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AMOGS UPDATE

VOL. 2

AMOGS President's message

It gives me great pleasure to know that the AMOGS Update newsletter is now ready for release. In these difficult times during the pandemic of COVID-19, all of us need to take the necessary precautions, use personal protective equipment and restrict our practice to the most essential emergency and obstetric cases. The health and safety of ourselves, our staff and our families remains an important responsibility on our shoulders at this time.

Through this AMOGS newsletter, the editors Dr Pratik Tambe, Dr Ameya Purandare and Dr Rohan Palshetkar have chosen to address contemporary important issues in the field of obstetrics. The articles include a synopsis of the recent ACOG Practice Committee Recommendations on prevention of neonatal Group B streptococcal disease and the effects of early vs delayed cord clamping and umbilical cord milking. They will be of interest to all obstetricians in their day-to-day practice.

My Presidential theme for the AMOGS year 2020-22 is “We for Stree – Safer, Stronger, Smarter”. During the year, we will attempt to focus on academic, social and community health initiatives aimed at improving the profile of women in our country. I am pleased to acknowledge the efforts of ordinary members who have united and stood with us to contribute to a series of initiatives which will refocus our contributions not only toward the health of Indian women, but their social, financial and educational upliftment as well.

I am committed to our AMOGS members, delivering continuing medical education programmes and to help our members to perform to the best of their ability in the areas in which they practice. We also plan to execute a raft of initiatives to help women from poor socioeconomic areas receive appropriate care through our social and community healthcare initiatives eg.the Saving Mothers initiative.

I congratulate the editors for their efforts towards bringing out this newsletter and wish them all the very best. We hope you enjoy this AMOGS Update and look forward to your comments and suggestions. If you would like to contribute articles of topical interest for subsequent issues, please feel free to get in touch with us and we look forward to publishing your contributions in the future!

Dr Nandita P. Palshetkar

Dr Nandita Palshetkar
President AMOGS 2020 - 22

Effect of Early cord clamping, Umbilical cord milking and Delayed cord clamping on neonatal outcomes

Dr Pratik Tambe, Dr Rohan Palshetkar

Background

The World Health Organization recommends delayed cord clamping (DCC), defined as a delay of 30 seconds or longer after birth, as standard delivery room care for infants who do not need resuscitation.[1] A delay in cord clamping from 30 to 120 seconds in preterm infants born at less than 37 weeks of gestation reduced the risk of intraventricular haemorrhage (IVH) (all grades), the need for blood transfusion, provided better circulatory stability and lowered the risk for developing necrotising enterocolitis compared to immediate cord clamping (ICC).[2]

Moreover, a recent systematic review and meta-analysis of DCC versus ICC for preterm infants concluded that DCC reduces hospital mortality.[3] However, most preterm neonates require resuscitation immediately after birth, and DCC may impede resuscitation, so the optimum timing of umbilical cord clamping has not been established.

An alternative to DCC is umbilical cord milking (UCM), in which the unclamped umbilical cord is squeezed and blood is massaged toward the preterm infant with the intention of transfusing blood. This is repeated up to four times before the cord is clamped. For infants born at less than 29 weeks of gestation, UCM reduces the need for red blood cell transfusion compared to ICC.[4]

Umbilical Cord Milking Vs Delayed Cord Clamping

A systematic review and meta-analysis by Nagano N et al published in PLOS One looked at two trials with 255 preterm infants (23 0/7 to 32 6/7 weeks' gestation). UCM was associated with fewer intraventricular haemorrhages (IVHs) (two trials, 255 infants; relative risk [RR] 0.45, 95% confidence interval [CI] 0.20 to 0.98) and UCM was associated with an increased proportion of infants with a Bayley score at 2 years of age (two trials, 174 infants; Cognitive: RR 1.14, 95% CI 1.03 to 1.26, Language: RR 1.24, 95% CI 1.03 to 1.49, low quality of evidence) compared to DCC.

No significant difference was observed in Apgar scores at 5 minutes between UCM and DCC (MD -1.00, 95% CI -2.24 to 0.24). UCM was not associated with a lower number of deaths than DCC (RR 0.44, 95% CI 0.14 to 1.34). No significant difference in the haematocrit was observed between UCM and DCC (MD 1.00, 95% CI -2.90 to 4.90). No significant difference was observed between neonatal transfusion rates after UCM and DCC (RR 0.93, 95% CI 0.70 to 1.22). The mean Hb level at birth did not differ significantly between UCM and DCC (MD 0.43, 95% CI -0.10 to 0.9). There was no difference in serum bilirubin values between the groups (MD 0.53, 95% CI -0.17 to 1.22). No significant difference was observed regards polycythaemia between UCM and DCC (RR 0.53, 95% CI 0.10 to 2.79). No significant difference in blood pressure was observed between UCM and DCC (MD 4.00, 95% CI 0.32 to 7.68). No significant difference in requirement of phototherapy was observed between UCM and DCC (MD -5.00, 95% CI -11.45 to 1.45). No significant difference in incidence in incidence of necrotising enterocolitis was observed between UCM and DCC (RR 0.62, 95% CI 0.13 to 2.86).No significant difference was observed in the duration of hospital stay between UCM and DCC (MD -4.00, 95% CI -17.93 to 9.93)

They concluded that UCM did not reduce in-hospital mortality and need for transfusion compared to DCC, but UCM may lower the risk of IVH and improve certain neurodevelopmental outcomes compared to DCC in preterm infants.[5]

Early vs delayed cord clamping

A systematic review and meta-analysis by Fogarty et al examined 18randomised controlled trials comparing delayed vs early clamping in 2,834 infants. Most infants allocated to have delayed clamping were assigned a delay of ≥60 seconds. Delayed clamping reduced hospital mortality (RR 0.68; 95% CI 0.52–0.90; risk difference, –0.03; 95% CI –0.05 to –0.01; P = .005; number needed to benefit, 33; 95% CI 20–100). In 3 trials in 996 infants ≤28 weeks' gestation, delayed clamping reduced hospital mortality (RR 0.70; 95% CI 0.51–0.95; risk difference, –0.05; 95% CI –0.09 to –0.01; P = .02, number needed to benefit, 20; 95% CI 11–100; I 2 = 0).

In subgroup analyses, delayed clamping reduced the incidence of low Apgar score at 1 minute, but not at 5 minutes and did not reduce the incidence of intubation for resuscitation, admission temperature, mechanical ventilation, intraventricular haemorrhage, brain injury, chronic lung disease, patent ductus arteriosus, necrotising enterocolitis, late onset sepsis or retinopathy of prematurity. Delayed clamping increased peak haematocrit by 2.73 percentage points (95% confidence interval, 1.94–3.52; P< .00001) and reduced the proportion of infants having blood transfusion by 10% (95% confidence interval, 6–13%; P< .00001). Potential harms of delayed clamping included polycythaemia and hyperbilirubinaemia.

The review concluded that there is high-quality evidence that delayed clamping reduced hospital mortality which supports current guidelines recommending delayed clamping in preterm infants.[6]

Conflicting evidence

A randomised clinical trial which was terminated early enrolled 474 out of a planned 1,500 infants born at less than 32 weeks' gestation. There was no significant difference in the composite primary outcome of death or severe intraventricular haemorrhage for the umbilical cord milking group vs the delayed umbilical cord clamping group (12% vs 8%, respectively), but umbilical cord milking was significantly associated with a higher rate of severe intraventricular haemorrhage (8% vs 3%). They concluded that among preterm infants born at less than 32 weeks' gestation, there was no significant difference in the rates of the composite primary outcome of death or severe intraventricular haemorrhage with umbilical cord milking vs delayed umbilical cord clamping, but a significantly higher rate of severe intraventricular haemorrhage (a signal of harm) in the umbilical cord milking group which led to early termination of the study.[7]

Cochrane meta-analysis

Published in September 2019, this included 48 studies involving 5,721 babies with data available from 40 studies involving 4,884 babies and their mothers. Babies were between 24 and 36+6 weeks' gestation at birth. Delayed clamping ranged between 30 to 180 seconds, with most studies delaying for 30 to 60 seconds. Early clamping was less than 30 seconds and often immediate. UCM was mostly before cord clamping but some were milked after cord clamping.

DCC probably reduces the number of babies who die before discharge compared with ECC (average risk ratio (aRR) 0.73, 95% CI 0.54 to 0.98, 20 studies, 2,680 babies). DCC may make little or no difference to the number of babies with severe intraventricular haemorrhage (IVH grades 3 and 4) (aRR 0.94, 95% CI 0.63 to 1.39, 10 studies, 2,058 babies) but slightly reduces the number of babies with any grade IVH (aRR 0.83, 95% CI 0.70 to 0.99, 15 studies, 2,333 babies).

Due to insufficient data, they were unable to form conclusions regarding periventricular leukomalacia (PVL) (aRR 0.58, 95% CI 0.26 to 1.30, 4 studies, 1,544 babies) or maternal blood loss of 500 mL or greater (aRR 1.14, 95% CI 0.07 to 17.63, 2 studies, 180 women).There are insufficient data for reliable conclusions about the comparative effects of UCM compared with delayed or early clamping.

They concluded that delayed cord clamping probably reduced the risk of death for babies born preterm. Early cord clamping probably causes harm. No studies showed what length of delay was best, and only a few studies followed babies for health outcomes in early childhood. There is insufficient evidence for reliable conclusions on providing immediate care for the baby beside the mother with the cord intact and on cord milking. [8]

Conclusions

Delayed cord clamping in the should be the standard of care in modern obstetrics. In term infants, delayed umbilical cord clamping increases haemoglobin levels at birth and improves iron stores in the first several months of life, which may have a favourable effect on developmental outcomes.[9]

DCC brings a host of benefits especially in the setting of preterm birth with reduction in neonatal death rates and lower incidence of intraventricular haemorrhage. Umbilical cord milking is another option where early neonatal resuscitation is necessary and delayed cord clamping may be counter-productive. However, large randomised trials and substantial robust evidence is required before this can be widely recommended.

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Induction of labour vs expectant management in low risk nulliparous women

Dr Ameya Purandare, Dr Pratik Tambe

Background

To induce or not to induce at term in low risk primigravid women has been the subject of debate for several decades. While many obstetricians in current practice lean favourably toward the empirical practice of elective induction with a view to reducing maternal and neonatal morbidity, evidence has been conflicting.

Many professionals have to balance multiple roles and avoiding routine obstetric work on weekends necessitates that women at term should be induced on a weekday when support teams consisting of consultants, assistants, anaesthetists, nursing and support staff are at full strength.

In this article, we examine the current scientific evidence regarding the elective induction of labour in low risk nulliparous women at term vis a vis the established practice of allowing women to go into labour spontaneously.

Previous recommendations

Elective delivery prior to 39 completed weeks has been associated with poorer perinatal outcomes than at term. Hence, the guidance so far has been to induce only after 41 completed weeks.[2,3] This stems from the concern that elective induction between 39+0 and 40+6 weeks may lead to higher rates of caesarean section and other adverse maternal and neonatal outcomes.

Unfortunately, these conclusions were derived from older studies with a flawed premise where induction of labourwas compared with spontaneously occurring labour.[4] This assumes that all women will go into spontaneous labour, which we are aware is not the case. To further complicate the issue, there are other studies which have demonstrated that induction of labour results in a lower frequency of caesarean sections and comparatively better perinatal outcomes when compared to expectant management.[5]

Evolution of ideas

A randomised controlled trial conducted in the UK in 2016 compared induction of labour at 39 weeks of gestation against expectant management among 619 women who were 35 years of age or older and who had no other concomitant high risk factor at 39 weeks of gestation.[6]

The frequency of caesarean delivery was similar in the two groups (relative risk[RR] 0.99; 95% confidence interval [CI] 0.87 to 1.14). However, the rate of operative vaginal delivery was more than 30%, which probably indicates that in a liberal environment, the rate of caesarean delivery would be much higher and leaning toward to expectant management alternative as a conclusion. The authors requested the scientific community to replicate the trial in different populations with a larger sample size so that appropriate conclusions could be drawn.

ARRIVE Trial

The ARRIVE trial (A Randomized Trial of Induction Versus Expectant Management) conducted in 2018 was designed to test the hypothesis that elective induction of labour at 39 weeks would result in a lower risk of a composite outcome of perinatal death or severe neonatal complications than expectant management among low-risk nulliparous women. This was funded by the Eunice Kennedy Shriver National Institute of Child Health and Human Development and Maternal–Fetal Medicine Units Network.[1]

Materials, methods and primary outcomes

The multicentric trial had women randomly assigned to two groups: A labour induction and B expectant management. The large sample size had 3,602 women in Group A and 3,044 women in Group B.

The cervix was examined before labour, from 72 hours before to 24 hours after randomisation, to assess dilation, effacement, and station of the fetus to determine a modified Bishop score. A specific induction protocol was not mandated for women who underwent induction in either group.

The primary outcome considered perinatal death or severe neonatal complications which included one or more of the following during the antepartum or intrapartum period or during the delivery hospitalisation: perinatal death, the need for respiratory support within 72 hours after birth, Apgar score of 3 or less at 5 minutes, hypoxic–ischemic encephalopathy, seizure, infection (confirmed sepsis or pneumonia), meconium aspiration syndrome, birth trauma (bone fracture, neurologic injury, or retinal hemorrhage), intracranial or subgalealhemorrhage, or hypotension requiring vasopressor support. The main secondary outcome was caesarean delivery.

Observations and discussion

The incidence of perinatal death and severe neonatal complications was 4.3% in the induction group and in 5.4% in the expectant-management group (RR 0.80; 95%CI, 0.64 to 1.00). This finding did not change after adjustment for previous pregnancy loss and was materially unchanged in the sensitivity analyses. Neonates in the induction group also had a shorter duration of respiratory support and total hospital stay.

The frequency of caesarean delivery was significantly lower in the induction group than in the expectant-management group (18.6% vs. 22.2%; RR 0.84; 95% CI, 0.76 to 0.93).This finding did not change after adjustment for previous pregnancy loss. Women assigned to induction of labour were significantly less likely than women assigned to expectant management to have hypertensive disorders of pregnancy (9.1% vs. 14.1%; RR 0.64; 95% CI 0.56 to 0.74; P<0.001) and to have extensions of the uterine incision during caesarean section; women in the induction group reported less pain. Women in the induction group spent more time in the labour and delivery unit, but the length of their postpartum hospital stay was shorter.

The specifics of the primary and secondary outcomes are reproduced below:

Primary Perinatal Outcome and Components

Outcome	Induction Group (N=3059)	Expectant Management Group (N=3037)	Relative Risk (95% CI)	P Value
Primary Composite outcome	132 (4.3)	164 (5.4)	0.80 (0.64-1.00)	0.049
Perinatal death	2 (0.1)	3 (0.1)	0.66 (0.12-3.83)	
Respiratory support	91 (3.0)	127 (4.2)	0.71 (0.55-0.93)	
Apgr Score <3 at 5 min	12 (0.4)	18 (0.6)	0.66 (0.32 - 1.37)	
Hypoxic - ischemic encephalopathy	14 (0.5)	20 (0.7)	0.70 (0.35 - 1.37)	
seizure	11 (0.4)	4 (0.1)	2.74 (0.91 - 8.12)	
infection	9 (0.3)	12 (0.4)	0.74 (0.31 - 1.76)	
Meconium aspiration syndrome	17 (0.6)	26 (0.9)	0.65 (0.35 - 1.19)	
Birth trauma	14 (0.5)	18 (0.6)	0.77 (0.38 - 1.55)	
Intracranial or subgaleal hemorrhage	9 (0.3)	7 (0.2)	1.28 (0.48 - 3.42)	
Hypotension requiring vasopressor support	2 (0.1	5(0.2)	0.40 (0.06 - 1.79)	

Secondary Outcomes

Outcome	Induction Group (N=3059)	Expectant Management Group (N=3037)	Relative Risk (95% CI)	P Value
Neonatal				
Transfusion of blood products no. (%)	4 (0.1)	5 (0.2)	0.79 (0.20-2.74)	0.75
Hyperbilirubinemia - no. (%)	145 (4.7)	142 (4.7)	1.01 (0.81-1.27)	0.91
Hypoglycemia - (%)	37 (1.2)	35 (1.2)	1.05 (0.66-1.66)	0.84
Admission to neonatal intermediate or intesive care Unit – no (%)	358(11.7)	394 (13.0)	0.90 (0.79-1.03)	0.13
Maternal				
Cesarean delivery – no (%)	569 (18.6)	674 (22.2)	0.84 (0.76-0.93)	<0.001
Operative vaginal delivery – no (%)	222 (7.3)	258 (8.5)	0.85 (0.72-1.01)	0.07
Hypertensive disorder of pregnancy – no. (%	277 (9.1)	427 (14.0)	0.64 (0.56-0.74)	0.001
Chorioamnionitis – no (%)	407 (13.3)	429 (14.1)	0.94 (0.83-1.07)	0.35
Third degree or fourth degree perineal laceration – no (%	103 (3.4)	89 (2.9)	1.15 (0.87-1.52)	0.33
Postpartum hemorrhage no (%)	142 (4.6)	137 (4.5)	1.03 (0.82-1.29)	0.81
Postpartum infection no (%)	50 (1.6)	65 (2.1)	0.76 (0.53-1.10)	0.15
Admission to ICU – no (%)	4 (0.1)	8 (0.3)	0.50 (0.13-1.55)	0.26
Death – no (%)	0	0	NA	NA
Median duration of stay in labor and delivery unit (QR) hrs	20 (13-28)	14 (9-20)		<0.001
				0.01
Postpartum hospital stay — no (%)	322 (10.5)	317 (10.4)		
<2 days	2191 (71.6)	2084 (68.6)		
2 days	399 (13.0)	452 (14.9)		
3 days	130 (4.2)	166 (5.5)		
4 days	17 (0.6)	18 (0.6		
> days				
Median scores on labor agentry Scale (IQR)				
At 6-96 hr after delivery	168 (148-183)	164 (143-181)		<0.001
At 4-8 wk after delivery	176 (157-183)	174 (154-188)		0.01
Median labor pain scores (IQR)				
Worst score	8 (7-10)	9(8-10)		<0.001
Overall score	7 (5-8)	7(5-9)		<0.001

This is the largest trial with over 6,000 women comparing induction of labour vs expectant management in low risk women. The researchers found that the relative risk of adverse perinatal outcomes was 20% lower in the induction group than in the expectant-management group and they concluded that induction of labourmay be associated with as much as a 36% lower risk than expectant management. Induction also resulted in a significantly lower frequency of caesarean section and hypertensive disorders of pregnancy than expectant management.

The results are in line with other recent smaller studies which compared these two groups when compared to the older studies which compared induction of labour vs spontaneously occurring labour.[7,8,9] Thisdata suggests that 1 caesarean delivery may be avoided for every 28 vaginal deliveries among low-risk nulliparous women who undergo elective induction of labour at 39 weeks when compared with expectant management.

Conclusions

There is now a paradigm shift in the approach to management of low risk nulliparous women. While previous guidance recommended awaiting spontaneous onset of labour and only inducing after 41 completed weeks, multiple studies including the ARRIVE Trial which is the largest on record has shown beyond any doubt that induction of labor at 39 weeks in low-risk nulliparous women resulted in a lower frequency of adverse perinatal outcomes and significantly lowers the frequency of caesarean section in the group of patients.

Institutional and practice based policies which defer induction of labour in favour of expectant management are unlikely to reduce the rate of caesarean section at the population level and the updated evidence needs to be taken into consideration when formulating policies and establishing algorithms for dealing with low risk nulliparous women who form the largest subset of patients in most obstetric practices.

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Prevention of Group B Streptococcal Early-Onset Disease in Newborns
ACOG Committee Opinion Number 797

Obstet Gynecol Feb 2020: 135(2): p e51-e72
doi: 10.1097/AOG.0000000000003668

Abstract

Group B streptococcus (GBS) is the leading cause of newborn infection. The primary risk factor for neonatal GBS early-onset disease (EOD) is maternal colonisation of the genitourinary and gastrointestinal tracts. Approximately 50% of women who are colonised with GBS will transmit the bacteria to their newborns.

Vertical transmission usually occurs during labour or after rupture of membranes. In the absence of intrapartum antibiotic prophylaxis, 1–2% of those newborns will develop GBS EOD. Other risk factors include gestational age of less than 37 weeks, very low birth weight, prolonged rupture of membranes, intraamniotic infection, young maternal age, and maternal black race.

The key obstetric measures necessary for effective prevention of GBS EOD include universal antenatal screening by vaginal–rectal culture, correct specimen collection and processing, appropriate implementation of intrapartum antibiotic prophylaxis and coordination with paediatric care providers.

The American College of Obstetricians and Gynecologists now recommends performing universal GBS screening between 36 0/7 and 37 6/7 weeks of gestation. All women whose vaginal–rectal cultures at 36 0/7–37 6/7 weeks of gestation are positive for GBS should receive appropriate intrapartum antibiotic prophylaxis unless a prelabour caesarean birth is performed in the setting of intact membranes.

Key recommendations and conclusions

Targeted intravenous intrapartum antibiotic prophylaxis has demonstrated efficacy for prevention of GBS early-onset disease (EOD) in neonates born to women with positive antepartum GBS cultures and women who have other risk factors for intrapartum GBS colonization. Neither antepartum nor intrapartum oral or intramuscular regimens have been shown to be comparably effective in reducing GBS EOD.

Regardless of planned mode of birth, all pregnant women should undergo antepartum screening for GBS at 36 0/7–37 6/7 weeks of gestation, unless intrapartum antibiotic prophylaxis for GBS is indicated because of GBS bacteriuria during the pregnancy or because of a history of a previous GBS-infected newborn. This new recommended timing for screening provides a 5-week window for valid culture results that includes births that occur up to a gestational age of at least 41 0/7 weeks.

All women whose vaginal–rectal cultures at 36 0/7–37 6/7 weeks of gestation are positive for GBS should receive appropriate intrapartum antibiotic prophylaxis unless a prelabour caesarean birth is performed in the setting of intact membranes.

Women with a positive prenatal GBS culture result who undergo a caesarean birth before the onset of labour and with intact membranes do not require GBS antibiotic prophylaxis. If the antenatal GBS culture result is unknown when labour starts, intrapartum antibiotic prophylaxis is indicated for women who have risk factors for GBS EOD.

At-risk women include those who present in labour with a substantial risk of preterm birth, who have preterm prelabour rupture of membranes (PPROM) or rupture of membranes for 18 or more hours at term, or who present with intrapartum fever (temperature 100.4°F [38°C] or higher).

If intraamniotic infection is suspected, broad-spectrum antibiotic therapy that provides coverage for poly-microbial infections as well as GBS should replace the antibiotic that provides coverage for GBS prophylaxis specifically.

If a woman presents in labour at term with unknown GBS colonisation status and does not have risk factors that are an indication for intrapartum antibiotic prophylaxis but reports a known history of GBS colonisation in a previous pregnancy, the risk of GBS EOD in the neonate is likely to be increased. With this increased risk, it is reasonable to offer intrapartum antibiotic prophylaxis based on the woman's history of colonisation. Health care providers also may consider discussing the option of empirical intrapartum antibiotic prophylaxis as a shared decision-making process in this clinical scenario.

Intravenous penicillin remains the agent of choice for intrapartum prophylaxis, with intravenous ampicillin as an acceptable alternative. First-generation cephalosporins (i.e., cefazolin) are recommended for women whose reported penicillin allergy indicates a low risk of anaphylaxis or is of uncertain severity. For women with a high risk of anaphylaxis, clindamycin is the recommended alternative to penicillin only if the GBS isolate is known to be susceptible to clindamycin.

Intravenous vancomycin remains the only pharmacokinetically and microbiologically validated option for intrapartum antibiotic prophylaxis in women who report a high-risk penicillin allergy and whose GBS isolate is not susceptible to clindamycin. The vancomycin dosage for intrapartum GBS prophylaxis should be based on weight and baseline renal function (20 mg/kg intravenously every 8 hours, with a maximum of 2 g per single dose.)

Obstetric interventions, when necessary, should not be delayed solely to provide 4 hours of antibiotic administration before birth. Such interventions include but are not limited to administration of oxytocin, artificial rupture of membranes, or planned caesarean birth, with or without precaesarean rupture of membranes. However, some variation in practice may be warranted based on the needs of individual patients to enhance intrapartum antibiotic exposure.

Fig 1 Management in the setting of Preterm Labour

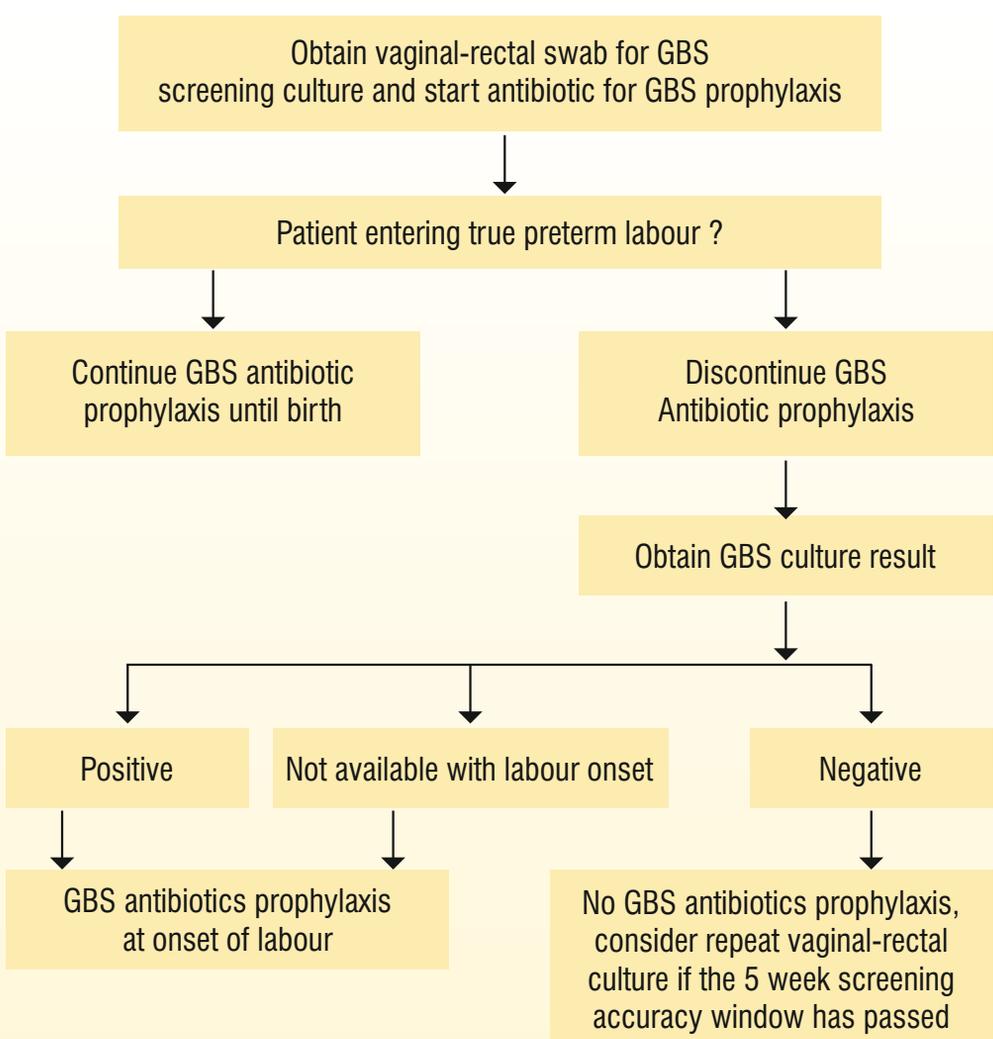

Fig 2 Management in the setting of PPROM

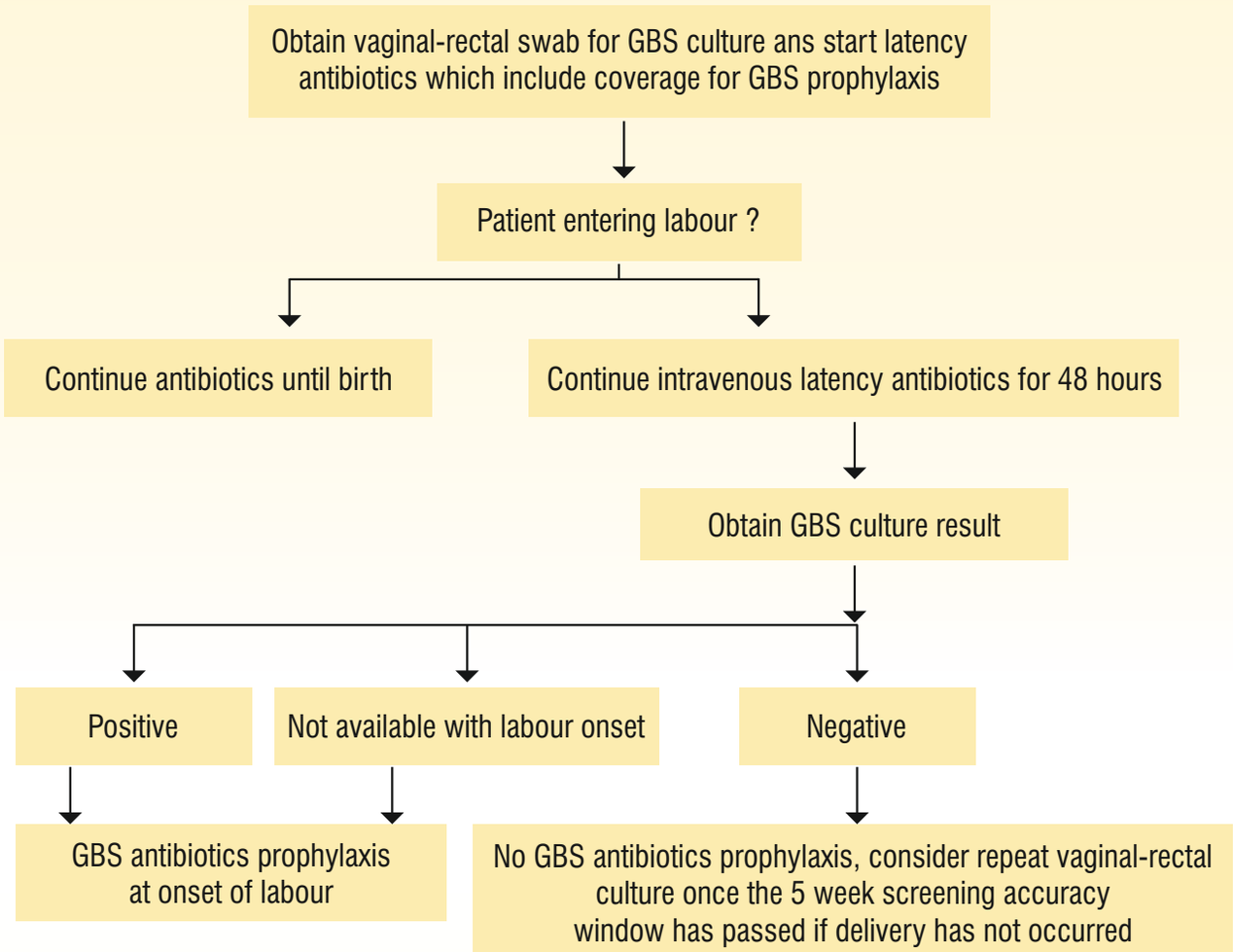

Fig 3 Choice of antibiotic for prophylaxis