Bringing to life the best in

SFH should be plotted on a customised chart rather than a population–based chart as this may improve prediction of a SGA neonate.

Women with a single SFH which plots below the 10th centile or serial measurements which demonstrate slow or static growth by crossing centiles should be referred for ultrasound measurement of fetal size.

Women in whom measurement of SFH is inaccurate (for example: BMI

Interventions to be considered in the prevention of SGA fetuses/neonates

Antiplatelet agents may be effective in preventing SGA birth in women at high risk of pre-eclampsia although the effect size is small.

In women at high risk of pre-eclampsia, antiplatelet agents should be commenced at, or before, 16 weeks of pregnancy.

There is no consistent evidence that dietary modification, progesterone or calcium prevent birth of a SGA infant. These interventions should not be used for this indication.

In the preterm SGA fetus, middle cerebral artery (MCA) Doppler has limited accuracy to predict acidaemia and adverse outcome and should not be used to time delivery.

In the term SGA fetus with normal umbilical artery Doppler, an abnormal middle cerebral artery Doppler (PI < 5th centile) has moderate predictive value for acidosis at birth and should be used to time delivery.

Ductus venosus Doppler has moderate predictive value for acidaemia and adverse outcome.

Ductus venosus Doppler should be used for surveillance in the preterm SGA fetus with abnormal umbilical artery Doppler and used to time delivery.

The optimal gestation to deliver the SGA fetus

1.2. Interventions to be studied

Comparison of modalities to screen for and diagnose a SGA fetus.
Comparison of modalities to monitor a SGA fetus.

2. Definitions

Small–for–gestational age (SGA) refers to an infant born with a birth weight less than the 10th centile. Historically SGA birth has been defined using population centiles. But, the use of centiles customised for maternal characteristics (maternal height, weight, parity and ethnic group) as well as gestational age at delivery and infant sex, identifies small babies at higher risk of morbidity and mortality than those identified by population centiles.[1–2]

exercise,[32] a short (< 6 months) or long (> 60 months) inter–pregnancy interval[33] and heavy vaginal bleeding during the first trimester.[34] The effect of some of these risk factors is reduced once adjusted for other associated factors and thus they are not included in Appendix 1. Maternal exposure to domestic violence during pregnancy has been shown in a systematic review to be associated with low birth weight (Adjusted OR [AOR] 1.53, 95% CI 1.28–1.82).[35] Low maternal weight gain has been shown to be associated with a SGA infant in a preterm population (OR 4.9, 95% CI 1.9–12.6)[13] but it is no longer recommended that women are routinely weighed during pregnancy.[36]

Several maternal exposures have a seemingly causative relationship with a SGA infant, including moderate alcohol intake,[37] drug use (with cocaine use during pregnancy being the most significant)[38] and cigarette smoking.[39] The effects of smoking are dose dependent.[29]

Other risk factors are maternal caffeine consumption ≥ 300 mg per day in the third trimester[40] and a low fruit intake pre–pregnancy, while a high green leafy vegetable intake pre–pregnancy has been reported to be protective (AOR 0.44, 95% CI 0.24–0.81).[32] Singleton pregnancies following IVF are also a risk factor for a SGA fetus.[41]

Changing paternity has been associated with an increased risk of a SGA infant,[42] although a recent systematic review demonstrated inconclusive evidence.[43] A paternal history of SGA birth is a risk factor for a SGA fetus.[44]

11.3–16.7; LR– 0.37, 95% CI 0.27–0.52) and < 32 weeks in one study (LR+ 14.6, 95% CI 11.5–18.7; LR– 0.31 0.18–0.53).

In approximately 60% of cases with abnormal uterine artery Doppler at 20–22 weeks of gestation, PI remains increased at 26–28 weeks.[56]

Use of a customised fetal weight reference may improve prediction of a SGA neonate and adverse perinatal outcome. In women having serial assessment of fetal size, use of a customised fetal weight reference may improve the prediction of normal perinatal outcome.

Routine measurement of fetal AC or EFW in the third trimester does not reduce the incidence of a SGA

has also been shown to improve the prediction of adverse prenatal outcome;[90,91] OR of adverse outcomes (stillbirths, neonatal deaths, referral to higher level or special care unit or Apgar score < 7 at 5 minutes) for SGA neonates versus those not SGA was 1.59 (95% CI 1.53–1.66) for the non–customised fetal weight reference compared with 2.84 (95% CI 2.71–2.99) for the customised reference.[90] Prediction of perinatal mortality was also improved by the customised reference (OR 3.65, 95% CI 3.40–3.92 versus OR 1.77, 95% CI 1.65–1.89).[91] A further study demonstrated that individual growth trajectories of low risk fetuses with normal outcome were less likely to cross below the 10th centile for fetal weight when using customised reference standards than when unadjusted standards were used.[92] However, no trials were identified that compared customised with non–customised EFW chartsp.

Evidence level 3

A meta–analysis, including eight trials comprising 27 024 women, found no evidence that routine fetal biometry (with or without assessment of amniotic fluid volume and placental grade) after 24 weeks of pregnancy improved perinatal outcome in a low risk population (SGA neonate relative risk [RR] 0.98, 95% CI 0.74–1.28; perinatal mortality RR 0.94, 95% CI 0.55–1.61).[93] The timing and content of the ultrasound scan varied substantially between studies and the authors noted high heterogeneity between studies in the reduction of the risk of a SGA neonate, mainly due to the findings of one study in which routine estimation of fetal weight, amniotic fluid volume and placental grading at 30–32 and 36–37 weeks of gestation was shown to result in the birth of fewer SGA neonates (10.4% versus 6.9%, RR 0.67, 95% CI 0.50–0.89).[94]

Evidence level 1+

The change in fetal size between two time points is a direct measure of fetal growth and hence serial measurement of AC or EFW (growth velocities) should allow the diagnosis of FGR. However the optimal method of using serial ultrasound measurements is not clear. Although 'eyeballing' a chart of individual AC or EFW measurements may give an impression of FGR a more objective definition requires establishment of growth rate standards from longitudinally collected data. Several standards have been reported,[95,96] including conditional centiles for fetal growth,[97] although none has been adopted in clinical practice. Reported mean growth rates for AC and EFW after 30 weeks of gestation are 10 mm/14 days and 200 g/14 days although greater variation exists in the lower limits (reflecting the methods used to derive the standard deviation [SD]).[98] However a change in AC of < 5mm over 14 days is suggestive of FGR.[95] In a high risk population, identified as being SGA, Chang et al.[99,100] showed that a change in AC or EFW (defined as a change in SD score of ≥ –1.5) were better predictors of wasting at birth (ponderal index, mid–arm circumference/head circumference ratio or subscapular skinfold thickness < 2 SD below mean) and adverse perinatal outcome than the final AC or EFW before delivery.

Evidence level 2+

Mongelli et al.[101] used a mathematical model to estimate the impact of time interval between examinations on the false positive rates for FGR (defined as no apparent growth in fetal AC between two consecutive examinations). When the initial scan was performed at 32 weeks of gestation, the false positive rates were 30.8%, 16.9%, 8.1% and 3.2% for intervals of 1,2,3 and 4 weeks respectively. False positive rates were higher when the first scan was performed at 36 weeks of gestation (34.4%, 22.1%, 12.7%, 6.9% respectively). These findings suggest that if two measurements are to be used to estimate velocity, they should be a minimum of 3 weeks apart to minimise false–positive rates for diagnosing FGR. This recommendation does not preclude more frequent ultrasound measurements of AC/EFW to predict fetal size at birth but rather indicates which measurements should be used to interpret growth.

Evidence level 3

6.2 Biophysical tests

Biophysical tests, including amniotic fluid volume, cardiotocography (CTG) and biophysical scoring are poor at diagnosing a small or growth restricted fetus.[102–104] A systematic review of the accuracy of umbilical artery

Doppler in a high–risk population to diagnose a SGA neonate has shown moderate accuracy (LR+ 3.76, 95% CI 2.96–4.76; LR– 0.52, 95% CI 0.45–0.61).[105]

7. What investigations are indicated in SGA fetuses?

Offer a referral for a detailed fetal anatomical survey and uterine artery Doppler by a fetal medicine specialist if severe SGA is identified at the 18–20 week scan.

Karyotyping should be offered in severely SGA fetuses with structural anomalies and in those detected before 23 weeks of gestation, especially if uterine artery Doppler is normal.

Serological screening for congenital cytomegalovirus (CMV) and toxoplasmosis infection should be offered in severe SGA.

Testing for syphilis and malaria should be considered in high risk populations.

Uterine artery Doppler has limited accuracy to predict adverse outcome in SGA fetuses diagnosed during the third trimester.

In severe SGA, the incidence of chromosomal abnormalities has been reported to be as high as 19%.[104] Triploidy was the most common chromosomal defect in fetuses referred before 26 weeks of gestation and trisomy 18 in those referred thereafter. Within this population, the risk of aneuploidy was found to be higher in fetuses with a structural abnormality, a normal amniotic fluid volume, a higher head circumference/AC ratio or a normal uterine artery Doppler.[106,107] One small study suggested that, in severely SGA fetuses, the rate of aneuploidy was 20% in fetuses presenting before 23 weeks of gestation, irrespective of the presence of structural anomalies, compared with 0% in fetuses presenting between 23–29 weeks of gestation.[107]

Fetal infections are responsible for up to 5% of SGA fetuses.[108] The most common pathogens are reported to be cytomegalovirus (CMV), toxoplasmosis, malaria and syphilis,[108] although a recent multicentre study found no association between congenital toxoplasmosis and incidence of a SGA infant.[109] Malaria is a significant cause of preterm birth and LBW worldwide and it should be considered in those from, or who have travelled in, endemic areas.[110]

The predictive value of uterine artery Doppler in SGA fetuses diagnosed during the third trimester is unclear and no systematic reviews on this topic were identified in the literature search for this guideline. Severi et al.[111] found that uterine artery RI > 0.50 and bilateral notching were independently associated with emergency caesarean section in this population (OR 5.0, 95% CI 2.0–12.4; OR 12.2, 95% CI 2.0–74.3 respectively). Other studies have suggested that uterine artery Doppler has no predictive value.

Smoking increases the risk of SGA, and 21 trials involving over 20 000 women have addressed the impact of interventions to promote smoking cessation in pregnancy.[124] Overall interventions reduced low birth weight (RR 0.83, 95% CI 0.73–0.95) and preterm birth but SGA was not reported in the systematic review as an outcome. Trials using cognitive behavioural therapy and incentives as the main intervention strategy demonstrated consistent improvements in birthweight.[124] Women who are able to stop smoking by 15 weeks of gestation can reduce the risk back to that of non–smokers.[39]

Antithrombotic therapy has been used to improve outcome in women considered at risk of placental dysfunction (primarily based on previous history of pre-eclampsia, FGR or stillbirth). A systematic review of five studies involving 484 women, four of which compared heparin (either alone or with dipyridamole) with no treatment, found that heparin reduced the incidence of SGA neonates from 25% to 9% (RR 0.35, 95% CI 0.20–0.64) and also reduced the incidence of pre-eclampsia.[125] However, no differences were evident in perinatal mortality or preterm birth below 34 weeks. The authors concluded that while this therapy appears promising, important information about serious adverse effects and long–term childhood outcomes is unavailable.

Antihypertensive drug therapy for mild to moderate hypertension in pregnancy does not seem to increase the risk of delivering a SGA neonate (19 trials, 2437 women, RR 1.02, 95% CI 0.89–1.16),[126] but treatment with oral beta–blockers was associated with an increased risk of a SGA neonate (RR 1.36, 95% CI 1.02–1.82), partly dependent on one small outlying trial involving atenolol.[127] Use of atenolol is therefore best avoided but no recommendation can be made regarding the best agent or target blood pressure to optimise fetal growth, especially when the fetus is known to be SGA.[128]

9. What interventions should be considered in the preterm SGA fetus?

Women with a SGA fetus between 24^{+0} and 35^{+6} weeks of gestation, where delivery is being considered, should receive a single course of antenatal corticosteroids.

Women with a SGA fetus between 24^{+0} and 35^{+6} weeks of gestation, where delivery is being considered, should receive a single course of antenatal corticosteroids to accelerate fetal lung maturation and reduce neonatal death and morbidity.[129]

Bed rest in hospital for a suspected SGA infant has only been evaluated in one trial of 107 women that showed no differences in any fetal growth parameters.[130]

Maternal oxygen administration has been investigated in three trials of SGA fetuses involving 94 women.[131] Methodological problems were identified in two of the studies, both of which had greater gestational ages of fetuses in the oxygen group. This may account for the increase in birth weight in the intervention group. Oxygenation was associated with a lower perinatal mortality (RR

allowing timely delivery prior to irreversible end–organ damage and intrauterine fetal dea

10.11 Umbilical artery Doppler

In a high–risk population, the use of umbilical artery Doppler has been shown to reduce p morbidity and mortality. Umbilical artery Doppler should be the primary surveillance too SGA fetus.

monitored with umbilical artery Doppler, unidentified SGA fetuses have a fourfold greater risk of adverse fetal outcome (OR 4.1, 95% CI 2.5–6.8) and fetal/infant death (OR 4.2, 95% CI 2.1–8.5).[144] In this large series, SGA fetuses (defined as a birth weight deviation 22–27% below the norm, equivalent to –2 SDs) were monitored with two weekly umbilical artery Doppler. However, compared to appropriate for gestational age (AGA) fetuses, SGA fetuses with a normal umbilical artery Doppler are still at increased risk of neonatal morbidity (OR 2.26, 95% CI 1.04–4.39)[141] and adverse neurodevelopmental outcome.[145]

Evidence level 2+

In SGA fetuses with abnormal umbilical artery Doppler where there is not an indication for delivery the optimal frequency of surveillance is unclear. Until definitive evidence becomes available it is reasonable to repeat surveillance twice weekly in fetuses with end–diastolic velocities present and daily in fetuses with absent or reversed end–diastolic velocities (AREDV).

Evidence level 4

In a low risk or unselected population, a systematic review of five trials, involving 14 185 women, found no conclusive evidence that routine umbilical artery Doppler benefits mother or baby.[146] As such, umbilical artery Doppler is not recommended for screening an unselected population.

Evidence level 1+

10.2 Cardiotocography (CTG)

CTG should not be used as the only form of surveillance in SGA fetuses.

A

Interpretation of the CTG should be based on short term fetal heart rate variation from computerised analysis.

A

Antenatal CTG has been compared with no intervention in a Cochrane systematic review of RCTs. Based on four trials (1627 fetuses) of high risk pregnancies there was no clear evidence that antenatal CTG improved perinatal mortality (RR 2.05, 95% CI 0.95–4.42). The included trials all employed visual analysis and only one trial was regarded as high quality.[147]

Evidence level 1+

Unlike conventional CTG, which has high intra– and interobserver variability, computerised CTG (cCTG) is objective and consistent.[148] Normal ranges for cCTG parameters throughout gestation are available.[149] Fetal heart rate (FHR) variation is the most useful predictor of fetal wellbeing in SGA fetuses;[150,151] a short term variation ≤ 3 ms (within 24 hours of delivery) has been associated with a higher rate of metabolic acidaemia (54.2% versus 10.5%) and early neonatal death (8.3% versus 0.5%).[151]

Evidence level 2+

Comparison of cCTG with traditional CTG in the Cochrane review (two trials, 469 high risk fetuses) showed a reduction in perinatal mortality with cCTG (4.2% versus 0.9%, RR 0.20, 95% CI 0.04–0.88) but no significant difference in perinatal mortality excluding congenital anomalies (RR 0.23, 95% CI 0.04–1.29), though the meta–analysis was underpowered to assess this outcome, or any other measure of adverse perinatal outcome.[147]

Evidence level 1–

10.3 Amniotic fluid volume

Ultrasound assessment of amniotic fluid volume should not be used as the only form of surveillance in SGA fetuses.

✓

Interpretation of amniotic fluid volume should be based on single deepest vertical pocket.

A

Amniotic fluid volume is usually estimated by the single deepest vertical pocket (SDVP) or amniotic fluid index (AFI) methods; although both correlate poorly with actual amniotic fluid volume.[152] A Cochrane systematic review (five trials, 3226 women) compared the two methods and concluded

Evidence level 1+

that there was no evidence that one method was superior in the prevention of adverse perinatal outcomes. However, compared to a SDVP < 2 cm, when an AFI ≤ 5 cm was used more cases of oligohydramnios were diagnosed (RR 2.39, 95% CI 1.73–3.28) and more women had induction of labour (RR 1.92, 95% CI 1.50–2.46) without an improvement in perinatal outcome.[153]

The incidence of an AFI ≤ 5 cm in a low risk population is 1.5%.[154] Compared to cases with a normal AFI, the risk of perinatal mortality and morbidity was not increased in cases with isolated oligohydramnios (RR 0.7, 95% CI 0.2–2.7) nor in those with associated conditions, including SGA fetuses (RR 1.6, 95% CI 0.9–2.6).

No systematic reviews of effectiveness of MCA Doppler as a surveillance tool in high risk or SGA fetuses were identified. A systematic review of 31 observational studies (involving 3337 fetuses) found that MCA Doppler had limited predictive accuracy for adverse perinatal outcome (LR+ 2.79, 95% CI 1.10–1.67; LR– 0.56, 95% CI 0.43–0.72) and perinatal mortality (LR+ 1.36, 95% CI 1.10–1.67; LR– 0.51, 95% CI 0.29–0.89).[165] Most studies investigating MCA Doppler as a predictor of adverse outcome in preterm SGA fetuses have reported low predictive value,[165–167] especially when umbilical artery Doppler is abnormal. In the largest study of predictors of neonatal outcome in SGA neonates of less than 33 weeks gestational age (n = 604), although MCA PI < –2 SDs was associated with neonatal death (LR 1.12, 95% CI 1.04–1.21) and major morbidity (LR 1.12, 95% CI 1.1–1.33), it was not a statistically significant predictor of outcome on logistic regression.[168] Initial findings of a pre–terminal increase (reversal) of MCA PI have not been confirmed in subsequent reports.[169,170]

MCA Doppler may be a more useful test in SGA fetuses detected after 32 weeks of gestation where umbilical artery Doppler is typically normal.[171] Studies suggest an elevated MCA PI is associated with emergency caesarean section and neonatal admission.[172,173] In one study of 210 term SGA fetuses with normal umbilical artery Doppler, MCA PI < 5th centile was predictive of caesarean section for nonreassuring fetal status (OR 18.0, 95% CI 2.84–750) and neonatal metabolic acidosis, defined as umbilical artery pH < 7.15 and base deficit > 12 mEq/L (OR 9.0, 95% CI 1.25–395).[174] Based on this evidence it is reasonable to use MCA Doppler to time delivery in the term SGA fetus with normal umbilical artery Doppler.

10.6 Ductus venosus (DV) and umbilical vein (UV) Doppler

Ductus venosus Doppler has moderate predictive value for acidaemia and adverse outcome.

Ductus venosus Doppler should be used for surveillance in the preterm SGA fetus with abnormal umbilical artery Doppler and used to time delivery.

The Ductus venosus (DV) Doppler flow velocity pattern reflects atrial pressure–volume changes during the cardiac cycle. As FGR worsens velocity reduces in the DV a–wave owing to increased afterload and preload, as well as increased end–diastolic pressure, resulting from the directs effects of hypoxia/acidaemia and increased adrenergic drive.[175] A retrograde a–wave and pulsatile flow in the umbilical vein (UV) signifies the onset of overt fetal cardiac compromise.[175]

No systematic reviews of effectiveness of venous Doppler as a surveillance tool in high risk or SGA fetuses were identified. A systematic review of 18 observational studies (involving 2267 fetuses) found that DV Doppler had moderate predictive accuracy for the prediction of perinatal mortality in high risk fetuses with placental insufficiency with a pooled LR+ of 4.21 (95% CI 1.98–8.96) and LR– of 0.43 (95% CI 0.30–0.61).[175] For prediction of adverse perinatal outcome the results were LR+ 3.15 (95% CI 2.19–4.54) and LR– 0.49 (95% CI 0.40–0.59).[176]

Observational studies have identified venous Doppler as the best predictor of acidaemia.[150,177] Turan et al.[150] reported an OR of 5.68 (95% CI 1.67–19.32) for an increased DV PI for veins (PIV) and 45.0 (95% CI 5.0–406.5) for UV pulsation compared to 2.12 (95% CI 0.66–6.83) for AREDV in the umbilical artery. In the large study of predictors of neonatal outcome in preterm SGA neonates referred to above, gestational age was the most significant determinant of intact survival until 29

considered viable and after completion of steroids. Even when venous Doppler is normal, delivery is recommended by 32 weeks of gestation and should be considered between 30–32 weeks of gestation.

If MCA Doppler is abnormal, delivery should be recommended no later than 37 weeks of gestation.

In the SGA fetus detected after 32 weeks of gestation with an abnormal umbilical artery Doppler, delivery no later than 37 weeks of gestation is recommended.

In the SGA fetus detected after 32 weeks of gestation with normal umbilical artery Doppler, a senior obstetrician should be involved in determining the timing and mode of birth of these pregnancies. Delivery should be offered at 37 weeks of gestation.

At present there is no effective intervention to alter the course of FGR except delivery. Timing delivery is therefore a critical issue in order to balance the risks of prematurity against those of continued intrauterine stay; death and organ damage due to inadequate tissue perfusion.[178]

Gestational age is a critical determinant in decision–making. Various tools exist to predict survival in very preterm births, such as the prematurity risk evaluation measure (PREM) score, which is a system derived from

A–wave becomes absent/reversed. This key question is being addressed in the ongoing trial of umbilical and fetal flow in a European RCT which aims to determine whether delivery based on reduced short term variability on cCTG leads to better neurodevelopmental outcome in surviving infants than delivery based on DV Doppler.[186]

By 31 weeks of gestation, neonatal mortality and disability rates in this population are low; in GRIT, mortality and disability rates in fetuses delivered at 31–36 weeks were 5% and 4% respectively[183] while in the large series of early onset FGR reported by Baschat et al.,[168] mortality was 8.6% in fetuses delivered at 31 weeks and 2.6% in those delivered at 32 weeks. Given the mortality associated with umbilical artery AREDV alone[178] delivery should be considered based on this finding alone after 30 weeks of gestation and recommended no later than 32 weeks of gestation.

11.2 Near term / term SGA fetus

One randomised equivalence trial exists comparing the effect of induction of labour or expectant monitoring in women beyond 36 weeks of gestation with suspected FGR (defined as a fetal AC or EFW < 10th centile or flattening of the growth curve in the third trimester, as judged by the clinician).[187]eeks a8 0 02B7

Delivery in all recent studies reporting outcome of viable SGA fetuses with umbilical artery AREDV has been by caesarean section and thus it is not possible to determine the likelihood of adverse outcome (including emergency CS for suspected fetal compromise) associated with induced/spontaneous labour. Older series report rates of intrapartum fetal heart decelerations necessitating CS of 75–95%.[193,194] More recent prospective data on the outcome of labour in SGA fetuses with an abnormal umbilical artery Doppler but end–diastolic velocities is also extremely limited; suspected fetal compromise (necessitating emergency CS) has been reported in 17–32% of such cases, compared to 6–9% in SGA fetuses with normal umbilical artery Doppler.[191,192,195] Although, it is acknowledged that knowledge of Doppler may lower obstetricians' threshold for emergency CS.[196] The offer of induction of labour with continuous FHR monitoring is therefore reasonable in term and near term fetuses, as well as SGA fetuses without umbilical artery AREDV. The procedures for induction of labour should follow existing guidance.[197]

13. Suggested audit topics

All units should audit their antenatal detection rate of the SGA neonate. Definition of a SGA neonate should be based on customised birthweight standards. Suggested auditable standards are as follows:

- All women should have a formal assessment of their risk of delivering a SGA neonate at booking.
- All women with a major risk factor for a SGA neonate should be offered serial ultrasound measurement of fetal size and assessment of wellbeing with umbilical artery Doppler.
- All women with a SGA fetus should have serial ultrasound measurement of fetal size and assessment of wellbeing with umbilical artery Doppler.
- All women with a SGA fetus where delivery is considered between 24^{+0} and 35^{+6} weeks of gestation should receive a single course of antenatal corticosteroids.

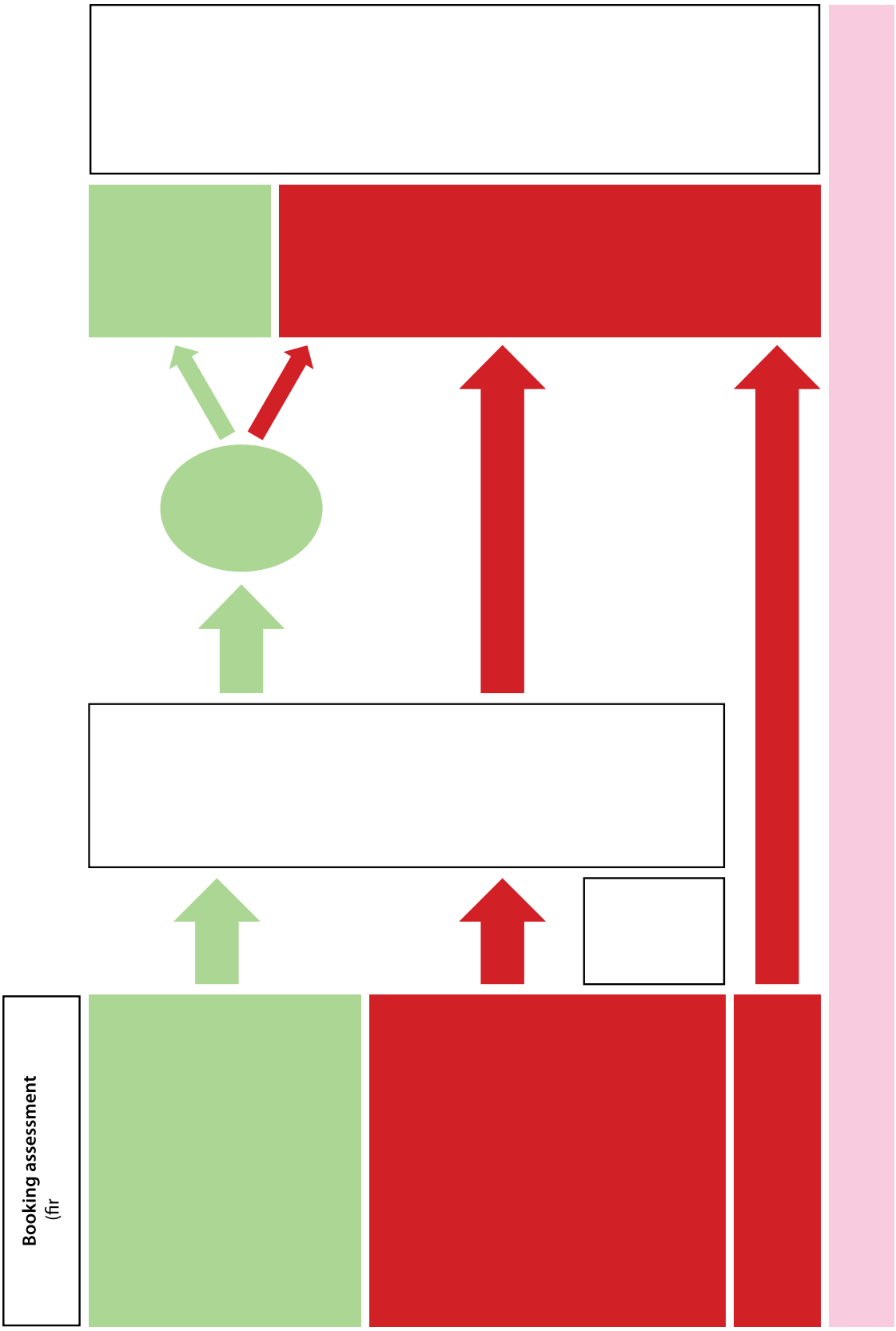
14. What are the areas for future research?

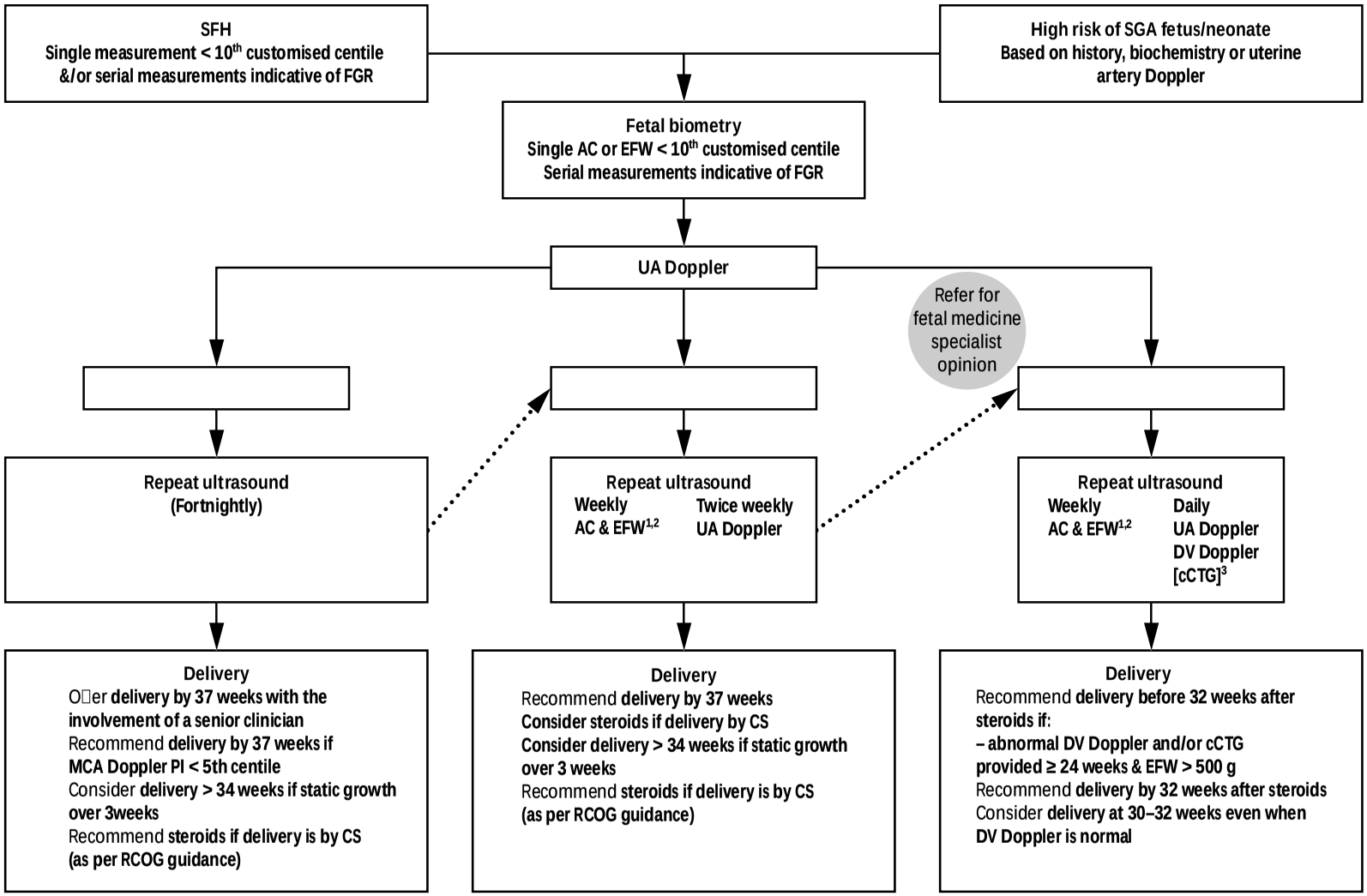
91. **Mikolajczyk RT, Zhang J, Betran AP, Souza JP, Mori R, Gülmezoglu AM, et al. A global reference for fetal–weight and birthweight percentiles.** *Lancet*

170. **Baschat AA, Gembruch U, Harman CR. The sequence of changes in Doppler and biophysical parameters as severe fetal growth restriction worsens.** *Ultrasound Obstet Gynecol* **2001;18:571–7.**
171. **Oros D, Figueras F, Cruz–Martinez R, Meler E, Munmany M, Gratecos E. Longitudinal changes in uterine, umbilical and fetal cerebral Doppler indices in late–onset small–for–gestational–age fetuses.** *Ultrasound Obstet Gynecol* **2011;37:191–5.**
172. **Hershkovitz R, Kingdom JC, Geary M, Rodeck CH. Fetal cerebral blood flow redistribution in late gestation: identification of compromise in small fetuses with normal umbilical artery Doppler.** *Ultrasound Obstet Gynecol* **2000;15:209–12.**
173. **Severi FM, Bocchi C, Visentin A, Falco P, Cobellis L, Florio P, et al. Uterine and fetal cerebral Doppler predict the outcome of third–trimester small–for–gestational age fetuses with normal umbilical artery Doppler.** *Ultrsound Obstet Gynecol* **2002;19:225–8.**
174. **Cruz–Martinez R, Figueras F, Hernandez–Andrade E, Oros D,**

Appendix I: Summary of Risk Factors for a Small–for–Gestational–Age Neonate.

Table A:





[1] Weekly measurement of fetal size is valuable in predicting birthweight and determining size-for-gestational age
[2] If two AC/EFW measurements are used to estimate growth, they should be at least 3 weeks apart
[3] Use cCTG when DV Doppler is unavailable or results are inconsistent – recommend delivery if STV < 3 ms
Abbreviations:

APPENDIX IV: Glossary

AC	Abdominal circumference
AFI	Amniotic fluid index
AFP	Alpha fetoprotein
AGA	Appropriate for gestational age
AOR	Adjusted odds ratio
APH	Antepartum haemorrhage
AREDV	Absent or Reversed End–Diastolic Velocity
BMI	Body mass index
BPP	Biophysical profile
CI	Confidence interval
CTG	Cardiotocography
cCTG	Computerised cardiotocography
CMV	Cytomegalo virus
DS	Down Syndrome
DV	Ductus venosus
EDV	End-diastolic velocities
EFW	Estimated fetal weight
FGR	Fetal growth restriction
FHR	Fetal heart rate
GRIT	Growth restriction intervention trial
hCG	Human chorionic gonadotrophin
IPD	Individual patient data
LBW	Low birth weight
LR	Likelihood ratio
LR+	Positive likelihood ratio
LR–	Negative likelihood ratio
MCA	Middle cerebral artery
MeSH	Medical subject heading
MoM	Multiples of the median
OR	Odds ratio
PAPP–A	Pregnancy associated plasma protein–A
PI	Pulsatility Index
PIV	Pulsatility Index for veins
PREM	Prematurity risk evaluation measure
RCT	Randomised controlled trial
RR	Relative risk
SDVP	Single deepest vertical pocket
SFH	Symphysis fundal height
SGA	Small–for–gestational–age
STV	Short term variation
TRUFFLE	Trial of umbilical and fetal flow in Europe

APPENDIX V: Explanation of Guidelines and Evidence Levels

Clinical guidelines are:'systematically developed statements which assist clinicians and women in making decisions about appropriate treatment for specific conditions'. Each guideline is systematically developed using a standardised methodology. Exact details of this process can be found in Clinical Governance Advice No.1: *Development of RCOG Green-top Guidelines* **(available on the RCOG website at**

This guideline was produced on behalf of the Guidelines Committee of the Royal College of Obstetricians and Gynaecologists by:
Professor SC Robson MRCOG, Newcastle–upon–Tyne; Dr WL Martin FRCOG, Birmingham and Dr RK Morris MRCOG, Birmingham.

Committee Lead Reviewers: Dr P Owen FRCOG, Glasgow, Scotland; Ms CJ Elson FRCOG, Leicestershire; Mr DJ Cruickshank FRCOG, Middlesborough

and peer–reviewed by: British Maternal and Fetal Medicine Society (BMFMS); British Medical Ultrasound Society (BMUS); British Society of Urogenital Radiology (BSUR); Clinical Studies Group for Stillbirth (CSGS, hosted by SANDS); International Society of Ultrasound in Obstetrics and Gynaecologist (ISUOG); Perinatal Institute; Dr UB Agarwal MRCOG, Liverpool; Professor JC Dornan FRCOG, County Down, Northern Ireland; Dr MA Harper FRCOG, Belfast; Mr B Kumar FRCOG, Wrexham; Dr AC McKelvey MRCOG, Norfolk; Professor LME McCowan, University of Auckland, New Zealand; Mr DJ Tuffnell FRCOG,

The review process will commence in 2016, unless otherwise indicated.

