acquired the conviction that we understood the problem well enough to recommend anything at all.

These remarks are not made in a spirit of criticism, but merely to point out some of the practical problems that face the practicing epidemiologist or the public health consultant in dealing with staphylococcal infections of the newborn. In our experience all concerned eventually welcome the assistance and accept the recommendations of the consultant. We feel that most local areas cannot adequately handle these situations without assistance. State and federal aid to local programs in terms of inservice training, epidemic aid, laboratory support, and a clearing house for the evaluation of evidence concerning newly discovered epidemic strains will become increasingly necessary.

In summary, we have presented data primarily confirming the work of others which clearly points to the hospital nursery as a source of family infections during epidemics of staphylococcal disease. We have attempted also to document the role of the staff person who is a chronic carrier in the epidemiology of epidemic staphylococcal disease and to establish the endemic phase as an important part of the natural history of these infections. Finally, we have pointed out some of the practical problems that face practicing epidemiologists in being of service to hospitals in controlling and preventing epidemic staphylococcal disease.

ACKNOWLEDGMENT—We wish to express our gratitude to Sister Eugene Marie and Sister Mary Florence of Good Samaritan Hospital, Cincinnati, and to Charles E. Mattix of the Lancaster-Fairfield Hospital, Lancaster, Ohio, for their cooperation and assistance in these studies. We also wish to thank Dr. Carl A. Wilzbach and Catherine Ludlow of the Cincinnati City Health Department and Dr. Lloyd Kersell and his staff of the Lancaster City-Fairfield County Health Department for their assistance in the field aspects of these studies. Dr. James Beasley and Elizabeth Bushell have worked very closely with us in these studies and know of our sincere appreciation.

REFERENCES

1. Colbeck, J. C. Extensive Outbreak of Staphylococcal Infections in Maternity Units (Use of Bacteriophage Typing in Investigation and Control). *Canad. M.A.J.* 61:557, 1949.
2. Barber, M.; Hayhoe, F. G. J.; and Whitehead, J. E. M. Penicillin-Resistant Staphylococcal Infection in Maternity Hospital. *Lancet* 2:1120-1125, 1949.
3. Rountree, P. M., and Barbour, R. G. H. Staphylococcus pyogenes in Newborn Babies in a Maternity Hospital, M. J. Australia 1:525, 1950.
4. Barber, M.; Wilson, B. D. R.; Rippon, J. E.; and Williams, R. E. O. Spread of Staphylococcus aureus in a Maternity Department in the Absence of Severe Sepsis. *J. Obst. & Gynaec. Brit. Emp.* 60:476, 1953.
5. Webb, J. F. Newborn Infections and Breast Abscesses of Staphylococcal Origin. *Canad. M.A.J.* 70:382, 1954.
6. Moseley, R. W. Acute Mastitis of the Newborn Due to Micrococcus pyogenes var. aureus. *U. S. Armed Forces M. J.* 5:371, 1954.
7. Rountree, P. M., and Freeman, B. M. Infections Caused by a Particular Phage Type of Staphylococcus aureus. *M. J. Australia* 42:157 (July 30), 1955.
8. Ravenholt, R. T., and LaVeck, G. D. Staphylococcal Disease—Obstetric, Pediatric and Community Problem. *A.J.P.H.* 46:1287-1296, 1956.
9. Shaffer, T. E.; Baldwin, J. N.; Rhaina, M. S.; and

- Sylvester, R. F., Jr. Staphylococcal Infections in Newborn Infants. I. Study of an Epidemic Among Infants and Nursing Mothers. *Pediatrics* 18:750, 1956.
10. Wysham, D. N.; Mulhern, M. E.; Navarre, G. C.; LaVeck, G. D.; Kennan, A. L.; and Giedt, W. D. Staphylococcal Infections in an Obstetric Unit. I. *Epidemiologic Studies of Pyoderma Neonatorum*. *New England J. Med.* 257:295 (Aug.), 1957.
11. Shaffer, T. E.; Sylvester, R. F., Jr.; Baldwin, J. N.; and Rheins, M. S. Staphylococcal Infections in Newborn Infants. II. Report of 19 Epidemics Caused by an Identical Strain of *Staphylococcus pyogenes*. *A.J.P.H.* 47:990, 1957.

Dr. Wentworth is chief; Mrs. Miller is nurse epidemiologist; and Mrs. Wentworth is bacteriologist-in-charge (Research Unit), Division of Communicable Diseases, State Department of Health, Columbus, Ohio.

This paper was presented before a Joint Session of the Engineering and Sanitation, Epidemiology, and Maternal and Child Health Sections of the American Public Health Association at the Eighty-Fifth Annual Meeting in Cleveland, Ohio, November 14, 1957.

CONTROL OF AN OUTBREAK OF STAPHYLOCOCCAL INFECTIONS AMONG MOTHERS AND INFANTS IN A SUBURBAN HOSPITAL

F. Robert Fekety, M.D.; Leon Buchbinder, Ph.D., F.A.P.H.A.; Elmer L. Shaffer, Ph.D., F.A.P.H.A.; Sidney Goldberg; H. Preston Price, M.D.; and Louis A. Pyle, M.D.

THIS REPORT describes an epidemic of staphylococcal disease affecting infants and mothers at the Valley Hospital in Ridgewood, N. J., from February to June, 1957. Thirty-three (10 per cent) of the 319 live births at the hospital during the epidemic were complicated by suppurative illnesses. Excluding carriers of the epidemic strain from the nursery and changing certain nursery procedures and technics temporarily controlled the epidemic. Despite the continuation of these measures, a second outbreak occurred. It ended when a carrier of the strain was found and excluded from contact with infants.

The Valley Hospital is a modern, attractive 118-bed general hospital; an average of 120 deliveries are performed there each month. The nursery consists of three connecting rooms with a capacity of 28 full-term infants in partitioned cubicles; it provides a minimum of 24 square feet of space for each infant. Additional rooms are provided for premature infants, isolation, examinations,

formula preparation, utility procedures, and as workrooms for doctors and nurses. The entire nursery is on one floor and is adjacent to the maternity unit. Prior to this study, air was supplied by a partial-recirculation air conditioner equipped with filters and an ultraviolet light.

Epidemic Background

Suppurative disease in infants and mothers was first noted in the latter part of 1955, whereupon isolation and aseptic technics were revised. Nursery linen was autoclaved, frequent hand-washing with hexachlorophene soap was required, individual bassinet technics were adopted, gown and mask technics were improved, and the nursery policy book was revised. A decline in the incidence of illness was subsequently noted.

Pustular infections were noted again in September, 1956. Despite the daily bathing of infants with hexachloro-