Outbreak column 14: *Staphylococcus aureus* - new outbreaks of old infections

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Outbreak Columns 12 and 13 provided an overview of historical nosocomial *Staphylococcus aureus* outbreaks, and reviewed the guidance designed to control both outbreaks and resistant epidemic strains (Curran, 2014a,b). Still with a focus on outbreaks of *S. aureus*, Outbreak Column 14 seeks to bring the reader up to date. Specifically, it will be looking at the epidemiology of outbreaks of nosocomial *S. aureus*, both methicillin resistant (MRSA) and methicillin sensitive (MSSA), in work published between 2010 and 2013. Where relevant, the findings will be compared to a review of 47 outbreaks published in a seminal work by Williams (1959). The aim is to identify salient points that infection prevention and control teams (IPCTs) should be mindful of in order to best prevent, prepare for and manage similar outbreaks in today's changed and changing healthcare systems.

I was concerned that a third column on the same topic might be somewhat tiresome, but I was reassured by some wise words published in a 1959 *Lancet* editorial.

'In the past year we have published four fairly long editorials and many shorter ones on the problems created by staphylococcal infections acquired in hospital; and we have also discussed at some length the related problems of inefficient sterilisation and misused antibiotics. Must we risk boring our readers with a further article on the same subject? We have no option. Patients, putting their lives in the hands of the profession, are entitled to be protected from any avoidable ignorance or indifference; and the time has not yet come when we can feel sure that they have such protection.'

[*Staphylococcal Disease in Hospitals*, (Anon, 1959).]

Be assured, I am not suggesting that IPCTs are indifferent to the topic of *S. aureus*. But I am suggesting that the literature is worth another trawl for infection prevention and control (IPC) treasure.

What should be highlighted is that attempts at epidemiology from published outbreak reports come with caveats, because as Williams (1959) also notes, this is not a random population. Published outbreak reports are an unrepresentative sample from an unknown population.

Staphylococcus aureus outbreaks as reported by Williams in 1959

Staphylococcus aureus was first recognised as a significant nosocomial pathogen in the 1950s. This was either because of its new emergence or because the arrival of penicillin and consequent successful removal of Streptococcal outbreaks had resulted in its pathogenic presence being revealed (Curran, 2014a). The seminal work by Williams (1959) summarises the learning from 47 outbreaks (32 in maternity units and 15 in surgical wards).

Williams (1959) used two data sources for his paper. He compared the 1954–57 data from the *Staphylococcus* Reference Laboratory and the published epidemic reports. Both sources of information came in for criticism: the Colindale data because it lacked epidemiological information, and the published reports because 'of the authors' natural preference for recounting detective stories in which the culprit is found and a tendency to accept the simplest explanation, or an explanation that best fits preconceived ideas' (Williams, 1959). This criticism is an early description of what Reason (1990) describes as 'problems of causality' (p. 91). Because readers of this column are involved in determining outbreak causation I shall provide a few words on other types of causality problems to illustrate some of the potential pitfalls arising from this bounded rationality:

- **Oversimplifying causality:** investigators guided by what is known to have happened in similar outbreaks in the past are likely to fail to identify differences in current outbreaks which have dissimilar causal transmission pathways (Reason, 1990).
- **Availability heuristic:** Causes which are not present and obvious to the investigators are not looked for and are ignored. This has been summarised as: what you see is all there is (WYSIATI) (Kahneman, 2011). IPCTs are particularly susceptible to this problem because the jigsaw puzzles that are given to outbreak investigators to solve, seldom contain all the pieces; and there is rarely a picture to follow.
- **Confirmation bias:** early hypotheses are usually based on poor and incomplete data; but even after new and more robust data favours a counter-hypothesis, investigators tend to be unwilling to part with their initial hypothesis (Reason, 1990).

Returning to the body of the paper by Williams (1959), there were just two clinical locations where these outbreaks took place: maternity units and surgical wards. At the time of his reporting it is worth remembering there were few specialist units and certainly no transplant units.

Epidemics in maternity units

Between four and 320 persons were involved in the 32 outbreaks reported from maternity units, of which:

- 31/32 (97%) outbreaks involved a single staphylococcus type
- 27/32 (84%) outbreak investigators screened people to look for a source
 - 13/27 (48%) outbreak investigators considered they had found a source
 - In 1/13 (8%) the source was thought to be a septic patient
 - In 12/13 (92%) the source was a nurse (of whom five had infected lesions)

Williams (1959) commented on the attribution of causation: '*In 4 of the epidemics the evidence implicating the carrier seems to have been good, but in most of the others the attribution is only a reasonable probability. In only 2 epidemics did removal of the carrier appear to have been the only successful method of control adopted.*' I take this to mean all other usual methods were first tried.

Epidemics in the surgical wards

The 15 surgical unit outbreaks reported involved between six and 230 cases, of which:

- 11/15 (73%) had a single staphylococcal type
- 14/15 (93%) investigators screened people
- 6/14 (43%) involved cross-infection in the theatre from a health-care worker, only one of whom had septic lesions. (In the one outbreak where there was no screening, cross-transmission is also thought to have taken place in the theatre).
- 2/15 (13%) implicated the theatre air supply
- 2/15 (13%) involved ward cross-transmission (one introduced by a septic patient)
- In 4/15 (27%) it was not possible to identify an individual source of the infection

Despite the stated limitations of this non-random sample, Williams (1959) commented '*that it is however, very striking how often in these outbreaks, a single individual [HCW] either was responsible for all the cases or was responsible for introducing the infection and communicating it directly to some of the patients.*' These individuals were more often surgeons or theatre nurses. Indeed, of the 14 surgical outbreaks which identified a source, 6/14 (43%) were considered to have involved a healthcare worker (HCW) as a source. Although in one outbreak transmission arose in both the ward and the theatre, in the remaining five, transmission took place in theatre. It appears that the source is more likely to be attributed to an individual when there are fewer (<30) cases (data not presented). This could be because early on in an outbreak there was only one mode of transmission, e.g. HCW-to-patients in an operating theatre. However, the longer the outbreak goes on, the greater the number of sources that are dispersing the organism and the more difficult it becomes to confirm any particular transmission pathway.

In summary, Williams (1959) described *S. aureus* outbreaks in two clinical locations involving surgical and maternity patient populations. The infections caused were surgical site infections, abscesses and

other superficial skin and soft tissue lesions. The sources were at times attributed to individual HCWs; in particular when the outbreak was investigated early. The next part of this column will try to determine what has changed with modern staphylococcal outbreaks.

Methods

For this column I took a more methodological approach. In order to look at recent outbreak reports I undertook a Pubmed and an outbreak-database.com search using the terms '*Staphylococcus aureus*' and 'hospital outbreak'. The search was limited to reports published in English since 2010. The outbreak reports were included if the outbreak ended in 2007 or later, and details of the number of cases and the investigations that took place were included in the report. This resulted in 59 abstracts being reviewed, of which 14 were rejected; 43 papers were reviewed (two could not be retrieved); this includes one paper identified via a separate outbreak search. Following full paper review, 32 papers were selected for outbreak epidemiology, which included three identified via outbreak-database.com.

Results

Thirty-two *Staphylococcus aureus* outbreak reports that contained sufficient information and were published since 2010, of outbreaks that ended on or after 2007 were included. Of the 32 outbreak reports, 24 involved MRSA and eight involved MSSA; one MSSA outbreak also involved two MSSA strains and one MRSA outbreak also involved two MSSA strains

Place

Eleven (34%) of the 32 reported outbreaks occurred in neonatal intensive care units (NICUs; includes one SCBU and one combined PICU and NICU), five (16%) arose from care delivered in outpatient settings and five (16%) in surgical wards. The surgical wards involved were specialist areas, three outbreaks having occurred in cardio-surgical wards (Table 1). Just three (9%), of the outbreaks were from maternity units (mothers and newborn babies); such settings had been responsible for 32/47 (68%) of similar outbreaks in the Williams' (1959) report. There were also three (9%) outbreaks reported from burns units and ICUs respectively (Table 1).

Duration of the outbreak

The outbreaks lasted between 1 week and 32 months (median 3 months; interquartile range 1 to 9 mths). The outbreak duration varied by clinical location. There were shorter lasting outbreaks arising in the outpatient settings (five outbreaks: median 1 week) compared with the burns (three outbreaks: 2, 16 and 32 months) or surgical wards (five outbreaks: median 12mths), Table 1.

People involved

In these 32 outbreaks, 525 persons were involved, of which 261 (243 patients, six family members and 12 HCWs) were infected (Table 1). It must be emphasised that the number of persons who were reported as being involved in these outbreaks is likely to have been an underestimate and dependent on how thoroughly, how frequently and when during the outbreak the investigators screened for additional cases. There were three no-infection outbreaks (all NICUs reporting colonisation). See Outbreak Column 8 for definitions of no-infection outbreaks (Curran, 2013).

Infections caused

The nosocomial infections that developed reflect the opportunities for *S. aureus* entry that arise as a consequence of the health care delivered, i.e. the opportunities that enable what is on our patients to get in them (Table 2). For example, surgical site infections in surgical ward patients,

Table 1. *Staphylococcus aureus* outbreak details (by location, duration, people infected and staff)

	Outbreaks	Median duration (range) (Mths = months)	Patients infected	Family members infected	Staff infected	Staff colonised or infected
NICU (inc I SCBU)	11	4 mths (0.5–12 mths)	38	2	1	18
Outpatient settings	5	1 week (1 week–1 mth)	31	0	0	3
Surgical wards (Cardiothoracic 3; liver transplant; neuro (including ICU))	5	12 mths (4–14 mths)	68	0	0	24
ICU (1 included general wards)	3	3 mths (3–5 mths)	26	0	0	0
Maternity/newborn nursery	3	2 mths (2–9 mths)	48	2	0	12
Burns	3	16 mths (2–32mths)	27	2	2	13
General wards	1	4 mths	4	0	6	6
Dermatology wards	1	1 mth	1	0	3	3
	32		243	6	12	79

Table 2. The infections caused in *Staphylococcus aureus* outbreaks by location

	Skin and soft tissue (SSTI)	Surgical site infections	Bloodstream infections	Pneumonia includes (ventilator associated)	Burn wound	Joint infections	Other
NICU (inc. SCBU)	X		X	X			
Outpatient settings	X		X			X	Meningitis
Surgical wards (Cardiothoracic 3; liver transplant; neuro (inc. ICU))	X	X	X	X			Abdo sepsis
ICU (1 included general wards)	X		X	X			
Maternity/newborn nursery	X		X				
Burns	X	X			X		
General wards	X		X				
Dermatology wards	X						

SSTI includes: abscess, furunculosis, bullous impetigo, mastitis in mothers, conjunctivitis

burn wound infections in burn patients and skin and soft tissue infections in neonates with immature skin. Bloodstream infections (BSI) arose in most clinical settings probably due to biofilm formation on invasive devices (Table 2). The BSI and joint infections that arose in the outpatient outbreaks underscore once again that unsafe injection practices cause significant problems. The types of infections that developed will also have been related to the specific strain types and toxins that the different staphylococci produced. There is, however, insufficient space within this column (and insufficient author expertise), to provide sufficient discourse on that aspect of the topic area here.

Microbiological screening of healthcare workers

Healthcare workers were screened in 24/32 (75%) of the outbreaks (Table 3). On two occasions, during protracted outbreaks, HCWs were screened three times (Pinto et al, 2013; Teare et al, 2010). The overall positivity rates were low— with a couple of exceptions: one surgical ward screening had a positive rate of 8% (21/256, of whom

five worked in theatre) (Tadros et al, 2013); one screening of newborn nursery staff yielded an 11% (9/86) positive rate (Sanchini et al, 2013) and the HCWs in the combined outpatient settings had an 18% positive rate. Of note the screening of outpatient staff involved a much lower denominator ($n=17$).

Healthcare worker screening was done in 18/24 (74%) of MRSA outbreaks; and non-outbreak strain MRSA were identified in 6/18 (33%) of these outbreaks.

MSSA outbreak – HCW screening was done in 6/8 (80%) MSSA outbreaks; and MRSA was an incidental finding in two of the six outbreaks (30%).

It is not possible to definitively state in how many of the outbreaks which resulted in surgical site infections whether HCW screening included all theatre staff – though this was definitely done in one screening. The use of the term ‘all staff with hands on care’ as written in one report does not suggest that theatre staff were included, but it is difficult to be sure.

Table 3. The number of healthcare workers (HCW) screened and positive for outbreak strain

Location	HCW outbreak screening	HCWs screened and positive	Mean & (range)
NICU (inc SCBU)	10/11	1/87; 0/181; 2/259; 2/166; 2/161; 7/142; 1/134; 1/154; 0/201; 2/138; 2/146; 1/284*	1.0%; (0–5%)
Outpatient settings	3/5	1/7; 1/3; 1/7	18%; (14.2–33%)
Surgical wards	4/5	1/NS; 1/76; 1/56; 21/256	6%; (1.3–8%)
ICU	1/3	0/114	0.0%; (0–0%)
Maternity/newborn nursery	3/3	0/NS; 3/564; 9/86	2%; (0.5–11%)
Burns	3/3	2/60; 1/80; 2/785; 2/515; 2/442*	0.5%; (0.3–3.3%)

NS = Not stated; *1 outbreak screened HCWs 3 times

Healthcare worker involvement

In 12/32 (38%) of the outbreak reports the investigators considered that one or more HCWs played a pivotal role. As in the Williams (1959) paper the evidence for this is variable. There are a couple of instances where the evidence is strong. For example, in one outbreak in a burns unit, the investigators identified a single colonised HCW with eczema and did further work to demonstrate that the worker was a heavy disperser (Boers et al, 2011). The staphylococcal content of the air was determined after the HCW undressed in a room (Boers et al, 2011). In another report, a dose-response was identified by a case-control study. There was only one of 76 HCWs colonised with the outbreak strain – who also had psoriasis (Crusz et al, 2013). The case-control study showed that the more exposure patients had had to the positive HCW, as measured in HCW shifts, the more likely they were to be positive (Crusz et al, 2013). There are also two reports of outbreaks with a strain more commonly found in a specific geographic area which was preceded by an HCW coming from that area – and being the only HCW positive for the outbreak strain in the outbreak ward (Ali et al, 2012; Scheithauer et al, 2013). In one of these reports, after having found a member of staff was positive and recently employed from Romania where the outbreak strain was prevalent, investigators also screened other new staff from that country and found a further 9/51 HCWs were also positive (Scheithauer et al, 2013).

Environmental screening

In 17/32 (53%) outbreaks investigators either did not attempt environmental screening, or if they did, they did not present any results. Of the 15 reports where investigators undertook environmental or equipment screening, six reports included a numerator but no denominator (three of these reported positive samples). In the remaining outbreaks 9/32 (28%) investigators screened between 23 and 169 sites with a median 1.1% positive (range 0–2%).

Discussion

This Outbreak Column aimed to bring those involved in outbreak management up to date with what is new on *S. aureus* outbreaks, but there have been several limiting factors in achieving this. Firstly, in four years of reporting (2010–13) and throughout the healthcare systems of the world there appear to be only 32 reported outbreaks. It can only be concluded therefore that, in something of a paradox, the under-reporting of outbreaks in the scientific literature is of pandemic proportions. Secondly, what is reported reflects an apparent journal requirement bias of *there must be something new* and finally, even when outbreaks are reported, because of the variations in investigation and reporting it is extremely difficult to undertake further study. Therefore, I also make a plea for IPCTs to publish and provide three explicit reasons for doing so:

- To enable preparedness by alerting colleagues as to what nosocomial outbreaks are current and how they are varying over time,
- To enable optimal outbreak management by shared learning of the value of various interventions – what is new, useful and what is of little value.
- To prevent outbreaks by identifying and rectifying outbreak-provoking conditions before outbreaks arise in other settings.

There should be a forum for reporting outbreaks which would at least ensure the sample is representative. There is a means of extracting outbreak data in a systematic way using the outbreak-database.com (Vonberg et al, 2011) and perhaps such a forum should be adapted to become a repository that could be used by investigators directly, rather than requiring extractions from peer-reviewed publications. It has previously been argued that healthcare-related outbreak reports should be published just because they arise (Curran and Coia, 2010). Also, investigators should see the publishing of outbreak reports as part of our duty of care (Curran and Wilson, 2008).

The ORION guidelines for transparent reporting were the first attempt to standardise outbreak reports and intervention study reports (Stone et al, 2007). Having previously commented on the limitations of this work (Curran and Coia, 2010), this review of *S. aureus* outbreaks has convinced me further that outbreak reports should be completed with their own specific reporting template and include outbreak-provoking factors, with full specifications of investigations and findings.

Having addressed the limitations of this work, there were some valuable insights in what has been published. The key material in this discussion relates to: variation in outbreak management, place, infections caused, populations affected and HCWs' involvement.

Variation in outbreak management: these reports show that outbreak investigations currently lack uniformity – some involve people, environment screening and analytical studies while others involve little more than the arrival of the IPCT and a reminder that what should be always done to prevent infection is not currently being done. Crucially, there is little or no explanation as to why what was done was done or omitted, during the outbreak management process. On the face of it this seems like an unsafe way to fly a plane. However, this variation may well reveal IPCTs taking a local pragmatic risk-based approach that reflects how the organism is presenting (infections or colonisations), the vulnerability of the patients affected and the epidemic curve/timeline assessment. This is in fact the local risk-based approach as advocated in the 1998 and 2006 MRSA guidance (Anon, 1998; Coia et al, 2006). Sometimes just re-emphasising the basics is sufficient to gain control. And, as soon as new cases cease to arise, it could be argued that the rationale for further investigations also ceases to be. There is however a case for a comprehensive

list of what are considered 'the IPC basics' for both the IPCT and the clinical team. This should include the *Actions to stop transmission* and the *Investigations to determine if...* This will enable us to provide a rationale for what was done and omitted. There also needs to be an additional list of control measures for the outbreaks that don't stop when the IPC basics are confirmed to be in place.

As the IPC basics should be sufficient to prevent transmission in most incidents, then such measures should also be acceptable for alert signals that at least initially cannot be confirmed as an outbreak. The benefits of such an approach were highlighted in the outcome of one outbreak. After all results were in, typing showed the incident involved only two babies colonised with the same strain of MRSA (Mangini et al, 2013). Unrelated strains were found in another baby and five HCWs. However, to get to that final summary of events, there had been screening of 21 neonates, 201 HCWs and two parents; a multidisciplinary team had been set up; and a review of pertinent patient histories and infection control practices had taken place. Alert signals must be acted upon, but these actions must be proportionate to the presentation of events. When it is not possible to confirm or refute an outbreak, these basic IPC measures should be sufficient to prevent spread and give the IPCT time to be sure what, if any, further investigations and actions are needed.

Outbreaks are reported as a single unique event but they are always set in a much bigger context of what, for both the clinical area and the IPCT, is a very big day job. A necessity to be always addressing the most pressing needs requires constant prioritisation and reprioritisation of the IPCTs' workload. What for the reader of outbreaks would be more complete investigation reporting is for the IPCTs, given these competing priorities, an unnecessary luxury.

Place: Reported *S. aureus* outbreaks arise from care delivered in similar and very different clinical locations to that which occurred in the past. More commonly today outbreaks are reported in units whose patients are much more vulnerable than in the past, e.g. from NICUs. Outbreaks still arise, albeit less frequently than in the past, from surgical wards and maternity units (affecting mothers and babies). This suggests that the general care given in these areas has improved markedly such that patients are not being exposed or acquiring infections as in the past. However, the outbreaks that arose from care delivered in outpatient settings must serve as a reminder to IPCTs that as health care changes so will the outbreaks that present. As a consequence IPCTs' detection systems must be modified to ensure they remain effective at detecting novel outbreak presentations.

Infections caused: The infections that arise in *S. aureus* outbreaks reflect the risks from the health care delivered within the units, and the ways that *S. aureus* can cause disease, e.g. surgical site infections in surgical wards, babies with skin pustules, mothers with abscesses in maternity units, and injection-site-related infections in day or outpatient services.

Healthcare worker involvement: In the Williams study, 43% of surgical outbreaks and 48% of maternity outbreaks were attributed to one or more HCW. In the recently reported outbreaks 12/32 (38%) implicated an individual HCW. In both Williams' review and the review for this column the evidence to support such claims is variable. The HCWs involved in the outbreaks reported by Williams (1959) were more likely to be theatre than ward-based; however, as noted above this may in part be due to a lack of screening theatre staff. It may also be that what was not known in the 1950s (HCWs in theatre with pustules cause surgical site infections), is now known and not practised, i.e. a surgeon or any theatre personnel would not be allowed or allow themselves to be present if they had a skin infection. Other theatre practices have no doubt also improved over time.

The incidental finding of non-outbreak MRSA strains in HCW nares is not surprising. Some people are colonised, which usually does them and their families no harm; and there is little evidence of cross-transmission from them. However, when colonised HCWs have an

upper respiratory tract infection it has been shown that they can increase the dissemination from their nares up to 40 fold (Sherertz et al, 1996) and become what is termed a 'cloud' adult. It is tempting to consider whether some of the *S. aureus* outbreaks resolve with the implementation of basic IPC measures and because one or more nasal carrier has had a cold (outbreak-provoking condition) and as the cold symptoms disappear so does the heavy dispersal of staphylococci and the outbreak. In the absence of a cold, it is also known that occasionally some HCWs are heavy dispersers, in particular – although not universally – if they have an exfoliating skin condition or an infection (Sherertz et al, 2001). A heavy disperser can increase the amount of *Staphylococcus aureus* in the environment to levels such that the usual cleaning and decontamination regimens may not be able to cope. One or two reports in both the Williams (1959) and this review identified outbreaks which only ceased with, as a very last resort and following failed decolonisation attempts, the removal of the HCW from duty.

Role of the environment: In these outbreak reports there was variation in environmental screening. Additionally, given the duration of the outbreaks (some running into many months) and the number of sites that could have been screened over that time period, it is difficult to conclude anything from the odd positive swab found in a mattress or a stethoscope. An odd positive swab suggests little more than at some point in time through direct contact, indirect contact or through airborne dissemination, contamination of a specific site occurred (and that there is no evidence of a heavy disperser being present). It does tell us what we already know – hand hygiene should be done as per the 5 moments, equipment should be decontaminated after each usage, environmental sites should be regularly and effectively cleaned. And that anything less than this could lead to cross-transmission to patients and staff (and their families). I refer the reader to a useful chapter on the 6 'classes of evidence' suggesting that a fomite has a role in causing disease by a particular pathogen (Rhame, 1986).

Conclusions

Staphylococcus aureus outbreaks, like our healthcare systems, are changing and we must change with them to ensure our systems retain the capability to prevent, detect and manage them.

The variation in outbreak management as reported here shows that sometimes it is possible to end an outbreak of *S. aureus* with limited interventions, investigations and control measures.

To reduce variation in outbreak management there should be an agreed list of IPC actions that should be instigated immediately on recognition of an alert signal; and a further list of additional IPC measures to be used should the initial measures be insufficient to gain control. Looking for and excluding HCWs with colds might prove a useful additional 'basic' control measure.

To learn from outbreaks there needs to be an improved reporting and an outbreak reporting system.

The changing locations for outbreaks of *S. aureus* reflect to some extent the improvements in the prevention of surgical site infections, mother and baby care and the environments in which that care is delivered. Nevertheless this column shows that *S. aureus* (in all its forms) remains a formidable challenge in our healthcare settings.

Limitations

There was insufficient space in this column to cover what whole genome sequencing is bringing to outbreak management.

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