*STAPHYLOCOCCAL NASAL CARRIAGE IN THE FAMILY

CLIFFORD R. KAY, M.D.
General Practitioner, Manchester

The importance of nasal carriage in the aetiology of staphylococcal infection has been repeatedly emphasised, although little is known of the factors which influence the uneasy relationship between *Staphylococcus aureus* and its host.

The predominant natural habitat of *Staph. aureus* is Man, and the natural habitat of modern Man is the Home. It was therefore felt that an investigation of staphylococcal nasal carriage in this environment might prove fruitful.

A different aspect of this study has been reported previously (Kay, 1962). In this paper when “staphylococcus” is used without qualification, the species *Staph. aureus* is implied.

**Material and Methods**

The investigation was conducted between 1956 and 1959 in a small, single-handed general medical practice situated in a pleasant residential area in south Manchester. Thirty-seven families were investigated for periods ranging from 12 to 24 months. Anterior nasal swabs were taken from all members of each household every six weeks—a purely arbitrary interval—while any intercurrent staphylococcal lesion was recorded and usually swabbed.

The selection of families was partly influenced by the expectation that they would co-operate throughout a rather long study. On the basis of the limited knowledge of staphylococcal epidemiology then available it was not suspected that any bias would be introduced in this way, and analysis of the data obtained has not contravened this belief.

Seventeen of the families were primarily selected because one

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of their members had *presented* with a staphylococcal infection. These families are here called Septic Families. The remaining 20 families were selected as controls and matched the Septic Families fairly closely in respect of numbers, age structure, and social status.

Originally 41 families were investigated, but 4 had to be excluded from the analyses. Two families, whose investigation was started before the main part of the study, had been swabbed at irregular intervals; a third family moved out of the district; while in the fourth an antibiotic cream had been applied to the anterior nares as a prophylactic measure and was effective in eradicating the colonizing staphylococcus, so that the changes observed were artificially contrived and the family had to be excluded from the main calculations. Individuals who were observed for less than half the period of that of the whole of their families, mainly visitors and domestic staff, were also excluded.

**Bacteriological examinations**

Almost all the swabbings were done by the author. For the nasal examinations the swab was first moistened in sterile normal saline, then firmly rotated round the periphery of each anterior naris before being replaced in its tube. Exceptionally, after demonstration of the simple technique, a responsible person in the household was entrusted with the swabbing of those members who were not present when the writer made his visit.

All the technical procedures were carried out by Dr M. T. Parker and his staff in the Manchester Public Health Laboratory. The swabs were sent there as quickly as possible, usually by hand on the day of collection. Some which had to be collected in the evening or at weekends were sent by post.

A report was made on any organism cultured from the swabs and taken from lesions, and where appropriate the sensitivity of the organism to antibiotics. In the case of the nasal swabs a positive report was given only when *Staph. aureus* was cultured.

The sensitivity of all staphylococci was determined to the following antibiotics: penicillin, streptomycin, chloramphenicol, chlorotetracycline, and erythromycin. All staphylococci were typed by the bacteriophage method; the main purpose being to decide whether or not strains isolated from different members of one family, from different sites on one person, or from one person on different occasions, originated from a single strain. It is relatively rare to have complete identity of phage pattern in two cultures thought to be the same (Williams and Rippon, 1952). Thus an attempt had to be made to define how much the phage pattern of two cultures
must vary before considering them different.

This analysis of the phage typing was made by Dr Parker, and in the final form in which he reported the results all the cultures isolated from one family were classified into a number of different strains and the modal pattern of phage susceptibility for each strain was determined. These modal patterns were allotted to the main phage groups (Williams and Rippon, 1952; Williams, Rippon and Dowsett, 1953) by the criteria outlined by Parker (1958). As an example of the results obtained, the chart of Family 29 is reproduced in figure 1.

![FAMILY 29 Chart](image)

**FIGURE 1.**—Chart of Control Family No. 29.
Definition

It is generally agreed that about half the population are nasal carriers (Hallman, 1937; Gillespie, Devenish and Cowan, 1939; Packalen and Bergqvist, 1947; Rountree and Barbour, 1951; Knight et al., 1956; Masters et al., 1958). Study of these papers gives rise to some confusion since the writers record different aspects of nasal carriage. Indeed, they use the same term "carriage (or carrier) rates" to describe two different measurements:

1. The proportion of individuals found at a single swabbing to be carrying staphylococci.
2. The proportion of swabs positive for staphylococci found in repeated swabbings of individuals.

It seemed desirable that different terms should be used for these two different parameters. In this study, therefore, where there have been repeated swabbings of individuals the term Carriage Coefficient has been introduced. It is defined as follows:

\[
\text{Carriage coefficient} = \frac{\text{Number of positive swabs}}{\text{Number of total swabs}} \times 100
\]

Standard error of the difference = 2.5
Difference = 8 \ (= 3.2 x s.e.d.)

This has proved to be a most useful expression since it can be used as a measure of the susceptibility to staphylococcal nasal colonization of an individual, a family, or any group of individuals. All of these can be directly compared with each other so long as a reasonable minimum of observations has been recorded and the interval between swabbings is the same. In this study a six weeks swabbing interval has been used throughout.

Characteristics of Nasal Carrier State

In figure 2 the frequency of occurrence of carriage coefficients of the 146 individuals in this study are plotted. This histogram shows a U-shaped curve, which is of great significance since a single normal distribution cannot have two peaks. The most likely explanation of the curve is that it contains two different, overlapping distributions, i.e., that there are two distinct groups of nasal carriers. It is just possible that the peak between carriage coefficients 50 and 60 represents a third distribution but it is more likely that this is merely a chance variation due to the small numbers involved; evidence given later supports this explanation. This figure gives no indication of the distinguishing features of the two groups of carriers, but further analysis of the data gives the clue to this information.

The study was originally planned so that the experience of the septic families (as defined above) could be compared with that of
the controls. In table I the carriage coefficients of these two groups are shown. As expected, the carriage coefficient of the septic families is higher than that of the controls. Though the difference is statistically significant, it is small.

However, some of the control families developed sepsis and in table II these have been put with the septic families to form a group

### TABLE I

**COMPARISON OF CARRIAGE COEFFICIENTS OF SEPTIC AND CONTROL FAMILIES**

<table>
<thead>
<tr>
<th></th>
<th>Number of individuals</th>
<th>Coefficients of nasal carriage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control families</strong></td>
<td>83 (20)</td>
<td></td>
</tr>
<tr>
<td>Positive swabs</td>
<td>370</td>
<td>42</td>
</tr>
<tr>
<td>Total swabs</td>
<td>872</td>
<td></td>
</tr>
<tr>
<td><strong>Septic Families</strong></td>
<td>63 (17)</td>
<td></td>
</tr>
<tr>
<td>Positive swabs</td>
<td>340</td>
<td>50</td>
</tr>
<tr>
<td>Total swabs</td>
<td>686</td>
<td></td>
</tr>
</tbody>
</table>
of families which had staphylococcal infections. These were compared with those control families that did not develop lesions. The families in which lesions occurred have a carriage coefficient of 50 while the families without sepsis have a carriage coefficient of 39. The difference is more significant than in table I.

TABLE II
COMPARISON OF CARRIAGE COEFFICIENTS OF FAMILIES WITH AND WITHOUT SEPTIC LESIONS

<table>
<thead>
<tr>
<th>Families with lesions (24)</th>
<th>Coefficients of nasal carriage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of individuals</td>
</tr>
<tr>
<td>Families with lesions</td>
<td>88</td>
</tr>
<tr>
<td>(24)</td>
<td></td>
</tr>
<tr>
<td>Families without lesions</td>
<td>58</td>
</tr>
<tr>
<td>(13)</td>
<td></td>
</tr>
</tbody>
</table>

Standard error of the difference = 2.55
Difference = 11 (=4.3 x s.e.d.)

Of course, the families with lesions had many members that did not suffer from any sepsis. In table III these individuals have been removed from the families with lesions and added to the group of individuals comprising the families without lesions. This adjustment

TABLE III
COMPARISON OF CARRIAGE COEFFICIENTS OF INDIVIDUALS WITH AND WITHOUT SEPTIC LESIONS

<table>
<thead>
<tr>
<th>Coefficient of nasal carriage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of individuals</td>
</tr>
<tr>
<td>------------------------</td>
</tr>
<tr>
<td>Individuals with sepsis</td>
</tr>
<tr>
<td>Individuals without sepsis</td>
</tr>
</tbody>
</table>

Standard error of the difference = 3.1
Difference = 21 (= 7 x s.e.d.)

Compare with table II. Thus individuals without sepsis in "septic" families have the same carriage coefficients as those in families without sepsis.
leaves a group of individuals all of whom had a septic lesion and they are compared with an enlarged group of individuals who did not suffer from sepsis. The carriage coefficient of the individuals with sepsis is 61 while that of the individuals without sepsis is 40. It should be noted that the addition of the individuals without sepsis from the lesion families to the families without lesions has not significantly altered the carriage coefficient of the latter. In other words, the carriage coefficient of groups without sepsis are the same whether or not they are derived from families in which the other members have had some sepsis.

It will now be seen that two groups with widely different carriage coefficients are emerging. On the one hand there are those who have not had a septic lesion and who appear to be a homogeneous group because of the low, consistent carriage coefficient. On the other hand there are those who have had sepsis, but it is known that this group is unlikely to consist of uniform nasal carrier types because it has been shown (Kay, 1962) that about one third of individuals with sepsis are not nasal carriers of the lesion-producing strain. In Table IV this latter group has been separated from the individuals with sepsis and its carriage coefficient is found to be 36. This does not differ significantly from the carriage coefficient of individuals without sepsis (Table III).

Thus, those individuals with sepsis who are not carrying in their noses the strain of staphylococcus causing their lesions appear to belong to the same group of nasal carriers as the individuals who have not had any sepsis.

**Table IV**

Carriage coefficient of individuals with sepsis not carrying sepsis-producing staphylococci

(i.e., persons who are not nasal carriers, at that time, of the same phage-type as that found in their lesion)

<table>
<thead>
<tr>
<th>Number</th>
<th>Coefficient of nasal carriage</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Positive swabs</td>
<td>54</td>
</tr>
<tr>
<td>Total swabs</td>
<td>151</td>
</tr>
</tbody>
</table>

*Compare with carriage coefficient of individuals without sepsis (40). (See table III)

Standard error of difference = 4.2
Difference = 4 ( < s.e.d.)
TABLE V.

COMPARISON OF CARRIAGE COEFFICIENTS OF THOSE INDIVIDUALS WHO ARE SOURCES OF SEPSIS WITH THOSE WHO ARE NOT

<table>
<thead>
<tr>
<th></th>
<th>Number</th>
<th></th>
<th>Coefficient of nasal carriage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sources of sepsis</td>
<td>42</td>
<td>Positive swabs 317</td>
<td>69</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total swabs 461</td>
<td></td>
</tr>
<tr>
<td>Not sources of sepsis</td>
<td>104</td>
<td>Positive swabs 393</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total swabs 1,097</td>
<td></td>
</tr>
</tbody>
</table>

Incidence of sources of sepsis in control families = 13 per cent

Sources of Sepsis

The residue of individuals with sepsis now consists only of those who are carrying the lesion-producing strains of staphylococci in their noses. This group has the high carriage coefficient of 75. This observation suggested the possibility that a high carriage coefficient was due to carriage of staphylococci which gave rise to sepsis. To be consistent it was necessary to study in addition those individuals who carried these strains, but who did not themselves develop any lesion. In order to provide a convenient expression that will include all these carriers, the term "source of sepsis" has been introduced, and is defined as one who carries a staphylococcus in his anterior nares of the same strain as that found in a septic lesion in himself, or in another member of his family, at the same time.

Forty-two individuals conformed to this definition and in table V their carriage coefficient is compared with that of the remainder. The carriage coefficient of sources of sepsis at 69 is seen to be almost double that of those individuals who are not sources of sepsis (36). Proof that these groups represent two distinct types of nasal carrier depends upon demonstrating that the carriage coefficients of the individuals within them form two distinct distribution curves. These are shown in the histogram to figure 3 and it will be seen that they afford a complete explanation of the composite distribution noted in figure 2.

As the number of individuals in the two groups was different the contrast between the two distributions is partially concealed. To overcome this difficulty, in figure 4 each is calculated as a percentage, and the histogram reveals how clearly the two types of
STAPHYLOCOCCAL NASAL CARRIAGE

Figure 3. Frequency distribution of carriage coefficients of sources of sepsis and others.

Figure 4. Percentage distribution of carriage coefficients of sources of sepsis and others.
nasal carrier are distinguished.

Analysis of Sources of Sepsis

Incidence in the general population. The frequency with which sources of sepsis occur in the general population may be assessed by determining their incidence in the control families. There were 83 individuals in the control families and 11 of these were sources of sepsis. This gives an incidence of 13 per cent of the population in about 12 months.

Age distribution. The incidence of all sources of sepsis at different ages is shown in table VI. Even when these results are put into broader age groups no significant difference can be demonstrated. \( \chi^2 = 1.18 \quad P = 0.6 \)

<table>
<thead>
<tr>
<th>TABLE VI</th>
<th>AGE DISTRIBUTION OF SOURCES OF SEPSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age group</strong></td>
<td><strong>Number of sources of sepsis</strong></td>
</tr>
<tr>
<td>0—10 .. ..</td>
<td>15</td>
</tr>
<tr>
<td>10—20 .. ..</td>
<td>4</td>
</tr>
<tr>
<td>20—30 .. ..</td>
<td>12</td>
</tr>
<tr>
<td>30—40 .. ..</td>
<td>5</td>
</tr>
<tr>
<td>40—50 .. ..</td>
<td>3</td>
</tr>
<tr>
<td>50—60 .. ..</td>
<td>1</td>
</tr>
<tr>
<td>60—70 .. ..</td>
<td>2</td>
</tr>
<tr>
<td>70—80 .. ..</td>
<td>0</td>
</tr>
<tr>
<td><strong>Totals .. ..</strong></td>
<td><strong>42</strong></td>
</tr>
</tbody>
</table>

*Note—Age differences are not statistically significant

Sex distribution. In table VII the sex distribution of the sources of sepsis is shown. The differences again are not significant.

<table>
<thead>
<tr>
<th>TABLE VII</th>
<th>SEX DISTRIBUTION OF SOURCES OF SEPSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
<td><strong>Number of sources of sepsis</strong></td>
</tr>
<tr>
<td>Female .. ..</td>
<td>27</td>
</tr>
<tr>
<td>Male .. ..</td>
<td>15</td>
</tr>
<tr>
<td><strong>Total .. ..</strong></td>
<td><strong>42</strong></td>
</tr>
</tbody>
</table>

Standard error of the difference = 7.9
Difference = 12 \( (< 2 \times \text{s.e.}) \)
Analysis of Individuals not Sources of Sepsis

Table VIII gives the variations in carriage coefficients due to age and sex in individuals who are not sources of sepsis. Carriage coefficients are higher below 20 years than above this age. This agrees with the estimates of carriage rates by other writers. Under the age of 20 there is no sex difference in carriage coefficients, but above 20 years the females have a significantly higher carriage coefficient than the males. In this study individuals over the age of 20 years were almost exclusively the parents in the families observed. Thus it could be said that the mothers had a higher carriage coefficient than the fathers. This is an interesting finding but its significance is not yet clear. No reference has been found in the literature to any sex difference amongst nasal carriers.

### TABLE VIII

RELATIONSHIP OF NASAL CARRIAGE TO SEX AND AGE (excluding sources of sepsis)

<table>
<thead>
<tr>
<th></th>
<th>All ages</th>
<th>0—20</th>
<th>20—80</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of individuals—</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>52</td>
<td>22</td>
<td>30</td>
</tr>
<tr>
<td>Female</td>
<td>52</td>
<td>23</td>
<td>29</td>
</tr>
<tr>
<td>Analysis of swabbings—</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male positive</td>
<td>185</td>
<td>119</td>
<td>66</td>
</tr>
<tr>
<td>Total</td>
<td>560</td>
<td>223</td>
<td>337</td>
</tr>
<tr>
<td>Female positive</td>
<td>208</td>
<td>112</td>
<td>96</td>
</tr>
<tr>
<td>Total</td>
<td>537</td>
<td>238</td>
<td>299</td>
</tr>
<tr>
<td>Carriage coefficients—</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>33</td>
<td>53</td>
<td>20</td>
</tr>
<tr>
<td>Female</td>
<td>39</td>
<td>47</td>
<td>32</td>
</tr>
</tbody>
</table>

Conclusions

The main finding here is that all individuals may be divided into two distinct groups according to the way they react to the staphylococci contaminating their anterior nares. One group of individuals carries the strains of staphylococci which have been found in lesions in themselves or in their families. They show a strong tendency to persistent nasal colonization with their strains but no significant age or sex differences within the group. The other group of individuals carries staphylococci which were not found to cause any lesions during the period of observation. This group shows a tendency to resist nasal colonization by these staphylococci and there are clear age and sex differences. It is interesting to recall
how closely these observations agree with the findings of Miles, Williams and Clayton-Cooper (1944) that “there is a marked tendency for the persons to be persistent carriers or persistently free from nasal Staphylococcus aureus”.

Implications of the Findings

Two groups of nasal carriers

In figure 4 it was demonstrated that there are two different groups of staphylococcal nasal carriers. It was a characteristic of one group (the sources of sepsis) that they carried strains which had caused lesions, while the second group had encountered staphylococcal strains which were not observed to cause sepsis.

The differences between the two groups is emphasized by the marked disparity of their carriage coefficients (69 and 36—see table V) and by the shapes of their distribution curves. The distribution curve of the sources of sepsis is very close to a straight line, while the distribution of the individuals who were not sources of sepsis closely follows a normal distribution curve.

Differences not an artefact

Before discussing what might be the reasons for these differences it is necessary to dispose of the possibility that they are an artefact, in that the association of sepsis with the persistent carriers (sources of sepsis) might be due to the greater length of exposure of these individuals to their colonizing staphylococci.

Figure 4 shows that no less than 35 per cent of the individuals who were not sources of sepsis were also persistent carriers in that they had carriage coefficients higher than 50. Yet none of the strains that these individuals persistently carried was the cause of any sepsis. Thus a different length of exposure to their colonizing staphylococci could not account for the differences between the two groups of carriers. Further, the different shapes of the two distribution curves, as mentioned above, is good evidence that the differences are real.

Significance of the two groups of carriers

Logically then, these differences could only be due to differences in the carriers or differences in the staphylococci.

It will be seen from the shape of their distribution curve that the majority who were not sources of sepsis tended to resist nasal colonization by the staphylococci they encountered. It is well-established that staphylococci will survive without difficulty in environments far less favourable than the human nose. Thus, as
there is universal contact with these organisms, it seems most likely that the resistance of these individuals to nasal colonization is due to their ability to secrete some substance that is inimicable to the staphylococci.

Such a factor was postulated by Bergqvist (1950) but has never been identified, though it is known that bacteriostatic substances exist on the skin and in sweat and sebaceous glands. Such secretions could well be dependent upon hormonal factors, variations in which could account for the observed differences due to age and sex (table VIII) and the presence of a minority of persistent carriers in this group. On the other hand, the marked tendency to persistent carriage in the sources of sepsis must be due either to the absence in these of inhibitory factors or to the resistance of the lesion-producing staphylococci. There are two arguments which favour the second hypothesis:

1. If the sources of sepsis were a special group of individuals who failed to produce this inhibitory substance one would expect some correlation of this characteristic with either age or sex. Yet, no such age or sex variations were observed.

2. The absence of inhibitory factors in an individual must be either permanent or temporary. The concept of a permanent change (that is, a congenital absence) implies a lifelong susceptibility to staphylococcal infection. This is contrary to clinical experience. Yet a temporary change would have to occur in several individuals simultaneously in order to account for the frequent finding of more than one source of sepsis in the same family. Such a coincidence is highly unlikely.

Negative evidence thus strongly favours the proposition that the differences observed between the two types of carrier are due to differences in the staphylococci they have encountered. Since one set of staphylococci have produced lesions and the others have not, these different host-microbe relationships appear to provide a clear corroboration of the long-suspected differences in staphylococcal virulence for man. If so, then one distribution represents a host-symbiont relationship while the other shows a host-parasite relationship.

The straight-line distribution of the sources of sepsis remains to be explained. It seems possible that this distribution represents the outcome of random, infrequent encounters between a susceptible host population and the organism. In addition, or alternatively, it may represent random mutation in the organism from virulence to non-virulence. Without invoking this mechanism it is difficult to see how colonization with a virulent strain would ever come to an
end. A further modifying factor is that the population is not uniformly susceptible to these organisms, since there is evidence from hospital that the presence of another staphylococcus in the nose may prevent colonization by virulent hospital strains (Rountree and Barbour, 1951; Clarke, 1957).

It is important to discover if any other characteristics of the lesion-producing and the other staphylococci can be demonstrated.

*Incidence of lesion-producing strains*

An estimate of the proportion of all strains that are capable of producing lesions in the general community may be obtained from their incidence in the control families. A total of 64 different strains of staphylococci were encountered and seven of these were found in lesions. This gives an incidence of 11 per cent. This prevalence of virulent strains is easily reconcilable with the observed incidence of staphylococcal infection in the community (Kay, 1962).

*Spread of staphylococci*

In table IX it is shown that a significantly higher proportion of lesion-producing strains than of other strains have been acquired from members of the family. This is evidence of the greater ability of virulent strains to spread to others, and was to be expected from the known facility with which these strains establish nasal colonization. Ability to spread from person to person is an important element of virulence.

**TABLE IX**

**COMPARISON OF PROPORTIONS OF LESION-PRODUCING STRAINS AND NON-LESION-PRODUCING STRAINS ACQUIRED WITHIN THE FAMILY**

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Acquired within family</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Percentage</td>
</tr>
<tr>
<td>Non-lesion-producing strains</td>
<td>148</td>
<td>54</td>
</tr>
<tr>
<td>Lesion-producing strains</td>
<td>73</td>
<td>40</td>
</tr>
</tbody>
</table>

Standard error of the difference = 7
Difference = 18 (= 2.6 x s.e.d.)

Hare and Thomas (1956) and Hare and Ridley (1958) found that certain individuals were especially prone to disperse large numbers of staphylococci into their immediate surroundings. They called these people "dangerous carriers". Williams (1959) suggested that
"a dangerous carrier ordinarily carries a dangerous staphylococcus", and the observations here support this view in that virulent strains more readily contaminate the skin in large numbers and so spread to clothing and the environment.

Barber and Burston (1955) and Roodyn (1960) have suggested that the presence of a lesion enhances the dispersion of staphylococci. This appears to be the result of the greatly increased contamination of the skin around lesions, and provides another reason for the ability of lesion-producing strains to spread to others.

**Correlation with bacteriophage groups**

The proportions of lesion-producing and non-lesion-producing strains belonging to each bacteriophage group were compared and the only difference noted was the absence of untypable strains from the lesion-producing group, as previously observed by Gould and McKillop (1954a).

Williams (1959) analysed phage patterns of strains obtained from hospitals during outbreaks of staphylococcal infection. He found that a certain few phage patterns were frequently present amongst the staphylococci which caused lesions, and he called these the "epidemic strains". The number of lesion-producing strains in this study was not sufficient to analyse them in this way. Correlation of virulence with phage pattern is important, because it provides supporting evidence that all strains of *Staph. aureus* are not uniformly virulent.

**Correlation with antibiotic resistance**

The proportions of antibiotic resistant organisms occurring amongst the lesion-producing strains and the non-lesion-producing strains in this study are compared in table X. There is a significantly higher proportion of antibiotic-resistant organisms amongst the virulent strains. Rountree and Rheuben (1956), similarly found that

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Antibiotic-resistant</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Number</td>
</tr>
<tr>
<td>Lesion-producing strains</td>
<td>31</td>
<td>19</td>
</tr>
<tr>
<td>Non-lesion-producing strains</td>
<td>102</td>
<td>38</td>
</tr>
</tbody>
</table>

Standard error of the difference = 10
Difference = 24 ( = 2.4 x s.e.d.)
25.7 per cent of strains derived from healthy carriers were penicillin resistant while there were 63.5 per cent of resistant strains obtained from lesions in private patients and those attending casualty departments.

In hospitals, practically all strains that cause lesions are now resistant to penicillin, and resistance to the tetracyclines has been used as a simple method of selection of the potentially virulent strains (Gillespie and Alder, 1957; Williams et al., 1959). However, it is natural that antibiotic resistant strains should concentrate in a hospital since lesions due to these organisms are most likely to require hospital treatment. Also, the frequent treatment with antibiotics in hospital eliminates the sensitive strains (Lepper et al., 1953; Laurell and Wallmark, 1953; Knight, 1954; Gould and McKillop, 1954b; Clarke, 1957; Francis and Spicer, 1960). Contamination of the air by antibiotics has the same effect, even in untreated individuals (Gould, 1958).

The finding of a correlation between lesion strains and antibiotic resistance outside hospital is of greater significance. The resistance of staphylococci to antibiotics is due to several different factors, and it is very difficult to postulate a biochemical connection between the property of virulence and these apparently unrelated mechanisms of antibiotic resistance. Yet the increasing occurrence of serious staphylococcal infections in hospitals has coincided with the introduction of the antibiotics (Finland et al., 1959) and it is difficult to escape the conclusion that there is some correlation between virulence and antibiotic resistance.

**Mechanism of staphylococcal infection**

The data given earlier strongly supports the view that staphylococci differ in their virulence for man and these differences may be recognized, though only in retrospect, by the way in which the strains colonize man.

In the past it has been difficult to explain outbreaks of infection because of the constant presence of staphylococci in the environment. If one accepts, however, that only about ten per cent of these strains are capable of producing lesions, it is clear that virulent staphylococci are by no means constantly present and there is no theoretical difficulty in accepting that infection follows chance contact with these virulent organisms. This mechanism was most clearly seen in the seven control families which developed lesions, and an example is shown in figure 1. In each, after a varying interval, infection followed the introduction of the lesion-producing strain into the family.
Possible host factors

It is not suggested that variations in host susceptibility never play a part in the initiation of staphylococcal disease. Indeed, in hospital, there can be little doubt that the increasing scope of major surgery and the use of potent drugs such as the corticosteroids are important additional factors (Howe, 1954; Dowling et al., 1955; McDermott, 1956; Slaney and Brooke, 1956). But in the home there is little evidence that changes in the resistance of the host to infection have much bearing on outbreaks of staphylococcal disease. It would be prudent, however, not to dismiss the possibility that there may be some truth in the frequently proffered explanation of the patient that he is "run down".

Comparison with other communicable diseases

It seems, then, that outbreaks of staphylococcal infection are probably the same in principle as outbreaks of any communicable disease, e.g., influenza. There are, however, several important differences in detail: (1) Man has a fairly high resistance to staphylococcal infection, even by virulent strains, (2) after contact, staphylococci establish a carrier state, often of long duration, and (3) there is virtually no immunity to staphylococcal infection—lesions can follow one another in quick succession.

Development of a lesion

Elek and Conen (1957), showed that very large numbers of staphylococci were required to initiate a purulent lesion experimentally, and they commented that it was highly unlikely that such a large number of organisms could be accidentally introduced into the tissues in natural infection. They did, however, provide a clue as to how infection might arise when they suggested:

"In naturally occurring skin lesions small changes in virulence may be far less important than the unexplained mechanism whereby secretions in sweat glands prevent the free growth of staphylococci".

Evidence has been presented here, however, that virulent strains may owe this property to their ability to overcome these inhibitory influences of surface secretions. Thus it is quite possible that once introduced into a sweat gland or hair follicle, virulent staphylococci can multiply freely until the critical size of micro-colony is reached. When this bursts into the surrounding tissues an abscess will result. It may well be that minor trauma is a precipitating factor, either by causing the rupture of the micro-colony or by forcing the organism into the deeper layers of the skin.
Summary

A study of staphylococcal nasal carriage in 37 families is described. It was found that two distinct types of staphylococcal carriage existed. In one, carriage tended to persist for a long time, and variations in the duration of carriage showed no correlation with the age or sex of the carrier. In this group all the staphylococci were strains that had been cultured from one or more lesions. In the other group there was a clear tendency for nasal colonization to be resisted by the carrier, and differences observed were clearly related to age and sex differences in the hosts. None of the staphylococcal strains in this group had been found in a lesion during the period of observation.

It is suggested that the difference between the two groups is due solely to differences in the colonizing staphylococci, and that the observations therefore distinguish between a group of staphylococci capable of causing lesions (estimated to be only about ten per cent of all strains encountered) and a much more widespread group of strains which do not give rise to infection under natural conditions.

On the basis of this theory and on the observation of the origin and course of infection in twenty-four families, an attempt is made to explain the mechanism of staphylococcal infection.

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The Australian College of General Practitioners. **Queensland Faculty.**

This faculty publishes a newsletter after each board meeting, and the July 1962 issue reveals that the Australian Council is, like its British counterpart, concerning itself with the question of holding examinations. Queensland Faculty favours an entrance examination for membership, and a status of Fellowship to be awarded to members for distinguished service, with associateship to remain for those not wishing to take the examination for membership.

Other reports in the newsletter show that the faculty is busy with the same sort of activities as occupy the College's attention elsewhere,