

Prevention of Invasive Group A Streptococcal Disease among Household Contacts of Case Patients and among Postpartum and Postsurgical Patients: Recommendations from the Centers for Disease Control and Prevention

The Prevention of Invasive Group A Streptococcal Infections Workshop Participants^a

The Centers for Disease Control and Prevention hosted a workshop to formulate recommendations for the control of invasive group A streptococcal (GAS) disease among household contacts of persons with invasive GAS infections and for responding to postpartum and postsurgical invasive GAS infections. Experts reviewed data on the risk of subsequent invasive GAS infection among household contacts of case patients, the effectiveness of chemoprophylactic regimens for eradicating GAS carriage, and the epidemiology of postpartum and postsurgical GAS infection clusters. For household contacts of index patients, routine screening for and chemoprophylaxis against GAS are not recommended. Providers and public health officials may choose to offer chemoprophylaxis to household contacts who are at an increased risk of sporadic disease or mortality due to GAS. One nosocomial postpartum or postsurgical invasive GAS infection should prompt enhanced surveillance and isolate storage, whereas ≥ 2 cases caused by the same strain should prompt an epidemiological investigation that includes the culture of specimens from epidemiologically linked health care workers.

Invasive group A streptococcal (GAS) infection is defined by the isolation of GAS from a normally sterile site (e.g., blood) or by the isolation of GAS from a nonsterile site in the presence of the streptococcal toxic shock syndrome or necrotizing fasciitis [1–4]. An es-

timated 8800 cases of invasive GAS disease and 1000 deaths due to invasive GAS infection occurred in the United States in 2000 (3.1 cases and 0.4 deaths per 100,000 population). The overall case-fatality rate for invasive GAS infections is 12%–13% [1, 5, 6]; the rate is highest (30%–80%) among persons with streptococcal toxic shock syndrome [3, 5–12]. Because of the burden and severity of invasive GAS infection, it is critical to identify opportunities for prevention of this disease.

To discuss new information [5, 13] (K. Robinson, personal communication) and to update previous recommendations [14], the Centers for Disease Control and Prevention (CDC) hosted a meeting in October 2000 to formulate recommendations for the control of the disease among household contacts of persons with invasive GAS infections and to formulate guidelines for

Received 12 March 2002; revised 28 May 2002; electronically published 26 September 2002.

This article presents the findings of the Prevention of Invasive Group A Streptococcal Infections Workshop held at the Centers for Disease Control and Prevention in October 2000.

Financial support: Centers for Disease Control and Prevention.

^a Workshop participants are listed at the end of the text.

Reprints or correspondence: Dr. Matthew R. Moore, Foodborne and Diarrheal Disease Branch, Centers for Disease Control and Prevention, 1600 Clifton Rd., Mailstop D-63, Atlanta, GA 30333 (mmoore4@cdc.gov).

Clinical Infectious Diseases 2002;35:950–9

This article is in the public domain, and no copyright is claimed.
1058-4838/2002/3508-0006

Table 1. Summary of 2 studies of the risk of subsequent invasive group A streptococcal (GAS) disease among household contacts of case patients.

Epidemiological feature	Canadian study [6]	US study ^a
Surveillance area	Ontario, Canada	CT, MN, and counties in CA and OR
Period of observation	Jan 1992–Jun 1995	Jan 1997–May 1999
Surveillance population, millions of persons	10.7	12.1
Sporadic cases detected, no.	732 ^b	1064
Incidence of sporadic disease, cases/100,000 population per year	2.4 ^b	3.5
Household contacts of index patients, no.	1360	1514
Subsequent cases detected, no. ^c	4	1
Syndromes observed in each index patient/ subsequent case patient pair	Bacteremia (index), pneumonia (subsequent); bursitis (both); soft-tissue infection (both); necrotizing fasciitis (both)	Bacteremia (index), necrotizing fasciitis (subsequent)
Attack rate, cases/100,000 population (95% CI)	294 (80–750)	66 (2–367)
Proportion of subsequent cases, % of all invasive GAS cases	0.6	0.1

^a K. Robinson, personal communication.

^b Figures listed differ from data published in the reference. Revised figures are based on follow-up surveillance data (A. McGeer, personal communication).

^c Within 30 days after a culture from the index patient was positive for group A streptococci.

responding to postpartum and postsurgical GAS infections. The CDC invited experts in the epidemiology and management of GAS infection and representatives of leading public health and clinical associations. The present report states the positions of the CDC and not the official policy of other organizations.

PREVENTION OF SUBSEQUENT INVASIVE GAS INFECTIONS AMONG HOUSEHOLD CONTACTS OF PERSONS WITH INVASIVE GAS DISEASE

Epidemiological Features of Invasive GAS Infection among Household Contacts of Case Patients

Risk of subsequent invasive GAS disease. For the purposes of the present article, a household contact is defined as a person who spent at least 24 h in the same household as the index patient during the 7 days before the onset of the case patient's symptoms [15]. This definition is intended to distinguish household contacts from other close contacts, such as children in day-care centers or schools or residents of long-term-care facilities. A case of subsequent invasive GAS disease is defined as invasive GAS infection that develops after exposure to a person with a confirmed case. Two studies have systematically estimated the risk of subsequent invasive GAS disease among household contacts of persons with culture-confirmed invasive GAS infections (table 1) [6] (K. Robinson, personal communication). Population-based active surveillance for invasive GAS infections in Ontario, Canada, from January 1992 through June 1995 identified 4 cases of subsequent invasive GAS disease among 1360 household contacts of persons with invasive GAS

infections [6]. All 4 subsequent cases occurred among spouses or adult siblings of index patients; there were no deaths.

The second study was performed between January 1997 and May 1999 among a surveillance population of 12.1 million (in Connecticut, Minnesota, and selected counties in Oregon and California) (K. Robinson, personal communication). The investigators identified 1 confirmed case of subsequent invasive GAS disease and 1 probable case (i.e., a culture-negative clinical syndrome consistent with invasive GAS disease) among 1514 household contacts. Both patients with subsequent invasive GAS disease were close adult contacts of the index patients, and neither died. In summary, 2 prospective studies that were designed to identify subsequent cases among household contacts (who were observed for a total of 66.5 million person-years) identified only 5 confirmed cases of subsequent invasive disease (table 1).

Potential burden of chemoprophylaxis. On the basis of these 2 prospective studies, we estimate that 12,000–22,000 household contacts per year would be candidates for chemoprophylaxis. If chemoprophylaxis were 100% effective, 8–64 cases of subsequent invasive GAS infection per year would be prevented in the United States.

Antimicrobial therapy can have undesirable effects, including adverse drug reactions and selection for resistant organisms [16]. One means of minimizing antibiotic use while maximizing its benefit would be to recommend prophylaxis only for those household contacts who are at the highest risk of subsequent invasive GAS infection and/or for those at the highest risk of death from invasive infection. It is not possible to identify such

risk factors on the basis of the 5 documented cases of subsequent invasive GAS disease. However, epidemiological studies of invasive GAS infection have identified several risk factors for sporadic disease (table 2) [1, 6, 17] (S. Factor, personal communication). The only risk factor consistently associated with mortality due to sporadic invasive GAS infection is being ≥ 65 years of age [1, 5, 6, 17].

Potential Effectiveness of Chemoprophylactic Regimens

No controlled trials have evaluated the effectiveness of chemoprophylaxis in preventing invasive GAS disease among household contacts of persons with invasive GAS infections. In studies of eradication of upper respiratory tract carriage of GAS, intramuscular administration of benzathine penicillin G in combination with orally administered rifampin was the only penicillin-containing regimen that eradicated chronic, asymptomatic pharyngeal carriage of GAS [18]. Orally administered clindamycin is as effective as intramuscular benzathine penicillin G plus rifampin [19, 20]. Orally administered azithromycin was 95% effective in eradicating asymptomatic pharyngeal carriage of GAS among school-aged children [13]. Among US Marine Corps recruits, orally administered azithromycin

prevented acquisition of pharyngeal colonization with GAS [21] (table 3).

Recommendations for the Prevention of Invasive GAS Disease among Household Contacts of Persons with Invasive GAS Infections

Antimicrobial chemoprophylaxis for any infectious disease is most desirable if disease is severe; if defined risk groups can be identified; and if a safe, affordable, and effective chemoprophylactic regimen is available [23]. Although the risk of subsequent invasive GAS disease among household contacts is higher than the risk among the general population, subsequent invasive GAS infections among household contacts are rare. Given the infrequency of these infections and the lack of a clearly effective chemoprophylactic regimen, the available data do not support a recommendation for routine testing for GAS colonization or for routine administration of chemoprophylaxis to all household contacts of persons with invasive GAS disease. The CDC recommends that health care providers routinely inform all household contacts of persons with invasive GAS disease about the clinical manifestations of pharyngeal and invasive GAS infection (e.g., fever, sore throat, and localized mus-

Table 2. Factors that increase the risk of sporadic invasive group A streptococcal disease.

Risk factor, age in years	Cases per 100,000 population/year	Relative risk (95% CI) ^a	Reference
Advanced age			
≥ 75	5.7	—	[6]
Overall ^b	1.5	—	
≥ 65	8.6	—	[1]
Overall ^b	3.5	—	
HIV infection, 20–60	—	9.4 (3.5–25) ^c	[6]
Diabetes mellitus, all ages	—	3.7 (2.4–5.8)	[6]
Chickenpox, <10 ^d	—	39 (16–90)	[6]
Cancer, all ages	—	6.9 (4.5–10)	[6]
Heart disease, all ages	—	8.4 (6.0–12)	[6]
Injection drug use			
18–44	—	14.7 (2.5–85.7)	
≥ 45	—	10.3 (1.1–94.4)	PC ^e
Steroid use, ≥ 45	—	7.1 (1.1–45.7)	PC ^e
Native Americans			
Without diabetes, all ages	32	—	[17]
With diabetes, all ages ^f	109	—	

^a Reference groups include persons in the same age group who did not have the indicated risk factor.

^b All age groups combined.

^c Not adjusted for history of injection drug use.

^d During the first 2 weeks after the onset of chickenpox.

^e S. Factor, personal communication.

^f All cases occurred in the group aged 45–64 years.

Table 3. Summary of trials that used antimicrobial agents for the eradication of group A streptococci (GAS) from the upper respiratory tract.

Reference	Study design	Main outcome measurement	Treatment (no. of patients)	Results
[22]	Unblinded clinical trial during an outbreak; patients self-selected a treatment group	Eradication of GAS from the oropharynx	BPG im (100)	75 courses of treatment (75%) eradicated GAS
			Penicillin V po (62)	45 courses of treatment (72%) eradicated GAS
[18]	Randomized, unblinded, controlled, crossover clinical trial	Eradication of GAS from the oropharynx of persistently colonized children	None (13)	Precrossover: GAS eradicated in 3 subjects (23%)
			BPG im (10)	Precrossover: GAS eradicated in 3 subjects (30%)
			BPG im plus rifampin po (14)	Precrossover: GAS eradicated in 13 subjects (93%); total (including crossovers): 81% of courses of BPG-rifampin eradicated GAS and 26% of non-rifampin-containing courses
[19]	Randomized, unblinded, controlled crossover clinical trial	Eradication of GAS from the oropharynx of persistently colonized children	Clindamycin po, then BPG im plus rifampin po (26)	GAS eradicated in 26 subjects (100%)
			BPG im plus rifampin po, then clindamycin po (22)	GAS eradicated in 15 subjects (68%); all subjects with cultures negative for GAS at 3 weeks were equally likely to have negative cultures at 6 and 9 weeks
[20]	Randomized, blinded, controlled clinical trial	Eradication of GAS from the oropharynx of persistently colonized children and adults	Clindamycin po (26)	GAS eradicated in 26 subjects (100%)
[13]	Unblinded, uncontrolled assessment of azithromycin for treatment of oropharyngeal carriage of GAS	Eradication of GAS from the oropharynx, determined 12 and 27 days after the completion of treatment	Penicillin V po (22)	GAS eradicated in 8 subjects (36%)
			Azithromycin po 5 times/day (152)	142 (95%) of 150 subjects had cultures negative for GAS at day 17, and 127 (91) of 140 subjects had negative cultures at day 32

NOTE. BPG, benzathine penicillin G.

cle pain) and emphasize the importance of seeking immediate medical attention if contacts develop such symptoms. Studies have suggested that a heightened index of suspicion for subsequent GAS disease should be maintained for 30 days after the diagnosis is made for the index patient [24–29] (K. Robinson, personal communication).

Although routine chemoprophylaxis for all household contacts is not recommended, subsequent invasive GAS infections do occur, albeit rarely [6, 24–28] (K. Robinson, personal communication). Certain underlying illnesses and other host factors are consistently associated with an increased risk of sporadic invasive GAS infection in persons exposed to the organism [6, 17] (S. Factor, personal communication). Once infected, persons aged ≥ 65 years are at increased risk of death [1, 5, 6, 17]. Therefore, although chemoprophylaxis is not recommended routinely for household contacts, health care providers may choose to offer chemoprophylaxis to household members aged ≥ 65 years or those at increased risk for sporadic invasive GAS

infection (table 2). The CDC does not recommend routine use of culture to identify household contacts who are colonized.

Clustering of asymptomatic carriage of GAS among members of a household is common, and the source of GAS in households is not necessarily the person with invasive GAS infection; therefore, providers who choose to prescribe chemoprophylaxis for an elderly or high-risk member of a household should prescribe chemoprophylaxis for all members of that household. If chemoprophylaxis is prescribed, the CDC recommends any 1 of 3 regimens (table 4). All are appropriate for nonpregnant patients who are not allergic to penicillin. There is limited, indirect evidence that first- and second-generation cephalosporins are effective in eradicating pharyngeal colonization with GAS [31, 32]. Therefore, these agents could be considered for patients allergic to penicillin whose allergic reactions are not anaphylactic [33]. All persons who receive chemoprophylaxis should watch for signs and symptoms of invasive GAS disease for 30 days after the diagnosis of invasive disease in the household contact.

Table 4. Recommended regimens for chemoprophylaxis against group A streptococcal infection.

Drug	Dosage(s)	Comment(s)
BPG plus rifampin	BPG: 600,000 U im in 1 dose for patients weighing <27 kg or 1,200,000 U im in 1 dose for patients weighing ≥27 kg; rifampin: 20 mg/kg/day po (max. daily dose, 600 mg) in 2 divided doses for 4 days	Not recommended for pregnant women because rifampin is teratogenic in laboratory animals. Because the reliability of oral contraceptives may be affected by rifampin therapy, alternative contraceptive measures should be considered while rifampin is being administered.
Clindamycin	20 mg/kg/day po (max. daily dose, 900 mg) in 3 divided doses for 10 days	Preferred for health care workers who are rectal carriers of GAS ^a
Azithromycin	12 mg/kg/day po (max. daily dose, 500 mg/day) in a single dose for 5 days	Pregnancy category B: human data reassuring (animal positive) or animal studies show no risk ^a

NOTE. All regimens are acceptable for nonpregnant persons who are not allergic to penicillin. BPG, benzathine penicillin G; max., maximum.

^a Pregnancy category as defined in [30, p. 344]. Clindamycin or azithromycin is acceptable for persons allergic to penicillin. If administered to health care workers implicated in an outbreak or to their colonized household contacts, susceptibility testing should be performed.

Although penicillin resistance in GAS has never been described [34], clindamycin resistance occurs rarely. The prevalence of macrolide resistance among invasive strains of GAS varies regionally [35] but remains <8% in most areas [36, 37]. If available, antibiotic susceptibility data should be used to select the most appropriate chemoprophylactic agent.

PREVENTION OF POSTPARTUM AND POSTSURGICAL INVASIVE GAS INFECTIONS

Epidemiological Features of Postpartum and Postsurgical Invasive GAS Infections

Burden of infection. A recent study has estimated that ~220 cases of postpartum invasive GAS disease occur annually in the United States (0.06 cases/1000 live births) [38]. These data likely underestimate the true incidence, because most cases are not invasive [39], and microbiological diagnoses of noninvasive postpartum infections are frequently not available. For example, although some experts recommend that samples of endometrial contents be obtained and cultured during the postpartum period for women suspected of having endomyometritis [40], neither blood nor endometrium cultures are done routinely.

In 2000, 1.5% of cases of invasive GAS disease detected by the CDC's Active Bacterial Core surveillance (ABCs) program were classified as postsurgical cases (i.e., invasive GAS infections that occurred during the first 7 days after surgery). On the basis of 1999 national census data, an estimated 135 postsurgical cases of invasive GAS disease occur in the United States annually (CDC, unpublished data). This estimate is conservative, because GAS infections do occur >7 days after surgery [41, 42].

Descriptive features of investigations of infection clusters. To understand the epidemiology of outbreaks of postpartum and postsurgical GAS infection, workshop participants reviewed reports of postpartum and postsurgical GAS infection clusters published during 1990–1999 (table 5) [42–47]. Case

definitions required isolation of GAS from case patients but not necessarily from sterile sites. Microbiology laboratories were the primary resource for identification of cases, but other sources included employee records that listed persons who provided preoperative and postoperative care [44] and operating room and medical records [42, 44, 45, 47].

In 6 [42–45, 47] of 8 investigations, an asymptomatic health care worker (HCW) carried the same strain of GAS as did the case patients. Each cluster of infections subsided after effective treatment of the HCW who was the carrier. In most reports, a focused epidemiological investigation limited screening for GAS carriage to HCWs who had contact with case patients. Among epidemiologically linked asymptomatic HCWs, outbreak strains of GAS have been isolated from the throat, the anus, the vagina, and skin lesions [41–45, 47, 48] (table 5).

In some instances, household contacts of epidemiologically linked, asymptomatic, colonized HCWs have been shown to be asymptomatic carriers of outbreak strains of GAS [42, 45, 48]. In at least 1 of these investigations, a household contact of the HCW may have served as the reservoir for GAS that led to the outbreak [42]. These carriers may play a role in recolonizing treated HCWs [45, 48].

Recommendations for the Prevention of Postpartum and Postsurgical Invasive GAS Disease

Case definitions. A postpartum case of invasive GAS is defined as isolation, during the postpartum period, of GAS in association with a clinical postpartum infection (e.g., endometritis) or from either a sterile site or a wound infection. To increase the likelihood of identifying nosocomial cases of postpartum GAS infection, the postpartum period of interest includes all inpatient days and the first 7 days after discharge. A case of postsurgical GAS infection is defined as isolation, during the hospital stay or the first 7 days after discharge, of GAS from a sterile site or a surgical wound in a postsurgical patient for whom the indication for surgery was not a preexisting GAS

Table 5. Descriptive features of published investigations of clusters of postpartum and postsurgical group A streptococcal infections, 1990–1999.

Type of infections in cluster [reference]	Cases identified, no.		Duration of cluster	Link to ≥ 1 HCW ^b	HCWs for whom cultures performed, no.	Sites with samples cultured (positive culture result) ^c
	Threshold for investigation ^a	Total				
Postpartum [41]	3	9	2 months	Yes	2	A (+), N, P, T
Postpartum [41]	4	4	2 days	Yes	1	A (+)
Postpartum [42]	7	9	12 months	Yes	198	A (+), S, T, V
Postsurgical [40]	4	4	1 month	Yes	16	A, N, T (+)
Postsurgical [44]	4	4	11 days	No	110	A, P, S, T
Postsurgical [45]	3	3	3 months	Yes	1	T (+)
Postsurgical [42]	2	3	7 days	Yes	41	A, P, T, V
Postsurgical [43]	17	20	39 months	No	109	A, S (+), T, V

NOTE. A, anus; HCW, health care worker; N, nose; P, perineum; S, skin; T, throat; V, vagina.

^a No. of cases identified that led to the investigation.

^b Epidemiological link to ≥ 1 HCW was identified before samples were obtained from HCWs for culture.

^c Culture result indicates site from which a cultured sample grew the outbreak strain of group A *Streptococcus*.

infection. The incubation period of severe GAS infections is usually short (1–3 days [49]), and, therefore, cases that occur >7 days after discharge are more likely to be of community origin. In states where invasive GAS infection is reportable, all cases of invasive postpartum and postsurgical GAS disease should be reported to the local or state health department [50].

Strategies for the investigation of a single case of infection. Given the potential for prevention of additional cases, even 1 case of postpartum or postsurgical GAS infection should prompt an epidemiological investigation by the hospital's infection control personnel (figure 1), which should include enhanced surveillance and storage of GAS isolates from the index patient and any additional cases.

Enhanced surveillance should include one or both of the following: (1) review of microbiology records and autopsy reports from the previous 6 months and/or (2) review of operative, labor and delivery, and medical records from within the hospital. To improve the identification of cases, obstetricians and surgeons should be encouraged, during the ensuing months, to perform appropriate pretreatment cultures for patients suspected of having postpartum or postsurgical infections. Isolates from the index case and any additional cases should be stored for at least 6 months to allow comparison of strains isolated at different times. If laboratory resources are available to type GAS isolates, some infection control practitioners might choose to screen HCWs after the occurrence of a single case of postpartum or postsurgical GAS disease. However, screening of HCWs is not a substitute for enhanced disease surveillance. If infection-control personnel choose to screen HCWs, screening should be considered for HCWs who were present at delivery and for those who performed vaginal examinations before delivery (for postpartum cases) and for all HCWs present in the operating room during surgery and those

who changed dressings on open wounds (for postsurgical cases).

If screening of HCWs is undertaken, sites from which specimens should be obtained and cultured include the throat, anus, vagina, and any skin lesions [45]. Screened HCWs may return to work pending the culture results. However, HCWs identified as colonized should be suspended from patient care duties until they have received chemoprophylaxis for 24 h [51].

Strategies for the investigation of ≥ 2 cases of infection. If ≥ 2 cases are identified within a 6-month period, they may have a common source of GAS transmission. Isolates should be compared using PFGE [52], serotyping, *emm* typing [53], or other molecular methods. Isolates that differ probably indicate a community source rather than a common source in an HCW [46]. Enhanced surveillance should be initiated, regardless of whether the strains are identical. Identification of 2 cases caused by identical strains should lead to enhanced surveillance and to an investigation of possible epidemiological links between cases.

If 2 cases are found to be caused by the same strain within a 6-month period, screening of HCWs is strongly recommended to prevent further cases of serious infection. For all HCWs epidemiologically linked to the case patients, specimens from the anus, skin lesions, throat, and vagina should be cultured. If no colonized HCW is identified or if HCWs are colonized with strains unrelated to the outbreak strain, the search for colonized HCWs should be broadened to include those HCWs without immediate epidemiological links to all case patients. This might include, for example, HCWs who had direct contact with most but not all of the case patients [42]. The use of standard precautions for infection control should supplement any investigation of postpartum or postsurgical GAS infections.

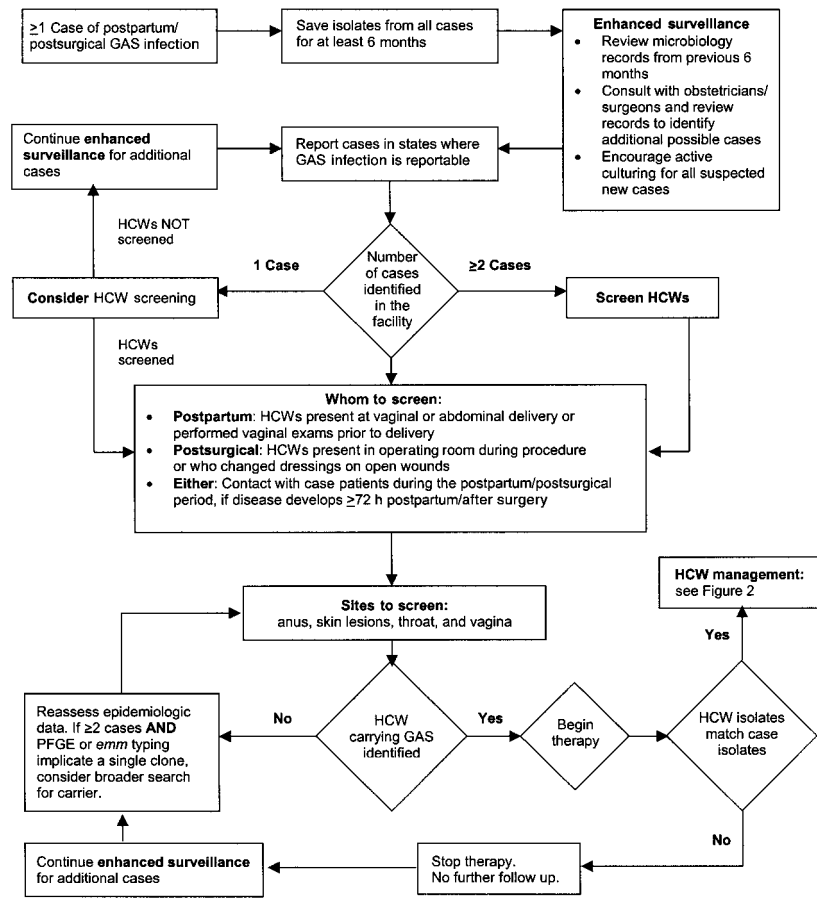


Figure 1. Recommended public health response to cases of postpartum and postsurgical group A streptococcal (GAS) disease. HCW, health care worker.

Management of epidemiologically linked, colonized HCWs. Because most HCWs associated with a given outbreak will not be colonized, HCWs may return to work pending culture results (figure 2). However, colonized HCWs should be suspended from patient care for the first 24 h that they receive chemoprophylaxis [51], and HCW strains should be compared with patient strains by use of the same typing method(s). If an HCW is epidemiologically linked to the case patients and the strain the HCW is carrying is the same as the strains isolated from patients, follow-up cultures should be done for the HCW 7–10 days after the completion of therapy.

In contrast to the recommendations for household contacts of persons with invasive GAS disease, it is recommended that HCWs included in investigations of postpartum or postsurgical GAS infection clusters have cultures performed if results of follow-up cultures from the implicated HCW remain positive 7–10 days after the completion of therapy [42, 45, 48]. For children who are household contacts of the HCW, cultures of specimens from the throat and skin lesions should be performed. For adult household contacts, cultures of specimens

from the throat, any skin lesions, the anus, and the vagina should be performed. Colonized household contacts of an HCW implicated in an outbreak should receive chemoprophylaxis.

Treatment of epidemiologically linked HCWs carrying outbreak strains. Treatment options for asymptomatic colonized HCWs have not been rigorously studied. Previous studies [13, 18–20] have suggested that 3 regimens may be effective (table 4). Any of these regimens is appropriate for nonpregnant HCWs who are not allergic to penicillin and for their colonized household contacts. Clindamycin or azithromycin is recommended for HCWs and colonized household contacts who are allergic to penicillin. Rectal carriage of GAS is difficult to eradicate with penicillin-based regimens [43, 48, 54–56]. Oral therapy with vancomycin in combination with rifampin has been recommended in such cases [57, 58]; however, no controlled trials have been done to support this recommendation. Given the well-documented effects of clindamycin on bowel flora, oral clindamycin is recommended for the treatment of HCWs and their household contacts who have rectal carriage of GAS. If

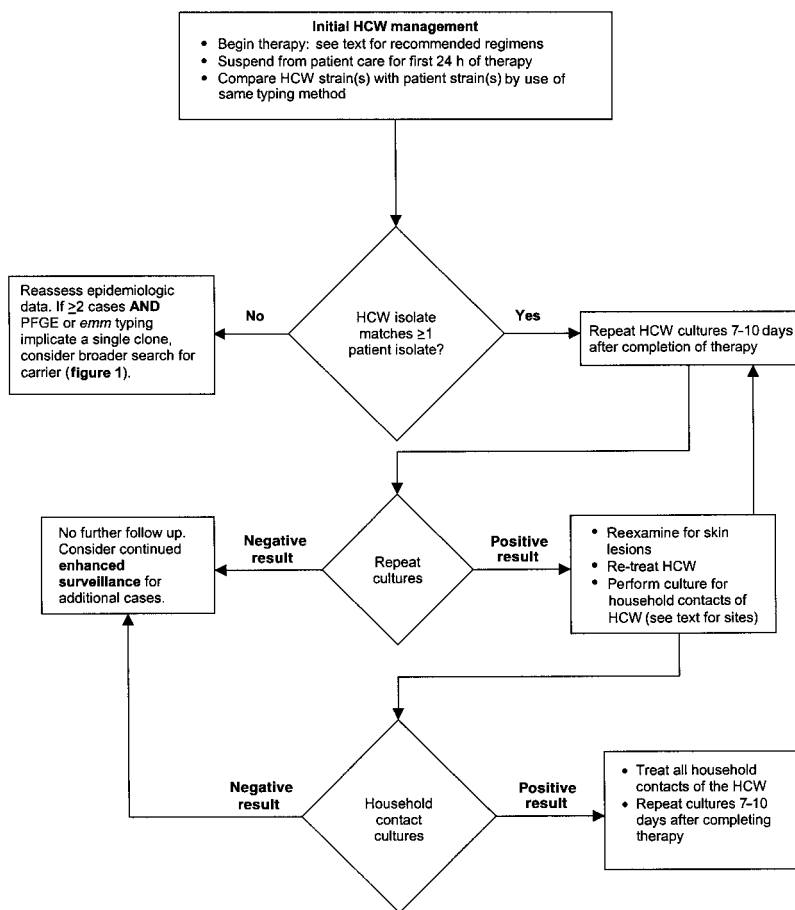


Figure 2. Recommended management for health care workers (HCWs) colonized with group A *Streptococcus*

azithromycin or clindamycin is used, susceptibility testing of the HCW strain of GAS against macrolides and clindamycin should be performed.

SUMMARY

Although the risk of subsequent invasive GAS disease among household contacts of persons with invasive GAS infections is higher than the risk among the general population, subsequent invasive GAS infections are rare. As a result, routine screening for colonization with GAS and routine administration of chemoprophylaxis against GAS are not recommended for household contacts of index patients. However, because of the increased risk of sporadic invasive GAS disease among certain groups (table 2) and the risk of death among persons aged ≥ 65 years who develop invasive GAS disease, health care providers may choose to offer chemoprophylaxis to members of the households of patients with invasive GAS infection that include persons aged ≥ 65 years or other high-risk individuals (table 4). Performance of cultures is not recommended for potential GAS carriers.

Postpartum and postsurgical GAS infections frequently are undetected and are underreported, and some of these cases present opportunities for prevention. Enhanced surveillance should be implemented after identification of a single case of postpartum or postsurgical GAS infection, and all GAS isolates from suspected cases should be stored and compared by serotyping or molecular techniques. Some infection control practitioners might choose to screen HCWs after a single case has occurred, but this should not interfere with the conduct of enhanced surveillance. The occurrence of ≥ 2 cases of infection with the same GAS type within a 6-month period suggests that an HCW might be the source of the cluster; therefore, screening of HCWs who are epidemiologically linked to the case patients is strongly recommended (figures 1 and 2).

It is important to indicate the settings that are not included in the present recommendations. Invasive GAS infections and clusters of noninvasive infections (e.g., pharyngitis or uncomplicated cellulitis) that occur in day-care centers, schools, military training facilities, and nursing homes present unique challenges. These recommendations do not apply to those settings.

The workshop participants identified several research issues

that deserve attention. These include antibiotic treatment of GAS carriage in adults, the prevalence of macrolide and clindamycin resistance among isolates of GAS, and the effectiveness of antimicrobial agents for the eradication of rectal carriage of GAS. Finally, because subsequent GAS disease accounts for <1% of all invasive cases and also because preventable postpartum and postsurgical infections constitute a small portion of disease burden, primary prevention of GAS infections remains a research priority. The development of GAS vaccines may offer the ultimate solution to this problem.

THE PREVENTION OF INVASIVE GROUP A STREPTOCOCCAL INFECTIONS WORKSHOP PARTICIPANTS

The participants in the workshop were as follows, in alphabetical order: Bernard Beall (Respiratory Diseases Branch, Division of Bacterial and Mycotic Diseases, Centers for Disease Control and Prevention [CDC], Atlanta, Georgia), John Besser (Minnesota Department of Health, Minneapolis), Alan Bisno (Miami Veteran's Affairs [VA] Medical Center and University of Miami School of Medicine, FL), Ilin Chuang (Respiratory Diseases Branch, Division of Bacterial and Mycotic Diseases, CDC), Allen S. Craig (Tennessee Department of Health, Nashville; representative, Active Bacterial Core Surveillance program), Richard Facklam (Respiratory Diseases Branch, Division of Bacterial and Mycotic Diseases, CDC); Janice Fetter (Northside Hospital, Atlanta, GA; representative, Association for Professionals in Infection Control and Epidemiology), Michael A. Gerber (Children's Hospital Medical Center, Cincinnati, OH; representative, American Academy of Pediatrics), Gregory Gray (University of Iowa College of Public Health, Iowa City; representative, US Navy [retired]), Harry Hill (University of Utah School of Medicine, Salt Lake City), Lisa Lepine (Emory University School of Medicine, Atlanta, GA), Orin Levine (National Institute for Allergy and Infectious Diseases, Bethesda, MD), Allison McGeer (Mt. Sinai Hospital, and Laboratory Medicine and Public Health Sciences, University of Toronto, ON, Canada), Matthew Moore (Epidemic Intelligence Service Program, Division of Applied Public Health Training, Epidemiology Program Office, and Respiratory Diseases Branch, Division of Bacterial and Mycotic Diseases, CDC), Michele Pearson (Division of Healthcare Quality Promotion, CDC), Katherine O'Brien (Johns Hopkins University Bloomberg School of Public Health, Baltimore, MD), Anne Schuchat (Respiratory Diseases Branch, Division of Bacterial and Mycotic Diseases, CDC), Mack Sewell (New Mexico Department of Health, Santa Fe; representative, Council of State and Territorial Epidemiologists), Stanford Shulman (Children's Memorial Hospital, and Department of Pediatrics, Northwestern University, Chicago, Illinois), Jane Siegel (University of Texas Southwestern Medical Center, Dal-

las, TX; representative, Healthcare Infection Control Practices Advisory Committee), Dennis L. Stevens (Boise VA Medical Center, ID; and University of Washington School of Medicine, Seattle), Larry Strausbaugh (Portland VA Medical Center and Oregon Health and Sciences University School of Medicine, Portland; representative, Infectious Diseases Society of America), and Chris Van Beneden (Respiratory Diseases Branch, Division of Bacterial and Mycotic Diseases, CDC).

Acknowledgments

We gratefully acknowledge the contributions of Katherine Robinson.

References

1. Centers for Disease Control and Prevention. Active bacterial core surveillance (ABCs) report: group A *Streptococcus*, 2000. Available at: <http://www.cdc.gov/ncidod/dbmd/abcs>. Accessed 23 May 2002.
2. The Working Group on Severe Streptococcal Infections. Defining the group A streptococcal toxic shock syndrome. Rationale and consensus definition. *JAMA* 1993;269:390-1.
3. Stevens DL, Tanner MH, Winship J, et al. Severe group A streptococcal infections associated with a toxic shock-like syndrome and scarlet fever toxin A. *N Engl J Med* 1989;321:1-7.
4. Centers for Disease Control and Prevention. Case definitions for infectious conditions under public health surveillance. *MMWR Recomm Rep* 1997;46(RR-10):1-55.
5. O'Brien KL, Beall B, Barrett NL, et al. Epidemiology of invasive group A *Streptococcus* disease in the United States, 1995-1999. *Clin Infect Dis* 2002;35:268-76.
6. Davies HD, McGeer A, Schwartz B, et al. Invasive group A streptococcal infections in Ontario, Canada. Ontario Group A Streptococcal Study Group. *N Engl J Med* 1996;335:547-54.
7. Zurawski CA, Bardsley M, Beall B, et al. Invasive group A streptococcal disease in metropolitan Atlanta: a population-based assessment. *Clin Infect Dis* 1998;27:150-7.
8. Quintiliani R, Engh GA. Overwhelming sepsis associated with group A β hemolytic streptococci. *J Bone Joint Surg Am* 1971;53:1391-9.
9. Aitken DR, Mackett MC, Smith LL. The changing pattern of hemolytic streptococcal gangrene. *Arch Surg* 1982;117:561-7.
10. Centers for Disease Control and Prevention. Group A β -hemolytic streptococcal bacteremia—Colorado, 1989. *MMWR Morb Mortal Wkly Rep* 1990;39:3-6, 11.
11. Thomas JC, Carr SJ, Fujioka K, Waterman SH. Community-acquired group A streptococcal deaths in Los Angeles County. *J Infect Dis* 1989;160:1086-7.
12. Centers for Disease Control and Prevention. Nursing home outbreaks of invasive group A streptococcal infections—Illinois, Kansas, North Carolina, and Texas. *MMWR Morb Mortal Wkly Rep* 1990;39:577-9.
13. Morita JY, Kahn E, Thompson T, et al. Impact of azithromycin on oropharyngeal carriage of group A streptococcus and nasopharyngeal carriage of macrolide-resistant *Streptococcus pneumoniae*. *Pediatr Infect Dis J* 2000;19:41-6.
14. The Working Group on Prevention of Invasive Group A Streptococcal Infections. Prevention of invasive group A streptococcal disease among household contacts of case-patients: is prophylaxis warranted? *JAMA* 1998;279:1206-10.
15. Weiss K, Laverdiere M, Lovgren M, Delorme J, Poirier L, Beliveau C. Group A streptococcus carriage among close contacts of patients with invasive infections. *Am J Epidemiol* 1999;149:863-8.

16. World Health Organization (WHO). Overcoming antimicrobial resistance. Geneva: WHO, 2000.
17. Hoge CW, Schwartz B, Talkington DF, Breiman RF, MacNeill EM, Engler SJ. The changing epidemiology of invasive group A streptococcal infections and the emergence of streptococcal toxic shock-like syndrome: a retrospective population-based study. *JAMA* 1993;269:384-9 (erratum: *JAMA* 1993;269:1638).
18. Tanz RR, Shulman ST, Barthel MJ, Willert C, Yogev R. Penicillin plus rifampin eradicates pharyngeal carriage of group A streptococci. *J Pediatr* 1985;106:876-80.
19. Tanz RR, Poncher JR, Corydon KE, Kabat K, Yogev R, Shulman ST. Clindamycin treatment of chronic pharyngeal carriage of group A streptococci. *J Pediatr* 1991;119:123-8.
20. Orrling A, Stjernquist-Desatnik A, Schalen C, Kamme C. Clindamycin in persisting streptococcal pharyngotonsillitis after penicillin treatment. *Scand J Infect Dis* 1994;26:535-41.
21. Gray GC, McPhate DC, Leinonen M, et al. Weekly oral azithromycin as prophylaxis for agents causing acute respiratory disease. *Clin Infect Dis* 1998;26:103-10.
22. Gastanaduy AS, Kaplan EL, Huwe BB, McKay C, Wannamaker LW. Failure of penicillin to eradicate group A streptococci during an outbreak of pharyngitis. *Lancet* 1980;2:498-502.
23. Schwartz B. Chemoprophylaxis for bacterial infections: principles of and application to meningococcal infections. *Rev Infect Dis* 1991;13(Suppl 2):S170-3.
24. Huang YC, Hsueh PR, Lin TY, Yan DC, Hsia SH. A family cluster of streptococcal toxic shock syndrome in children: clinical implication and epidemiological investigation. *Pediatrics* 2001;107:1181-3.
25. Weiss K, Roger M, Maziade P, et al. A household cluster of fulminant group A streptococcus pneumonia associated with toxic shock syndrome. Ottawa, Quebec, Canada: Health Protection Branch, Laboratory Centre for Disease Control, 1996:41-3.
26. Schwartz B, Elliott JA, Butler JC, et al. Clusters of invasive group A streptococcal infections in family, hospital, and nursing home settings. *Clin Infect Dis* 1992;15:277-84.
27. Gamba MA, Martinelli M, Schaad HJ, et al. Familial transmission of a serious disease-producing group A streptococcus clone: case reports and review. *Clin Infect Dis* 1997;24:1118-21.
28. DiPersio JR, File TM Jr, Stevens DL, Gardner WG, Petropoulos G, Dinsa K. Spread of serious disease-producing M3 clones of group A streptococcus among family members and health care workers. *Clin Infect Dis* 1996;22:490-5.
29. Demers B, Simor AE, Vellend H, et al. Severe invasive group A streptococcal infections in Ontario, Canada: 1987-1991. *Clin Infect Dis* 1993;16:792-800, 801-2.
30. Physician's desk reference. 55th ed. Montvale, NJ: Medical Economics Company, 2001.
31. Gerber MA, Tanz RR, Kabat W, et al. Potential mechanisms for failure to eradicate group A streptococci from the pharynx. *Pediatrics* 1999;104:911-7.
32. Shulman ST, Gerber MA, Tanz RR, Markowitz M. Streptococcal pharyngitis: the case for penicillin therapy. *Pediatr Infect Dis J* 1994;13:1-7.
33. Anne S, Reisman RE. Risk of administering cephalosporin antibiotics to patients with histories of penicillin allergy. *Ann Allergy Asthma Immunol* 1995;74:167-70.
34. Macris MH, Hartman N, Murray B, et al. Studies of the continuing susceptibility of group A streptococcal strains to penicillin during eight decades. *Pediatr Infect Dis J* 1998;17:377-81.
35. York MK, Gibbs L, Perdreau-Remington F, Brooks GF. Characterization of antimicrobial resistance in *Streptococcus pyogenes* isolates from the San Francisco Bay area of northern California. *J Clin Microbiol* 1999;37:1727-31.
36. Kaplan EL, Johnson DR, Del Rosario MC, Horn DL. Susceptibility of group A β -hemolytic streptococci to thirteen antibiotics: examination of 301 strains isolated in the United States between 1994 and 1997. *Pediatr Infect Dis J* 1999;18:1069-72.
37. Van Beneden CA, Facklam R, Lynfield R, et al. Erythromycin resistance among invasive group A streptococcal infections in the United States, 1999 [abstract 13]. In: Program and abstracts of the International Conference on Emerging Infectious Diseases. Atlanta, GA: Centers for Disease Control and Prevention, 2002:131.
38. Chuang I, Van Beneden C, Beall B, Schuchat A. Population-based surveillance for postpartum invasive group A streptococcus infections, 1995-2000. Active Bacterial Core Surveillance/Emerging Infections Program Network. *Clin Infect Dis* 2002;35:665-70.
39. Claesson BE, Claesson UL. An outbreak of endometritis in a maternity unit caused by spread of group A streptococci from a showerhead. *J Hosp Infect* 1985;6:304-11.
40. Faro S, Soper D, eds. Infectious diseases in women. 1st ed. Philadelphia: Saunders, 2001:181-93.
41. Stamm WE, Feeley JC, Facklam RR. Wound infections due to group A streptococcus traced to a vaginal carrier. *J Infect Dis* 1978;138:287-92.
42. Paul SM, Genese C, Spitalny K. Postoperative group A β -hemolytic streptococcus outbreak with the pathogen traced to a member of a healthcare worker's household. *Infect Control Hosp Epidemiol* 1990;11:643-6.
43. Viglionese A, Nottbart VF, Bodman HA, Platt R. Recurrent group A streptococcal carriage in a health care worker associated with widely separated nosocomial outbreaks. *Am J Med* 1991;91:329S-33S.
44. Centers for Disease Control and Prevention. Nosocomial group A streptococcal infections associated with asymptomatic health-care workers—Maryland and California, 1997. *MMWR Morb Mortal Wkly Rep* 1999;48:163-5.
45. Mastro TD, Farley TA, Elliott JA, et al. An outbreak of surgical-wound infections due to group A streptococcus carried on the scalp. *N Engl J Med* 1990;323:968-72.
46. Jamieson FB, Green K, Low DE, et al. A cluster of surgical wound infections due to unrelated strains of group A streptococci. *Infect Control Hosp Epidemiol* 1993;14:265-7.
47. Kolmos HJ, Svendsen RN, Nielsen SV. The surgical team as a source of postoperative wound infections caused by *Streptococcus pyogenes*. *J Hosp Infect* 1997;35:207-14.
48. Berkelman RL, Martin D, Graham DR, et al. Streptococcal wound infections caused by a vaginal carrier. *JAMA* 1982;247:2680-2.
49. Chin J, ed.. Control of communicable diseases manual. Washington, DC: American Public Health Association, 2000:470-6.
50. Roush S, Birkhead G, Koo D, Cobb A, Fleming D. Mandatory reporting of diseases and conditions by health care professionals and laboratories. *JAMA* 1999;282:164-70.
51. Snellman LW, Stang HJ, Stang JM, Johnson DR, Kaplan EL. Duration of positive throat cultures for group A streptococci after initiation of antibiotic therapy. *Pediatrics* 1993;91:1166-70.
52. Centers for Disease Control and Prevention. Use of pulsed-field gel electrophoresis for investigation of a cluster of invasive group A streptococcal illness—Spokane, Washington, 1999. *MMWR Morb Mortal Wkly Rep* 1999;48:681-3.
53. Facklam R, Beall B, Efstratiou A, et al. *emm* Typing and validation of provisional M types for group A streptococci. *Emerg Infect Dis* 1999;5:247-53.
54. Kokx NP, Comstock JA, Facklam RR. Streptococcal perianal disease in children. *Pediatrics* 1987;80:659-63.
55. Krol AL. Perianal streptococcal dermatitis. *Pediatr Dermatol* 1990;7:97-100.
56. Rehder PA, Eliezer ET, Lane AT. Perianal cellulitis. Cutaneous group A streptococcal disease. *Arch Dermatol* 1988;124:702-4.
57. McKee WM, Di Caprio JM, Roberts CE Jr, Sherris JC. Anal carriage as the probable source of a streptococcal epidemic. *Lancet* 1966;2:1007-9.
58. Schaffner W, Lefkowitz LB Jr, Goodman JS, Koenig MG. Hospital outbreak of infections with group A streptococci traced to an asymptomatic anal carrier. *N Engl J Med* 1969;280:1224-5.

Three errors appeared in a major article published in the 15 October 2002 issue of the journal (The Prevention of Invasive Group A Streptococcal Infections Workshop Participants. Prevention of Invasive Group A Streptococcal Disease among Household Contacts of Case Patients and among Postpartum and Postsurgical Patients: Recommendations from the Centers for Disease Control and Prevention. *Clin Infect Dis* 2002;35:950–9). First, in the “Risk of subsequent invasive GAS disease” sub-subsection of the Prevention of Subsequent Invasive Gas Infections among Household Contacts of Persons with Invasive Gas Disease section, the beginning of the last sentence of paragraph 2 should have appeared as “In summary, 2 prospective studies that were designed to identify subsequent cases among household contacts in a population observed for a total of 66.5 million person-years...” [not “In summary, 2 prospective studies that were designed to identify subsequent cases among

household contacts (who were observed for a total of 66.5 million person-years)...”]. Second, the 8 reference citations presented in column 1 of table 5 should have been [43], [43], [44], [42], [46], [47], [44], and [45] (not [41], [41], [42], [40], [44], [45], [42], and [43]). The references as cited in the text are correct. Third, in the “Management of epidemiologically linked, colonized HCWs” sub-subsection of the Prevention of Postpartum and Postsurgical Invasive Gas Infections section, the beginning of the first sentence of paragraph 2 should have appeared as “In contrast to the recommendations for household contacts of persons with invasive GAS disease, it is recommended that household contacts of HCWs...” [not “In contrast to the recommendations for household contacts of persons with invasive GAS disease, it is recommended that HCWs...”]. The authors and the journal regret these errors.