

Evolution of the hospital staphylococcus

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In accepting the invitation to give this paper, I was pleased that one question had been settled for me, and that I was not going to have to justify the existence of such an organism as a hospital staphylococcus. To those of us working in hospitals twenty years ago, hospital staphylococci were very real entities. Many epidemiologists have commented on doctrines founded on impressions, but I believe that our impressions then can be supported by a reasonable amount of evidence. Most of this evidence, if not nearly all of it, depends on the ability to type staphylococci and therefore dates from the general introduction of phage typing in the immediate post-war years.

I have, though, spent some time wondering if we can get any hints of hospital or epidemic staphylococci in the prephage typing days. It may be that epidemic staphylococci arose *de novo* as a result of antibiotic treatment, but this seems improbable, and it is much more likely that in the long evolutionary history of the staphylococcus from time to time strains appeared with the special properties of the epidemic. In looking back over past records, one is handicapped by the late adoption of coagulase testing as a method of differentiating *Staphylococcus aureus* from other staphylococci, and by the general paucity of accurate records. Trying out one of the earlier penicillins, we thought that if we used it in the treatment of two then common afflictions—boils and carbuncles, we would not need a control series of untreated patients, because published records in the past would surely tell us how long these lesions took to heal. Unfortunately, despite many papers about them, we were not able to find one that gave us this vital fact.

Hospital staphylococci have probably been studied most intensively in surgical wards and here accurate records from the past would have been invaluable, but again the reports are not as numerous or as detailed as one would have wished. It would have been particularly interesting to know what was happening in the early years of the century. At the time of Sir

Alexander Ogston's discovery, staphylococcal infection of wounds was obviously common. By the end of the century, antiseptic and then aseptic measures had generally been adopted by surgeons, and hospitals were becoming healthier places. My uncle's copy of the 1910 edition of Rose and Carless' *Manual of Surgery*, then in its seventh edition, makes practically no reference to wound infection by staphylococci. Was it because the various preventive measures had resulted in nearly all surgical wounds healing quickly and soundly or was it perhaps because staphylococcal infection was a relatively unregarded form of sepsis when compared with streptococcal infection with its much greater potential hazards?

After this length of time, I doubt if there is any hope of finding out how often staphylococcal sepsis really occurred. That it did occur, we know. Staphylococci were isolated from gunshot wounds in the 1914–18 war (Stokes & Tytler, 1918–19) and a number of papers record the isolation of staphylococci from the wounds of patients undergoing surgery in hospital (see McKhann *et al.*, 1938; Devenish & Miles, 1939). What may not be remembered, now that most doctors in practice have been trained after the discovery of antibiotics and the renewed attention to asepsis that occurred at about the same time, is that circumstances that we now know to be conducive to staphylococcal infection and spread were common in the earlier years of the century. With my own students I do not think I could claim, as was said of Sir Alexander Ogston that 'absolute order prevailed in his class, preserved simply by the consciousness of his presence', but I am sure that mine would agree that my student days were a very long time ago, and in 1937, when I did my first surgical dressing, we were, in fact, only a little over halfway through the known life of the staphylococcus. In that appointment for five days a week for three months I dressed a patient with a chronic empyema; and I can well remember that during that time I noticed that the pus surrounding the drainage tube had become tinged with green. Looking back, it is not difficult to see what might have happened. The dressing was carried out, as was quite proper then, with fingers and forceps, and at the end I used to reverse the forceps, putting the handle into the communal ward pot of vaseline so as to obtain enough to make the safety pin through the drainage tube sit comfortably. Practices like these were helped to disappear in this country by the publication of the M.R.C. War Memorandum No. 6 1941—The Prevention of Hospital Infection of Wounds, that described non-touch techniques for wound dressing; and that drew heavily on the observations of Miles *et al.* (1940).

Anecdotes about faulty sterilization are familiar, and I can recall listening

to Dr Carl Walter of Boston describing an autoclave that he had examined in the post-war period, that had been in use for a considerable time, but in which the steam had never been connected to the autoclave chamber. It reminded me of one hospital I visited where woollen blankets were being autoclaved with no harmful effects in an autoclave used for sterilizing dressings. Nearer home I knew of one very large dressing autoclave used for theatre drums that produced such wet dressings and theatre drapes that the theatre sister hung them out on the swab rack to dry before the operating session began.

Leaving surgical patients for the time being, I have looked to see if any help could be gained from medical records of staphylococcal infection, but again, with little success. There are, though, accounts of patients with influenza complicated, sometimes fatally, by staphylococcal pneumonia in the influenza epidemic at the end of 1914–18 war (Chickering & Park, 1919; Patrick, 1923). The doctors concerned, in America and in Malta, would have recognized familiar features in the staphylococcal infections that occurred in this country during the outbreak of Asian influenza in 1957.

Hospital, or as they were called by many, epidemic staphylococci, first appeared officially in Australia where Rountree and her colleagues (Isbister, Durie, Rountree & Freeman 1954; Rountree & Freeman, 1955; Rountree & Beard, 1958) isolated a group I staphylococcus of phage type 80 at the time of an extensive outbreak of sepsis in a Sydney hospital. A similar organism was isolated from hospitals in other parts of Australia and very soon afterwards, type 81 was described in Canada (Bynoe *et al.*, 1956). Most type 80 strains were lysed by phage 81 and were thought to be the same type as the 81 or the 80/81 of the USA and Canadian reports.

By 1957, 80/81 was responsible for nearly all the epidemics in maternity units in the United States, and in this country for nearly half of the epidemics in hospital. It was common as an epidemic strain in the Netherlands, in Eastern Europe and in Uganda (for references, see Williams, 1959). Based on his experience as Director of the Staphylococcus Reference Laboratory at Colindale, Williams stated that 'We are in fact at present observing the pandemic spread of type 80—a strain that has more recorded epidemics to its credit than any other type; whose epidemics are, in our experience, twice as extensive as the average of all other types; and which (like other group I strains) commonly generates a high rate of carriage (and often a high incidence of skin lesions) among the hospital staff'.

Where type 80/81 came from was never known, and indeed it is not certain that Australia was its country of origin, rather than the country in

which it was first isolated and recognised. It may have arisen spontaneously in different parts of the world but the view is held that all strains sprang from a single source (Asheshov & Winkler, 1966) and if so, one of its properties must have been the ability to spread on a very wide scale.

By the early 1960s, type 80/81 and its lysogenized derivatives (the 52, 52A, 80, 81 complex) began to disappear, and with this disappearance there was a dramatic change in the picture of staphylococcal disease in hospitals in this country (Parker, 1980). Two isolated examples in which this type retained or rediscovered its earlier ability are worth noting. Poole & Baker (1966) reported it as being responsible for sepsis in nineteen members of the staff of a veterinary laboratory, and two years later we reported an outbreak due to type 52/52A/80/81 that had many features in common with outbreaks caused by type 80/81 when it was first recognized, and was of a kind that we had not seen for at least eight years (Shooter *et al.*, 1968). The outbreak was in a female medical ward in which, within a few days, no less than ten nurses and eight patients developed boils. Further evidence of the inherent capacity of this staphylococcus to cause infection was shown by the facts that the only patient in the adjacent male ward carrying the strain developed a boil, as did a patient discharged home, and two nurses who were thought not to be carriers, reported with boils due to this organism three and five months later. Were we seeing an outbreak due to a staphylococcus that had lain dormant for eight or nine years, or was it the result of some combination of events that restored to this particular strain the properties that made the original strains of type 80 so notorious?

Although type 80/81 was the first and best known of the epidemic staphylococci, it was by no means the only staphylococcus qualifying for this unsatisfactory description. For outbreaks in maternity units, Williams (1959) lists four types—80, 52A/79, 71 and 7/47/53/54/75 as being responsible for half the outbreaks, and three types 80, 75/77 and 47/53/75/77 as being responsible for half the outbreaks in surgical wards. At the time, with the exception that type 80 was more likely to cause boils, there appeared to be little difference in the behaviour of these epidemic strains.

One other example of epidemic staphylococcal infection is worth reference, although in some respects it differs from those mentioned so far. In 1951 Jackson *et al.*, described from Boston staphylococcal enterocolitis occurring as a complication of the treatment of patients with oxytetracycline. It was characterised by the acute onset of tachycardia, fever, circulatory collapse and profuse watery diarrhoea occurring shortly after surgical operation, and its distinguishing feature was an invasion of the bowel wall by staphylococci. This condition was soon reported from other parts of America and, after a short delay, from Europe. The great majority

of staphylococci responsible belonged to group III, and although it is probable that a limited number of types in this group could produce the disease, it was not associated as closely with individual types as were some other staphylococcal infections. At its peak, staphylococcal enterocolitis was not uncommon. On one occasion we had three patients with it in a surgical ward at the same time, and Cooke *et al.*, (1957) described two outbreaks involving 31 cases, 14 of whom died. With the general decline of the epidemic staphylococcus, the disease became progressively more uncommon and, as far as I know, is very rarely seen today.

The reasons why some strains of *S. aureus* two decades or so ago should have possessed those qualities that entitled them to be labelled 'epidemic' have never been clearly defined. Neither had it been possible by laboratory examination of a culture to say whether a staphylococcus had these qualities or not. In this country nearly all epidemic strains were resistant to tetracycline, and had more resistance to mercury than less virulent strains (Moore, 1960), but there were many tetracycline and mercury resistant staphylococci isolated that did not have epidemic propensities or, at any rate, had not been displaying them when isolated from patients, and the first strains of type 80 isolated in Australia were resistant to penicillin, but not to other antibiotics. However, although antibiotics may not have been directly responsible for the appearance of epidemic strains, it is hard to resist the thought that in some way they exerted a population pressure that assisted the emergence of epidemic strains. In a somewhat different context, staphylococcal enterocolitis depended on the prior use of antibiotics to condition the gut before staphylococci could multiply in its wall.

Staphylococci will survive for weeks in floor dust and on blankets and it could have been that epidemic strains were those that retained their virulence longer in the dried state than did other strains. This hypothesis has been studied but no conclusive evidence has been put forward on its behalf and it would not fit with our own experience. When studying a large number of patients nursed in beds with unwashed woollen blankets, we were able to find no clear example of transfer of a staphylococcus from one patient to the next occupant of the bed, and at a time when epidemic strains were common. In fact, my own view then was once a staphylococcus had been shed by a carrier, in normal circumstances it might remain viable for weeks, but it had lost its ability to infect a fresh patient within twenty-four hours or less.

The ability to spread easily from person to person was obviously high on the list of properties needed by epidemic staphylococci, and this was shown very clearly by the world-wide spread of type 80 within a few years; and yet

ability to spread easily was not enough alone. In a study of a surgical ward extending over eight and a half months, we found over 180 different types of staphylococci (Shooter *et al.*, 1958). Of this number, however, only thirteen were responsible for any observable clinical sepsis, and only three types for sepsis in more than one patient. There was nothing to suggest that lack of sepsis with some strains could be explained by lack of opportunity. In fact, three of the strains present in the ward almost all the time and often in very large numbers were amongst those strains not responsible for sepsis.

Carriers of *S. aureus* vary greatly in their ability to shed the organism to their surroundings, and plainly the patients, nurses or doctors who were heavy shedders and who carried epidemic staphylococci, constituted a greater danger than those who dispersed staphylococci in small numbers. But this was a reflection of the carrier's behaviour, and there was no evidence that epidemic strains were more likely to be shed more easily than other strains.

Perhaps the outstanding property of epidemic staphylococci was their ability to cause infection in a large number of the patients—and for type 80 the nurses—that carried them. In one study (Williams *et al.*, 1959) we found that no less than a third of hospital patients, whose noses were colonized with epidemic strains, went on to develop sepsis as compared with only 2.5 per cent of patients who were nasal carriers of other types. The earlier papers about type 80 suggested that it caused more serious sepsis as well as more sepsis, but in the years that followed, this was not our experience. Serious sepsis certainly occurred but not more than would have been expected considering the number of patients infected.

Looking back, it seems to me to be indisputable that in the 1950s around the world we were confronted with staphylococci quite remarkably able to cause infections, and that this ability, whatever it was, was not possessed by one phage type alone. It would be gratifying to think that the work devoted then to studying the natural history of the staphylococcus and the resulting preventive measures had been so successful that it could take credit for the present demotion of the staphylococcus as a cause of infection in a hospital. But although hospitals are greatly improved, I believe that this would be an illusion. Some day, I fear, another strain of staphylococcus will get afresh the ability shown so well by 80/81, and doctors and surgeons will have to face again an epidemic staphylococcus. When this occurs perhaps modern techniques of genetic investigations will enable microbiologists to say just why some strains of *S. aureus* are epidemic and others are not.

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