

Group B Streptococcus (GBS) are bacteria that live inside (or colonise) the gastrointestinal tract, bladder and / or throat of many people, including pregnant and labouring women. Estimates of the prevalence of colonisation range from 5% to 80% of the population (which shows, if nothing else, the difficulty with have with deducing some of these numbers). GBS is generally benign in adults, only posing a danger to people with compromised immune systems. It is also benign in most of the babies who pick up these bacteria during their birth, but can occasionally cause serious harm to others. Because of this, pregnant women may be offered screening and intervention ~ yet another pond of uncertainty to have to swim in, which is beset with complex numbers, difficult decisions and tiny chances of very serious conditions.

For several years, most areas in the UK have taken a “risk-based” approach to GBS screening, where practitioners attempt to identify the babies who are at increased risk of becoming infected with GBS. Risk factors (which vary between hospitals) include premature labour (before 37 weeks of pregnancy), a woman with a high temperature during labour or a woman whose waters have been broken for 18-24 hours (1). Where such a risk factor is identified, the woman will be offered intravenous antibiotics in labour. In these situations, swabs may be taken to see if the woman has GBS (or other bacteria), but it takes a while to perform the testing, so the results are usually only available after the birth. As a consequence of this, the woman is making a decision based on the possibility that, IF she has GBS, her baby might be more at risk of becoming infected, rather than knowing (from screening tests) THAT she has GBS.

By contrast, pregnant women in the USA are offered routine screening for GBS at the end of pregnancy (2), and are offered intravenous antibiotics in labour if they are found to have GBS in their vagina or rectum. Some UK practitioners have mooted the idea of replacing the current “risk-based approach” with the approach used in the US, termed “culture-based screening” (3). With culture-

based screening, some of the women who have risk factors but not GBS may escape having unnecessary antibiotics (although some practitioners may recommend them anyway, in case other pathogens are present). However, hospitals will instead offer antibiotics to all of the 10-30% of women whose vaginas and rectums are colonised with GBS (4), even though only a tiny proportion of these babies will be affected with GBS disease (5,6).

GBS disease itself comes in two forms; early onset and late onset. Early onset GBS disease occurs within the first week of life; three-quarters of babies who develop GBS disease will do so at this stage. Problems usually become apparent within a few hours of birth and can include generalised infection (sepsis), pneumonia or meningitis. GBS disease is termed “late onset” when it occurs between one week and a few months of age. Not all cases of late onset will occur because the baby’s mother transmitted GBS during birth; some will occur from other (but usually unknown) sources. The impact of late-onset disease is usually less severe. Babies found to have GBS disease are treated with antibiotics and given whatever other support they need in Special Care Units.

There are large discrepancies between the findings of different research studies as far as the outcomes of babies who contract GBS are concerned. It is probably fair to say that, currently, researchers are more concerned with how to prevent GBS disease than what the prognosis of this is in the babies who do contract this. In 2002, researchers (7) published an analysis of two years’ worth of data from births in Northern England to look at a number of aspects of GBS. They found that:

- ❖ The prevalence of early onset group B streptococcal sepsis was 0.57 per 1000 live births. Put another way, 1 in every 1754 women had a baby with GBS disease.
- ❖ Premature babies accounted for 38% of all cases of GBS disease and 83% of

the deaths from GBS disease during the time of the study.

- ❖ Of the 39 (out of 62, 786) babies who developed GBS disease, three were stillborn and six died after birth. Five of the six babies who died after birth were born prematurely (before 36 weeks of pregnancy).
- ❖ Four of the mothers of the babies who contracted GBS disease had been given antibiotics in labour.

We can then say, in this study, that around one in four babies who were known to have GBS died as a result. In reality, the mortality (death) rate from GBS may be lower than this, as some babies may have had GBS disease and recovered without this having been diagnosed. Indeed, the Centres for Disease Control quote a US mortality rate during the 1990s of 4 per cent (3). It is difficult to know which of these figures is the more accurate; the real figure may be somewhere between the two and, as with many things, will be partially dependent on local expertise and technology.

Another interesting finding of the study of GBS in Northern England was that, had they used risk-based screening, they would have identified 78% of the babies who developed GBS disease. (They would still have missed 22%, which is one reason that some people are calling for culture-based screening). However, they calculate that the administration of antibiotics according to the results of risk-based screening would have meant that 16% of all women in labour were taking antibiotics. Sixteen percent of 62, 786 equates to 10,046 women who would have had antibiotics, in order to attempt to prevent the deaths of nine babies. (And let's not forget that four of the mothers of the babies who had GBS disease did have antibiotics). In other words, 1116 of the women *who have risk factors* in this study would have needed to take antibiotics in labour to prevent one baby dying from GBS ~ but without a solid guarantee that this hypothetical baby would be saved.

As above, culture based screening would identify the 10 to 30% of pregnant women whose vaginas or rectums are colonised with GBS (4). The screening test involves taking swabs of the inside of a woman's vagina and rectum ~ not a particularly pleasant procedure, but not as invasive as some. Yet only 1 or 2 in every thousand of the women who have a positive result if we screen this way will have a baby who ends up with GBS disease (5,6). Even taking the most conservative estimate (assuming 2 in a thousand women with GBS have a baby with GBS disease and using a mortality rate of 25%), this would mean that 2000 women who tested positive for GBS would need antibiotics in labour to prevent the death of one baby.

There are a number of other factors which women may want to take into account here. According to the CDC (3), your baby is at highest risk of contracting GBS disease if you test positive and also have any of the following conditions:

- Previous baby with GBS disease
- Urinary tract infection due to GBS
- Fever during labour
- Rupture of membranes 18 hours or more before delivery
- Labour or rupture of membranes before 37 weeks

It may be that, while policymakers are debating which of the two approaches to use, women might be better served by research which looked at the outcomes where the two were combined. Even this means that many more women would have antibiotics than needed them, but at least we are getting a bit more specific for the women who would prefer to take them if they were at risk. Perhaps we could also see whether there are other factors that could help us be even more specific about who is at risk. While this might not be deemed cost-effective on a population basis, it may be more helpful for the women who want to avoid unnecessary intervention.

There are, inevitably, a number of reasons why women may not want antibiotics in labour

unless they are truly necessary. Apart from the side effects, and the discomfort of having movement hindered by an intravenous cannula in labour, there are more serious ramifications of policies advocating mass antibiotic cover. While penicillin (8) and ampicillin (9) are currently effective for treating GBS disease in babies, since antibiotics have been used to treat large numbers of women whose babies are deemed at risk of GBS disease (whether this is on a risk-based or culture-based policy), the rate of *Escherichia coli* infections in premature babies has more than doubled. Around 85 percent of the *E. coli* infections in one study were resistant to the drugs prescribed to treat GBS (10).

There is a huge debate about antibiotic resistant bacteria generally, and these policies involve giving antibiotics to a lot of women, which may have ramifications for the population as a whole. It has also been suggested that giving antibiotics while babies are still in their mother's uterus might delay the baby's gut being colonised with normal, "good" bacteria and allow dangerous penicillin-resistant bacteria to become established there instead (11).

There is also a need to find out whether giving antibiotics actually makes a difference to the outcome. The assumption that this is the case has long been just that ~ an assumption. Cochrane reviewers (12) who looked at the trials comparing women who had been given antibiotics with women who had not been given antibiotics found that, although antibiotics reduced the incidence of GBS infection in babies, there was no significant difference in the numbers of babies who died. They found the few trials that had looked at this area to be of poor quality, and called for further research ~ something which surely needs to be done before even more women receive unnecessary drugs in labour. Having said that, there is plenty of research to support the fact that midwifery and medical interventions in labour, such as vaginal examination, can increase the rate of infection (13-17), yet there is no evidence to suggest that hospitals are making attempts to limit

these interventions. Added to the suggestion, from an American review of laboratory procedures (18), that these may not always be effective at detecting GBS in cultures, the decision can become fraught for some women.

Women looking for information about GBS on the Internet are likely to come across some of the most emotive websites in existence. Some are named for babies who died from GBS disease or who continue to suffer from the effects. While I have enormous sympathy for these families, this is only one side of the picture. The other, I hope, can be seen by looking at some of the numbers in this article. The promotion of GBS screening is likely to increase over the next few years, yet the available data show that there is no simple answer to this issue ~ and no way of screening for GBS in babies that doesn't lead to thousands of women having antibiotics they don't need.

It is appealing to want to reduce the rate of GBS infection; it is the commonest cause of infectious disease in babies, and it can be fatal. Yet, as with the cases of rhesus disease and haemorrhagic disease, we are often using sledgehammers to crack nuts, potentially at the expense of our future health. Antibiotics have been a marvellous and life-saving discovery. When used appropriately, they are truly useful to humanity. We are already suffering some consequences of our over-use of antibiotics, which is surely something we need to temper. GBS is increasingly seen as a public health issue. However, one's position on GBS (and many other birth interventions) really depends on two things. It depends on whether you are happy to be gathered together with all of the other Ms. Publics and be told what is best for your health (and that of your children), or whether you want to make the choices that suit you as an individual. And, perhaps more importantly, it depends on how you define health, on whether you are happy to accept the potential costs of medical technology, and how comfortable you are with the very unfashionable idea that nothing in life is certain.

REFERENCES

1. Boyer KM, Gotoff SP. Strategies for chemoprophylaxis of GBS early-onset infections. *Antibiotic Chemotherapy* 1985; 35: 267-80.
2. Schrag SJ, Zell ER, Lynfield R and others. (2002) A Population-Based Comparison of Strategies to Prevent Early-Onset Group B Streptococcal Disease in Neonates. *New England Journal of Medicine*, July 25, 2002. 347: 4, 233-239.
3. Schrag S, Gorwitz R, Fultz-Butts K and others. (2002) Prevention of Perinatal Group B Streptococcal Disease. Revised Guidelines from CDC. August 16, 2002 / 51 (RR11); 1-22.
4. Regan JA, Klebanoff MA, Nugent RP, Vaginal Infections and Prematurity Study Group. The epidemiology of group B streptococcal colonization in pregnancy. *Obstetrics and Gynecology* 1991; 77: 604-10.
5. Gilbert GL, Garland SM. Perinatal group B streptococcal infections. *Medical Journal of Australia* 1983; 1: 566-571.
6. Isaacs D, Royle JA. Intrapartum antibiotics and early onset neonatal sepsis caused by group B Streptococcus and by other organisms in Australia. *Australasian Study Group for Neonatal Infections. Pediatric Infectious Diseases Journal* 1999; 18: 524-528.