Spread of Hospital Staphylococci in Healthy Families—a Study from General Practice

Report* to the M.R.C. Committee for Research in General Practice

Babies born in hospital become carriers of *Staphylococcus aureus* very early in life (Hurst, 1957). The groin and umbilical stump are usually colonized first (Jellard, 1957; Gillespie et al., 1958) but soon tend to lose the organism; in the nose staphylococci settle rather later but are more firmly established, and in some maternity units over 90% of the babies are nasal carriers of *Staph. aureus* on the day they go home (Hurst, 1957). Staphylococci that colonize these infants are derived much more often from hospital sources than from the infants' own mothers (Barber et al., 1953) and are often antibiotic-resistant strains known to cause outbreaks of sepsis in hospitals. Their introduction into private homes might therefore lead to nasal colonization and sepsis in other members of the family. This has sometimes happened (Wentworth et al., 1958; Hurst and Grossman, 1960), but these incidents occurred at the time when *Staph. aureus* of phage type 80/81 was spreading freely and causing many outbreaks of sepsis in maternity units. Later studies suggested that babies lost their hospital-acquired staphylococci quite soon after going home (Miller et al., 1962; Hurst et al., 1964) and that the incidence of sepsis in their families was much the same as in control families (Oliver et al., 1964; Payne et al., 1965) found that staphylococci which were highly communicable in hospital spread less freely in the different environment of the private home.

The general practitioner is concerned to know which of these apparently opposing views is usually correct. He would like to know the answers to three questions about the behaviour of hospital staphylococci: first, is introduction into otherwise healthy families: do they spread to the noses of the family as they spread to other patients in hospital; do they have any special tendency to cause septic lesions; and what precautions, if any, should the family doctor take to protect families from the potentially dangerous strains that the babies, and to a lesser extent their mothers, bring from hospital?

This survey was an attempt to answer these questions. The plan was to determine the extent to which new strains of *Staph. aureus* were introduced into families by infants and mothers returning from maternity units and spread by them to other members of the family, and to record cases of sepsis caused by these organisms. The findings were compared with the spread of presumed non-hospital staphylococci introduced by other members of the same families. Comparative studies were also made on organisms introduced into families where a baby was born at home.

Material and Methods

A group of general practitioners—24 in Edinburgh and 25 in North-east England—called in a questionnaire for women booked for delivery during a period of six months. The details of each family were then analysed and families were chosen by a statistician to give equal numbers of hospital and home deliveries. The statistician also managed to ensure that the number of families chosen from the two areas was about equal, that the social status of the families in the hospital and the home group was comparable, and that there were enough schoolchildren in the families for a study of the part they played as introducers of non-hospital staphylococci.

Of the 1,780 families considered 220 were chosen, but 97 were unable to participate because of miscarriage, removal from the district, illness, and other reasons. Thus 123 families were studied and swabbed—52 in Edinburgh and 71 in North-east England. These families contained 615 persons whose noses were swabbed regularly. The swabs from 34 lodgers or occasional visitors had to be discarded because so many had moved out of the area; but, apart from this, co-operation was extremely good and at least 18 swabs were obtained from every member of all the families except five. Even in these five families there were enough swabs for satisfactory analysis.

Once a family was regarded as suitable the practitioner was notified, and with the co-operation of the family, the local authority, the health visitors, and the local bacteriological laboratory nasal swabs were taken every fortnight from all occupants of the house. Swabbing started four to six weeks before the baby was due and continued every two weeks until the baby was a year old. Septic lesions in any members of the family were reported to the doctor and swabs taken from all those with accessible pus.

One dry cotton-wool swab was used for both anterior nares of each subject. The swabs were sent to one laboratory in Edinburgh and six in North-east England. All swabs reached a laboratory within 24 hours, and most of them very much sooner than this. Swabs were plated, without preliminary enrichment, on blood-agar or milk-nutrient-agar. The plates were examined after overnight incubation at 37° C, and, when necessary, again after 24 hours at room temperature to increase pigment production. Colonies of bacteria thought to be *Staph. aureus* were tested for coagulate production by the slide technique, and a tube test was also used in doubtful cases.

All colonies of *Staph. aureus*—that is, coagulate-positive colonies—were tested for antibiotic sensitivity and were phage-typed. Some of the laboratories to which specimens were sent were not equipped to do phage-typing, and sent cultures to the typing centres that they normally used. Phage-typing methods and the criteria for identifying different strains were those specified by Blair and Williams (1961). In a few difficult cases antibiotic-sensitivity patterns were used to clarify the evidence of phage-typing.
Results

The fortnightly swabs from the noses (and lesions) of 615 persons in 123 different families were analysed. Into these families 125 babies were introduced—63 from hospital and 62 from home deliveries.

General Pattern of Staph. aureus Carriage

The widespread existence of Staph. aureus in normal families was confirmed. Staph. aureus was present at some time in 119 (97%) of the families and in 475 (77%) of persons. Table I shows the carriage rates for different family members. We have distinguished between "temporary" and "frequent" carriers—defined arbitrarily as those from whom Staph. aureus was recovered on less than and more than 20% of swabs respectively. Twenty-three per cent. of persons never carried Staph. aureus in their noses, 40% were temporary carriers, and 37% were frequent carriers (Table I). Gould and McKillop (1954) obtained almost identical results from a study of 520 persons.

Table I.—Nasal Carriage of Staph. aureus in Different Members of the Family

<table>
<thead>
<tr>
<th>Group</th>
<th>No. Examined</th>
<th>Non-carriers</th>
<th>Temporary Carriers (&lt;20% swabs positive)</th>
<th>Frequent Carriers (&gt;20% swabs positive)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Fathers</td>
<td>123</td>
<td>80</td>
<td>80</td>
<td>66</td>
</tr>
<tr>
<td>Mothers</td>
<td>123</td>
<td>30</td>
<td>37</td>
<td>28</td>
</tr>
<tr>
<td>Schoolchildren</td>
<td>112</td>
<td>24</td>
<td>48</td>
<td>35</td>
</tr>
<tr>
<td>Pre-schoolchildren</td>
<td>112</td>
<td>28</td>
<td>50</td>
<td>44</td>
</tr>
<tr>
<td>Newborn infants</td>
<td>125</td>
<td>31</td>
<td>63</td>
<td>50</td>
</tr>
<tr>
<td>All except newborn</td>
<td>490</td>
<td>102</td>
<td>22</td>
<td>181</td>
</tr>
</tbody>
</table>

There was no significant difference between the carriage rates of the various family members other than the newborn babies. In this group temporary carriage was more common than in any other family member, while frequent carriage was less common. The reason for this difference is shown in the Chart. The babies' carriage rates were high during the first few weeks after birth but declined to very low levels by the end of the year. From the eighteenth week onwards the mean carriage rate for all babies was only 7.6%. The Chart also shows great differences between the initial carriage rates of babies born in hospital and those born at home.

Staph. aureus Acquisition by Babies in Hospital

During the first few weeks of life Staph. aureus carriage was more common in babies born in hospital than in those born at home, but after 18 weeks there was no significant difference between the two groups (see Chart).

Staph. aureus was isolated from 31 (49%) of the 63 babies arriving home from hospital. Phage-typing and antibiotic sensitivity patterns suggested that in every case the strain was one that had not previously been present in the household, so we presumed, as other workers have found—for example, Barber et al. (1953) and Cook et al. (1958)—that the babies acquired the staphylococci from hospital sources. Thus there were 31 families in which hospital strains of Staph. aureus were introduced by a baby and had the opportunity to spread to other members of the family.

Effect of Introducing a Newborn Baby into a Family

After the three initial fortnightly swablings showed that the family pattern of Staph. aureus carriage, a new strain was reckoned to have been introduced if it had not been previously isolated from any member. The person responsible for the introduction was assumed to be the member who first yielded the new strain. Sometimes a new strain was first isolated from two members of the same family in the same fortnight; both were then counted as "introducers." Of the babies who came home after birth in hospital, 31 (49%) introduced new strains of staphylococci into their families; this was more than twice the rate of introduction by other members of the family (Table II).

Table II.—Introduction and Spread of New Strains of Staph. aureus in 123 Families

<table>
<thead>
<tr>
<th>Place of Confinement</th>
<th>Newborn Infants</th>
<th>Mothers</th>
<th>Members of Family other than Infants</th>
<th>All Persons</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Hospital Home</td>
<td>63</td>
<td>62</td>
<td>23</td>
<td>23</td>
</tr>
<tr>
<td>Hospital Home</td>
<td>62</td>
<td>61</td>
<td>24</td>
<td>24</td>
</tr>
<tr>
<td>Hospital Home</td>
<td>11</td>
<td>12</td>
<td>7</td>
<td>7</td>
</tr>
</tbody>
</table>

* See note in text.

Of the babies born at home 14 (23%) appear to have introduced staphylococci into the family. In two instances this was a "shared" introduction and the baby may not have been responsible. In one instance the home-born baby was admitted to hospital, and this was the probable source of the new strain. The remainder of new strains that were introduced by home-born babies probably resulted from fondling by neighbours and friends.

Of the new strains introduced by non-infant members of the family (presumably from non-hospital sources), just under half (49% and 47% in the home and the hospital groups respectively) spread to other members of the family (Table II). The 31 hospital strains introduced by infants showed no tendency to spread more freely than this; indeed, in only 11 (36%) was there any evidence from phage-typing that it had spread to other family members. These hospital strains were transmitted mainly to the mothers of the infants and to a lesser extent to the fathers and school-age siblings.

In 82 families one or more individuals were carrying staphylococci before the baby arrived. In 45 (56%) of these the baby picked up the family strain; Table III shows when this occurred. The percentage acquiring the original family strain...
of staphylococcus was the same for babies born in hospital as for babies delivered at home.

<table>
<thead>
<tr>
<th>Table III.</th>
<th>45 Babies (Hospital and Home-born) Who Picked up a Family Strain of Staph. aureus; Showing the Age (in Weeks) When the Pre-existing Family Strain Was First Isolated from the Baby's Nose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (weeks)</td>
<td>No.</td>
</tr>
<tr>
<td>-------------</td>
<td>-----</td>
</tr>
<tr>
<td>These 45 babies include those born in hospital and those born at home.</td>
<td></td>
</tr>
</tbody>
</table>

Thus our evidence suggests that staphylococci brought home by hospital-born babies were soon replaced by family strains and that they showed, if anything, less capacity to spread within the family than staphylococci introduced from other sources.

Incidence of Staph. aureus Sepsis

Possible staphylococcal lesions occurred in 50 (41%) of the 123 families. Twenty-eight (23%) of the 123 families and 43 (7%) of the 615 individuals in the survey had lesions that yielded *Staph. aureus* (11 boils; 7 septic spots; 4 impetigo; 4 infected eczema; 3 septic wounds, 2 each of whitlow, conjunctivitis, and sty; 1 each of paronychia, ophthalmia neonatorum, and nappy rash; and 5 unspecified).

Phage-typing failed to show any special tendency for staphylococci from hospital to cause lesions. In only one instance was a lesion shown to be of a similar strain to that introduced into the family by a hospital-born baby.

Relation between Nasal Carriage and Septic Lesions

Of the 43 persons with *Staph. aureus* lesions there were 34 in whom clear-cut phage types enabled a comparison to be made with nasal strains carried by other family members. In 24 (70%) of these the strain found in the lesion was the same as that in the patient's nose; in 5 (15%) it was the same as that in the nose of another member of the family; and in 5 (15%) it was not associated with any nasal strain or with any other manifestation in the family. In families with staphylococcal lesions nasal carriage was somewhat more common than in families without lesions (45% as compared with 36%).

Nasal carriage rates in families with lesions were the same before the appearance of the lesion as after it, suggesting that lesions followed rather than caused the higher nasal carriage rates.

Discussion

This work confirms the many earlier studies on the frequency of *Staph. aureus* nasal carriage. Of the 615 persons studied in the survey 40% were classed as “temporary” carriers and another 37% as “frequent” carriers. During their first few weeks of life newborn babies had the highest carriage rates, but these quickly declined to a mean rate of 7.6% from the eighteenth week after birth onwards. The reason for this very low rate for babies once they have become established members of the family is not clear; perhaps it is due to unknown immunological mechanisms or perhaps to their having more thorough washing and fewer outside contacts than the rest of the family. Schoolchildren, who had the highest carriage rates, usually wash the least thoroughly and have many close outside contacts.

We have also confirmed the earlier reports that newborn babies acquire *Staph. aureus* much more often in hospital than at home. At the time of the first swabblings the carriage rates were 49% for babies born in hospital and 18% for babies born at home, but by the eighteenth week there was no difference between the two groups (see Chart). The rate for hospital-born babies is relatively low when compared with the rates obtained by other workers. This could be a result of the slight delay between the time of discharge and the babies' first swabbing. It might also reflect the general trend towards lower nasal carriage rates in hospital-born babies that has been noted since 1961.

There are two possible explanations for the difference between carriage rates in the hospital and those in home-born babies: one is that staphylococci inhabiting hospitals are strains of greater inherent “communicability”; the other is that infants in hospital are exposed to a greater concentration of staphylococci and more opportunities for acquiring them. Our observations do not support the first of these possibilities; indeed, strains brought into the family by hospital-born babies spread within the family rather less often than strains introduced by other members from (presumably) non-hospital sources.

Staphylococcal lesions occurred with what seems to be the usual frequency. We detected them in 7% of our subjects; Kay (1962) reported a sepsis rate of 9% from a general practice in Manchester. But of the 34 lesions in our series from which typable strains of *Staph. aureus* were recovered, in only one had the organism been brought into the family by a hospital-born baby.

Families with staphylococcal lesions had higher nasal carriage rates than families without, and it has already been noted that the lesions appeared to be the result and not the cause of the high carriage rate.

It therefore seems that the questions we set out to study may be answered thus: (1) hospital staphylococci have no special tendency to spread in ordinary homes; (2) they have no special tendency to cause septic lesions outside hospitals; and therefore (3) the family doctor need make no special arrangements to protect families from staphylococci brought home from hospital by mothers and infants.

Summary

Noses and septic lesions of 615 persons in 123 families were swabbed every fortnight for four to six weeks before and for 52 weeks after the birth of a baby. Sixty-two babies were born at home and 63 at hospital. During the first 18 weeks of life babies born in hospital had higher *Staphylococcus aureus* carriage rates than babies born at home.

Hospital staphylococci brought home by babies were no more apt than were “domestic” strains to spread within the family or to cause septic lesions.

Staphylococcal lesions appeared to be the result rather than the cause of high nasal carriage rates within a family.

Thanks are due to the medical officers and health visitors of the following areas: Edinburgh, Newcastle, Northumberland, North Shields, Sunderland, Durham, Stockton, Middlesbrough, and Redcar.

References
