

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 > 35, large fibroids, hydramnios) should be referred for serial assessment of fetal size using ultrasound. ✓

Optimum method of diagnosing a SGA fetus and FGR

Fetal abdominal circumference (AC) or estimated fetal weight (EFW) < 10th centile can be used to diagnose a SGA fetus. A

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. C

Routine measurement of fetal AC or EFW in the third trimester does not reduce the incidence of a SGA neonate nor does it improve perinatal outcome. Routine fetal biometry is thus not justified. A

Change in AC or EFW may improve the prediction of wasting at birth (neonatal morphometric indicators) and adverse perinatal outcome suggestive of FGR. C

When using two measurements of AC or EFW to estimate growth velocity, they should be at least 3 weeks apart to minimise false–positive rates for diagnosing FGR. More frequent measurements of fetal size may be appropriate where birth weight prediction is relevant outside of the context of diagnosing SGA/FGR. C

Where the fetal AC or EFW is < 10th centile or there is evidence of reduced growth velocity, women should be offered serial assessment of fetal size and umbilical artery Doppler. ✓

Investigations that are indicated in SGA fetuses

Offer 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. C

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. C

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

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. C

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. C

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

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. A

Interventions to promote smoking cessation may prevent delivery of a SGA infant. The health benefits of smoking cessation indicate that these interventions should be offered to all women who are pregnant and smoke. A

Antithrombotic therapy appears to be a promising therapy for preventing delivery of a SGA infant in high-risk women. However there is insufficient evidence, especially concerning serious adverse effects, to recommend its use. D

Interventions to 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. C

Optimal method and frequency of fetal surveillance in SGA

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

When umbilical artery Doppler flow indices are normal it is reasonable to repeat surveillance every 14 days. B

More frequent Doppler surveillance may be appropriate in a severely SGA fetus. ✓

When umbilical artery Doppler flow indices are abnormal (pulsatility or resistance index > +2 SDs above mean for gestational age) and delivery is not indicated repeat surveillance twice weekly in fetuses with end–diastolic velocities present and daily in fetuses with absent/reversed end–diastolic frequencies. ✓

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

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

Biophysical profile should not be used for fetal surveillance in preterm SGA fetuses. A

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

methodology register, ACP journal club, DARE HTA, Maternity and Infant Care), EMBASE and TRIP were searched for relevant randomised controlled trials (RCTs), systematic reviews, meta–analyses and cohort studies. The search was restricted to articles published between 2002 and September 2011. Search words included 'fetal growth retardation', 'fetal growth restriction', 'infant, small for gestational age', including all relevant Medical Subject Heading (MeSH) terms. The search was limited to humans and the English language.

5. What are the risk factors for a SGA fetus/neonate? What is the optimum method of screening for the SGA fetus/neonate and care of "at risk" pregnancies?

Methods employed in the first and second trimesters, to predict the likelihood of a SGA fetus/neonate include: medical and obstetric history and examination, maternal serum screening and uterine artery Doppler. Methods of screening for the SGA fetus/neonate in the second and third trimester are abdominal palpation and measurement of symphysis fundal height (SFH) (including customised charts).

5.1 History

All women should be assessed at booking for risk factors for a SGA fetus/neonate to identify those who require increased surveillance.

Women who have a major risk factor (Odds Ratio [OR] > 2.0) should be referred for serial ultrasound measurement of fetal size and assessment of wellbeing with umbilical artery Doppler from 26–28 weeks of pregnancy (Appendix 1).

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]

There is insufficient evidence to determine how risk factors relate to each other in the individual woman and consequently how these risk factors should be managed. This includes abnormal maternal Down syndrome serum markers (see below). Further evidence may become available from the SCOPE study.[45] This guideline has therefore categorized risk factors into major and minor based on published ORs for the birth of a SGA neonate. Major risk factors (OR > 2.0) should prompt referral for serial ultrasound measurement of fetal size and assessment of wellbeing with umbilical artery Doppler. The presence of multiple minor risk factors is likely to constitute a significant risk for the birth of a SGA neonate and there is a rationale for further screening using uterine artery Doppler at 20 weeks (see below).

5.2 Biochemical markers used for Down Syndrome (DS) Screening

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).

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 > 35, large fibroids, hydramnios) should be referred for serial assessment of fetal size using ultrasound.

Cohort and case–control studies performed in low risk populations have consistently shown abdominal palpation to be of limited accuracy in the detection of a SGA neonate (sensitivity 19–21%, specificity 98%) and severely SGA neonate (< 2.3rd centile, sensitivity 28%).[65,66] In mixed risk populations, the sensitivity increases to 32–44%.[67,68] In high risk populations sensitivity is

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. C

Routine measurement of fetal AC or EFW in the third trimester does not reduce the incidence of a SGA neonate nor does it improve perinatal outcome. Routine fetal biometry is thus not justified. A

Change in AC or EFW may improve the prediction of wasting at birth (neonatal morphometric indicators) and adverse perinatal outcome suggestive of FGR. C

When using two measurements of AC or EFW to estimate growth velocity, they should be at least 3 weeks apart to minimise false–positive rates for diagnosing FGR. More frequent measurements of fetal size may be appropriate where birth weight prediction is relevant outside of the context of diagnosing SGA/FGR. C

Where the fetal AC or EFW is < 10th centile or there is evidence of reduced growth velocity, women should be offered serial assessment of fetal size and umbilical artery Doppler (see Section 7). ✓

6.1 Ultrasound biometry

Two systematic reviews have assessed the accuracy of ultrasound biometric measures, both as individual measures, as ratios, and combined (as the EFW).[3,79] Use of the 10th centile had better sensitivities and specificities than other commonly used centiles.[66] In a low risk population sensitivity varies from 0–10% and specificity 66–99% for any parameter. In a high risk population, fetal AC < 10th centile had sensitivity ranging from 72.9–94.5% and specificity 50.6–83.8%. For EFW < 10th centile, sensitivity was 33.3–89.2% and specificity 53.7–90.9%.[3,79] Meta–analysis was not performed in these systematic reviews due to the considerable clinical and methodological heterogeneity within the included papers. The potential advantage of EFW is that customised standards exist and accuracy can more easily be determined against birthweight.

A retrospective study has shown that among high risk patients, EFW and AC < 10th centile within 21 days of delivery better predicted a SGA infant than AC < 10th centile (80% versus 49%, OR 4.26, 95% CI 1.94–9.16).[80] Adverse perinatal outcome was also highest when both measures were < 10th centile.[80] Kayem et al.[81] found that measurement of AC in low risk women at term was a better predictor of birth weight ≤ 2.5 kg than a single measurement of SFH (LR+ 9.9 versus 7.1, LR– 0.5 versus 0.6). Evidence level 2++

Several studies have compared various formulae for estimating fetal weight in unselected patients. A prospective study compared 35 different formulae and found that most are relatively accurate at predicting birth weight up to 3500 g.[82] Another study found the Shepard and Aoki formulae to have the best intraclass correlation coefficient, with EFW showing the smallest mean difference from actual birth weight.[83] Although formulae have been developed for SGA fetuses, there is little evidence that accurate prediction of weight is substantially improved[84,85] and in this population the Hadlock formula[86] may be most appropriate to use. Evidence level 2–

There is no evidence to recommend one specific method of measuring AC (directly or derived from abdominal diameters) nor which centile chart to use. The centile charts produced by Chitty et al.[87] were optimally constructed and are widely used.

The same maternal characteristics (maternal height, weight, parity and ethnic group) that affect birth weight affect fetal biometric measures and fetal weight gain,[88,89] providing a rationale for the use of a customised AC or EFW chart.[9] A customised EFW < 10th centile is predictive of a SGA neonate (sensitivity 68%, specificity 89%).[90] Use of customised fetal weight centiles to define SGA Evidence level 3

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]

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

Interventions to promote smoking cessation may prevent delivery of a SGA infant. The health benefits of smoking cessation indicate that these interventions should be offered to all women who are pregnant and smoke.

Antithrombotic therapy appears to be a promising therapy for preventing delivery of a SGA infant in

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 0.50, 95% CI 0.32–0.81). The authors of the systematic review concluded there was not enough evidence to evaluate the benefits and risks of maternal oxygen therapy.[131]

A proportion of growth restricted fetuses will be delivered prematurely and consequently be at an increased risk of developing cerebral palsy. Maternally administered magnesium sulphate has a neuroprotective effect

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

10.1 Umbilical artery Doppler

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

When umbilical artery Doppler flow indices are normal it is reasonable to repeat surveillance every 14 days.

More frequent Doppler surveillance may be appropriate in a severely SGA fetus.

When umbilical artery Doppler flow indices are abnormal (pulsatility or resistance index >

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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+

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

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.

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?

Research may be required to evaluate the effectiveness of/determine:

- How combinations of risk factors for a SGA neonate (historical, biochemical and ultrasound) relate to each other in the individual woman.
- Interventions, specifically aspirin, in women classified as being at high risk of delivering a SGA neonate based on combined historical, biochemical, and ultrasound marker screening in the first trimester.
- Introducing customised SFH and EFW charts into clinical practice on substantive clinical endpoints (perinatal mortality/morbidity and service utilisation).
- Routine third trimester ultrasound assessment of fetal size combined with umbilical artery Doppler on substantive clinical endpoints (perinatal mortality/morbidity and service utilisation).
- Oxygen therapy in severe early–onset SGA foetuses associated with umbilical artery AREDV on substantive clinical endpoints (perinatal mortality/morbidity and service utilisation).
- Optimal frequency and content of fetal surveillance in SGA fetuses with both a normal umbilical artery Doppler and also an abnormal umbilical artery Doppler but with end–diastolic frequencies present.
- Measuring amniotic fluid volume and MCA Doppler in the near term SGA fetuses with a normal umbilical artery Doppler on substantive clinical endpoints (perinatal morbidity and service utilisation).
- Potential health economic benefit of investment in maternity services to provide recommendations in this guideline and future health outcomes of the children.

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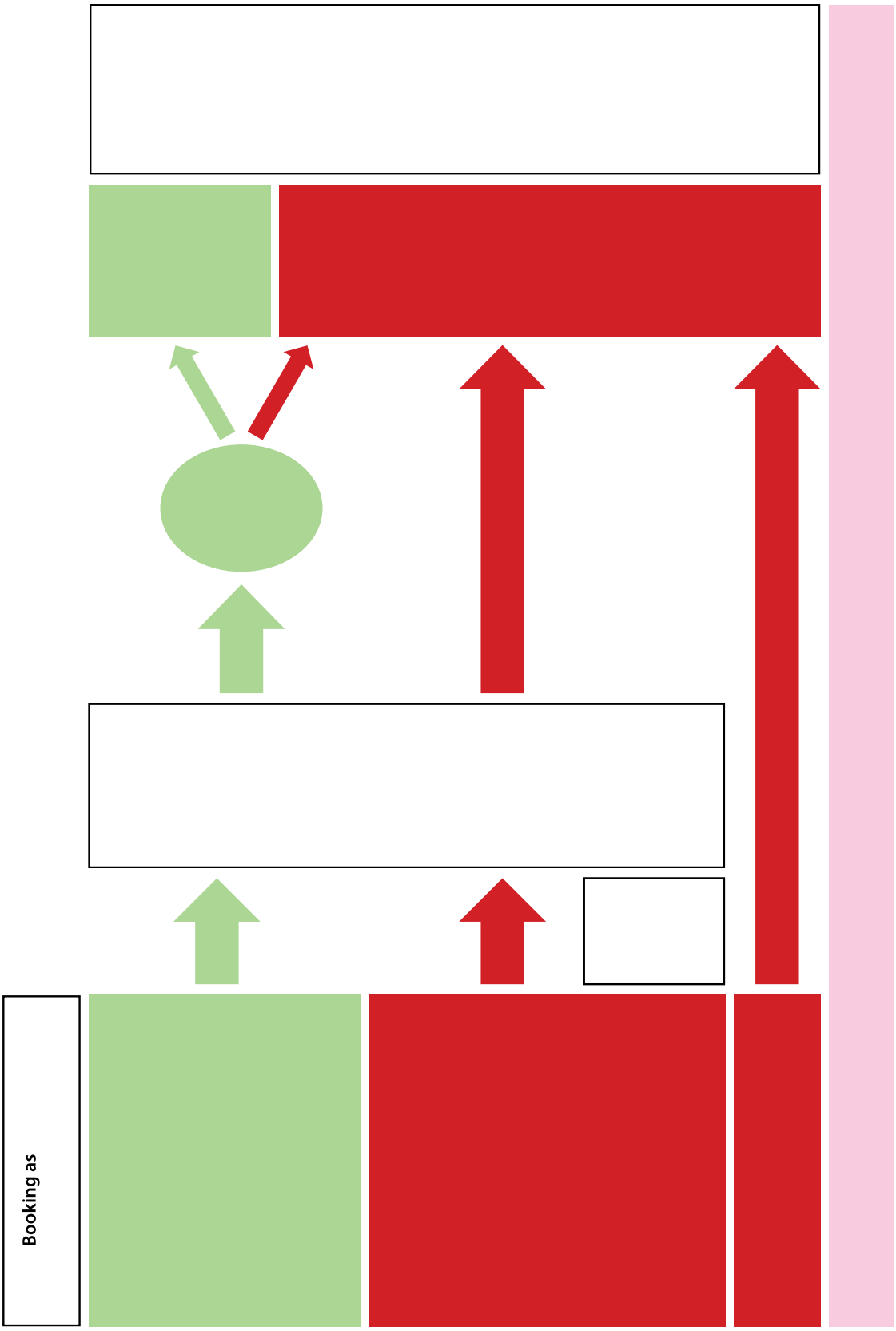
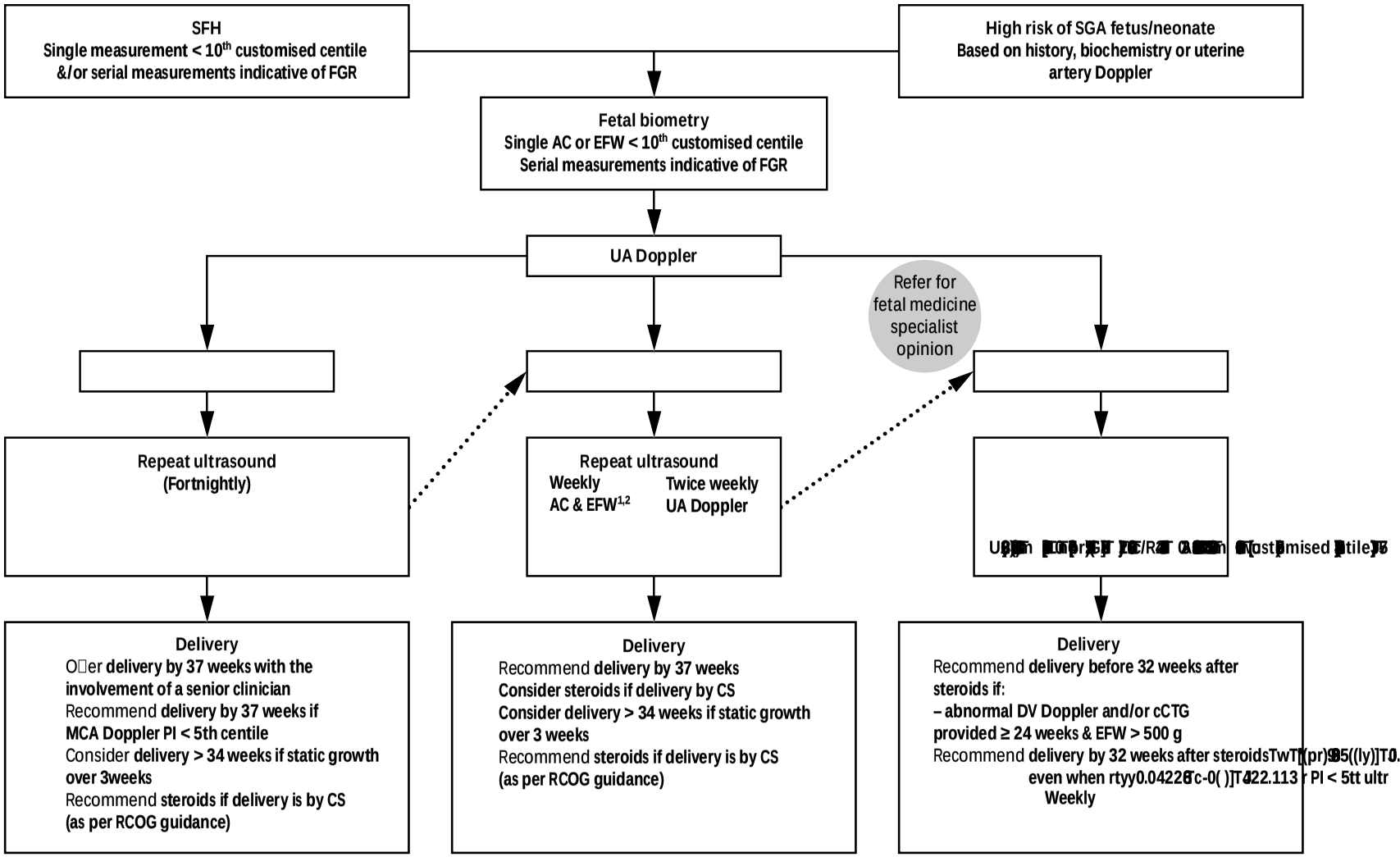
Appendix I: Summary of Risk Factors for a Small–for–Gestational–Age Neonate.

Table A:





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 http://www.rcog.org.uk/green-top-development). These recommendations are not intended to dictate an exclusive course of management or treatment. They must be evaluated with reference to individual patient needs, resources and limitations unique to the institution and variations in local populations. It is hoped that this process of local ownership will help to incorporate these guidelines into routine practice. Attention is drawn to areas of clinical uncertainty where further research might be indicated.**

The evidence used in this guideline was graded using the scheme below and the recommendations formulated in a similar fashion with a standardised grading scheme.

Classification of evidence	
1++	High-quality meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a very low risk of bias
1+	Well-conducted meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a low risk of bias
1–	Meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a high risk of bias
2++	High-quality systematic reviews of case–control or cohort studies or high-quality case–control or cohort studies with a very low risk of confounding, bias or chance and a high probability that the relationship is causal
2+	Well-conducted case–control or cohort studies with a low risk of confounding, bias or chance and a moderate probability that the relationship is causal
2-	Case–control or cohort studies with a high risk of confounding, bias or chance and a significant risk that the relationship is not causal
3	Non-analytical studies, e.g. case reports, case series
4	Expert opinion

Grades of recommendations	
A	At least one meta-analysis, systematic review or randomised controlled trial rated as 1++ and directly applicable to the target population; or A systematic review of randomised controlled trials or a body of evidence consisting principally of studies rated as 1+ directly applicable to the target population and demonstrating overall consistency of results
B	A body of evidence including studies rated as 2++ directly applicable to the target population, and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 1++ or 1+
C	A body of evidence including studies rated as 2+ directly applicable to the target population and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 2++
D	Evidence level 3 or 4; or Extrapolated evidence from studies rated as 2+
Good practice points	
✓	Recommended best practice based on the clinical experience of the guideline development group