Assessment of Small for Gestational Age fetus

Rami Kilani

Importance

- 40% of Unexplained stillbirths
- 30% of sudden infant death syndrome (SIDS)
- 8 fold increased risk of Infant mortality
- High risk of prenatal hypoxia, acidaemia (2 fold), operative delivery and HIE(4-6 fold)
- Risks of prematurity
- Barkers hypothesis-adult metabolic syndrome

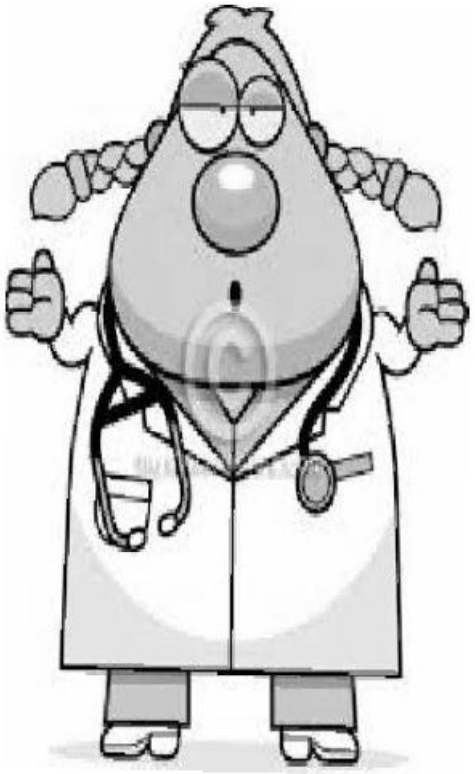
Perinatal outcome and later implications of intrauterine growth restriction. Pallotto EK, Kilbride HW Clin Obstet Gynaecol. 2006 Jun;49(2):257-69.

Dilemmas !

Terminology – SGA/FGR/IUGR?

How to monitor ?

When to deliver?



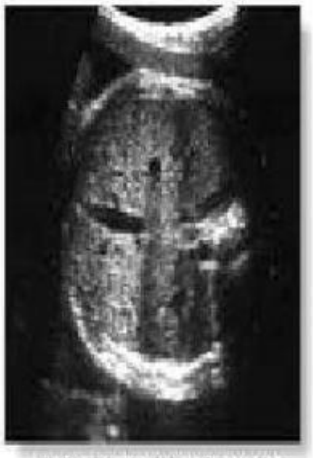
Definition

* AC/EFW

  * Defining threshold :

    < 10th centile for gestational age

    < 5th centile for gestational age (worse outcome)



abdominal circumference

Phases of fetal growth

First 16 weeks: mostly cellular hyperplasia

16-32 weeks: both hyperplasia and hypertrophy

>32 weeks: mostly hypertrophy

Thus: early growth restriction will affect cell numbers and have a global (symmetrical) effect. Later cell size will be affected.

Factors influencing intrauterine growth

Altitude Race (Pygmy=2.64kg

USA Amerindian=3.6kg)

Multiple gestation Gender

Socioeconomic level Smoking, alcohol

* Maternal Pathology
* Fetal (genetic disorders, infections)
* Placental

Early I.U.G.R.

Triploidy and Tri 18: very early and severe.

Tri 13: less severe

Tri 21; no IUGR but short femur and humerus

Cardiac malformations

Early intrauterine infection: early SAB, IUFD

fetal hydrops, IUGR

Symmetric I.U.G.R.

1/3 of all cases

Fetus is proportionally small
(HC, AC, FL)

Diagnosed early

Early insult affecting cell number (intrauterine infection, karyotype anomaly, genetic syndrome)

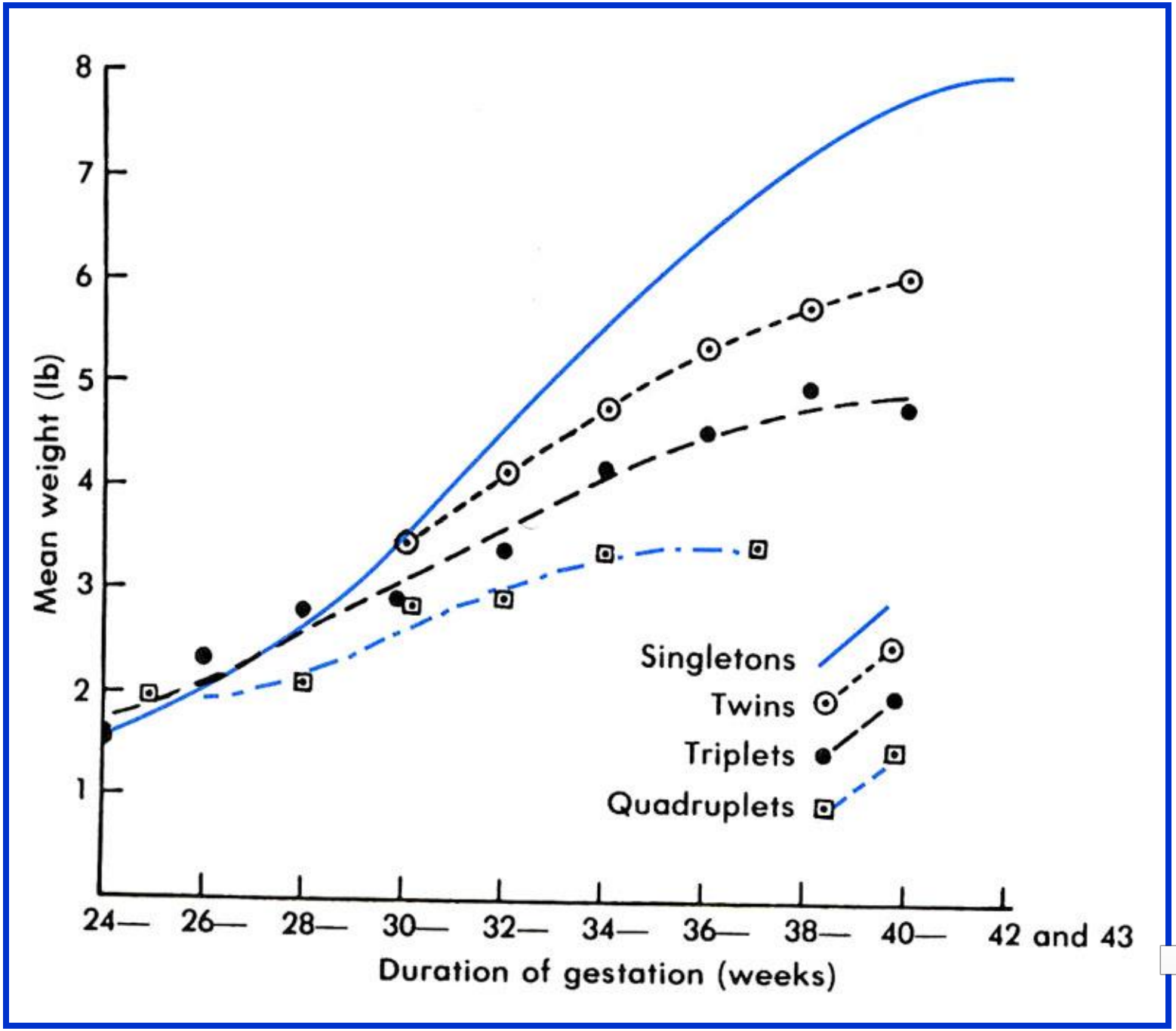
Asymmetric I.U.G.R.

"nutritional"

Placental insufficiency, late $2^{nd}/3^{rd}$ trimesters

Slow AC growth vs normal HC and FL

(glycogen utilization by liver, liver shrinkage, decreased AC; preferential shunting to brain thus maintained HC)


Mean weight (lb)
8
7
6
5
4
3
2
1
Singletons
Twins
Triplets
Quadruplets
24—
26—
28—
30—
32—
34—
36—
38—
40—
42 and 43
Duration of gestation (weeks)

Fetoplacental Etiologies of Fetal Growth Retardation

- Chromosomal abnormalities
  - Trisomies (13, 18, 21)
  - Trisomy 9 mosaicism
  - Trisomy 4p
  - 4p−, 5p−, 11p−, 13q− syndromes
  - Partial trisomy 10q
- Genetic syndromes
  - Cretinism (hypothyroidism)
  - Russell-Silver
  - Bloom's
  - Lowe's
  - De Lange's
  - Progeria
  - Leprechaunism
- Congenital malformations
- Infectious diseases
  - Cytomegalovirus
  - Toxoplasmosis
  - Rubella
- Placental pathology
  - Previa
  - Abruption
  - Circumvallate
  - Mosaicism
  - Infarctions
  - Twins

Risk Factors for Fetal Growth Restriction: Indications for Ultrasound

- History of fetal growth restriction
- Hypertension
- Diabetes mellitus
- Elevated MSAFP/hCG
- Antiphospholipid syndrome
- Chronic medical illnesses
- Low maternal prepregnancy weight (<90% IBW)
- Poor maternal weight gain
- Twin gestation
- Substance abuse (tobacco, alcohol, drugs)
- Preterm labor
- Abnormalities of placentation
- Vaginal bleeding
- Maternal anemia (Hgb < 10)
- Maternal hypoxia (cyanotic cardiac or pulmonary disease, altitude)
- Maternal hemoglobinopathies
- Drug ingestion (hydantoin, coumarin)

I.U.G.R. – diagnosis (2)

Ultrasound:

1. BPD, HC, AC, FL, transcerebellar distance, cheek-to-cheek diameter, HC:AC ratio, FL:AC ratio, EFW.

2. Serial measurements (not less than 2 weeks interval)

3. Oligohydramnios

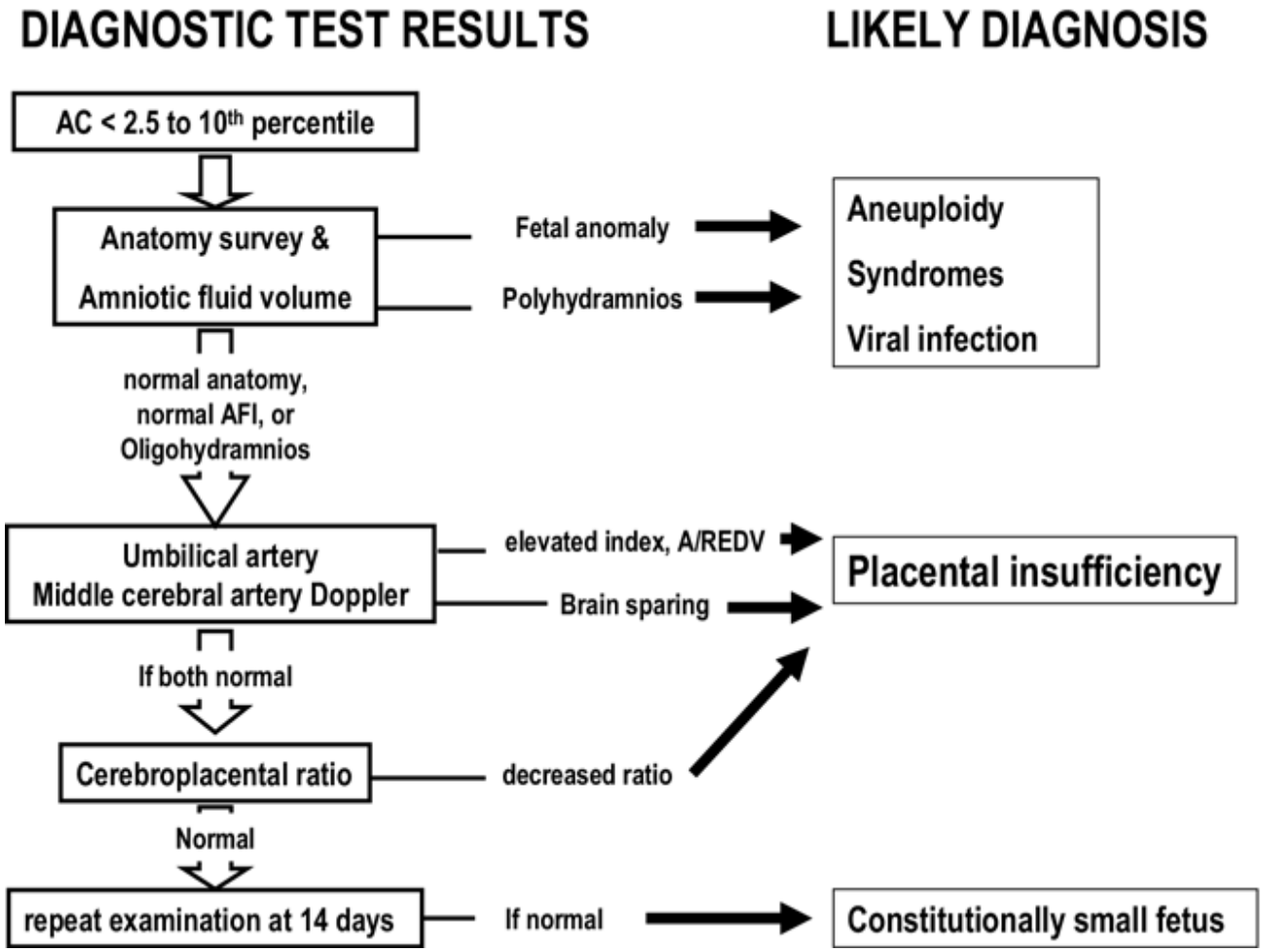
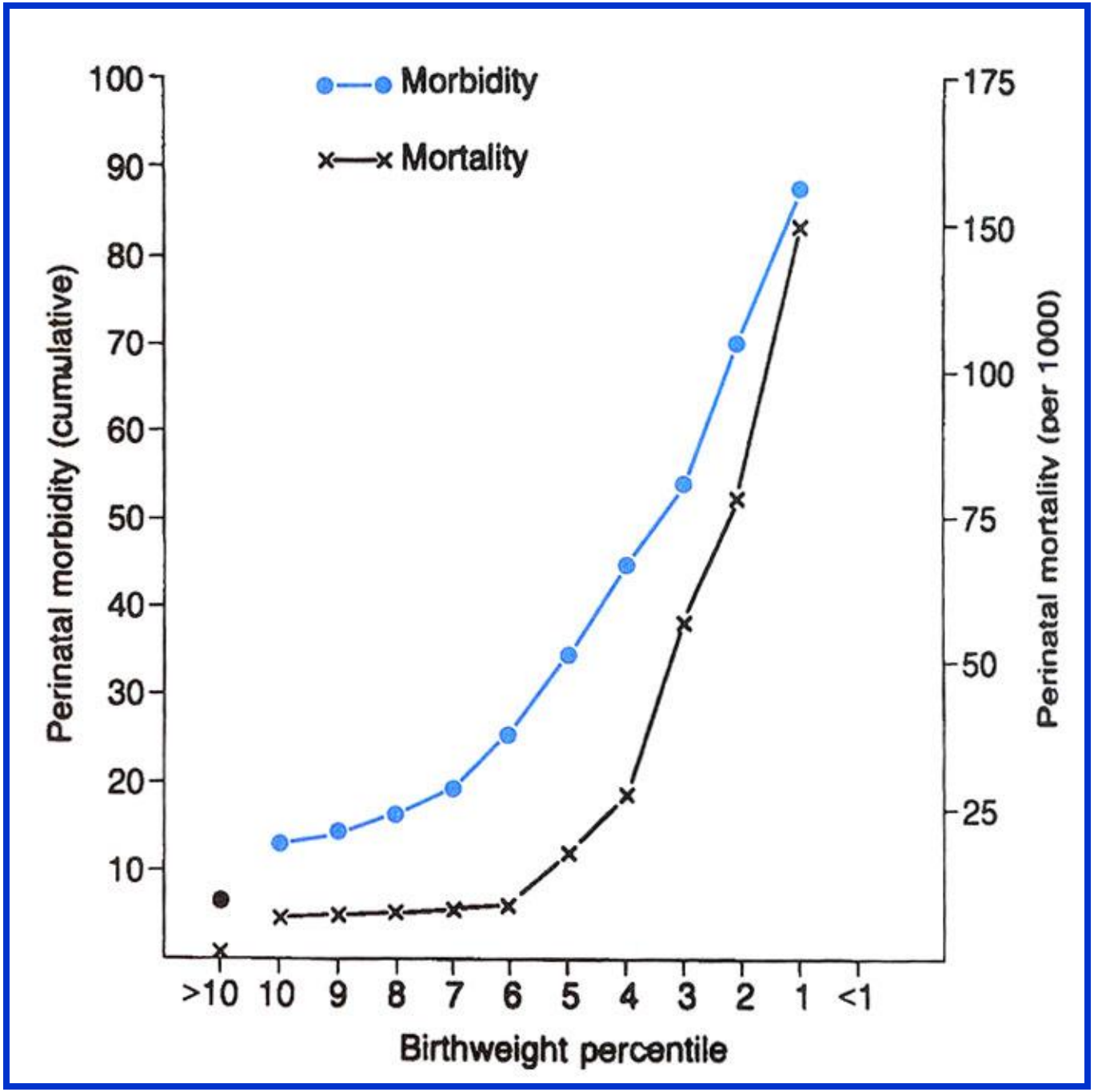


Figure 3 This figure displays a decision tree following the evaluation of fetal anatomy, amniotic fluid volume, umbilical and middle cerebral artery Doppler. The most likely clinical diagnosis based on the test results is presented on the right hand side. A high index of suspicion for aneuploidy, viral, and nonaneuploid syndrome needs to be maintained at all times. (Reproduced with permission: Baschat A: Intrauterine growth restriction, in Gabbe SG (ed): Obstetrics: Normal and Problem Pregnancies (ed 5). Philadelphia, PA, Churchill Livingstone, 2007, pp 771-814.)

I.U.G.R. – clinical significance

Increased Perinatal morbidity and mortality (x10)

↑ Fetal distress, stillbirth, neonatal hypoglycemia, polycythemia, meconium aspiration, hypocalcemia


Morbidity
Mortality
Perinatal morbidity (cumulative)
Perinatal mortality (per 1000)
100
90
80
70
60
50
40
30
20
10
175
150
100
75
50
25
>10
10
9
8
7
6
5
4
3
2
1
<1
Birthweight percentile

CHROMOSOMAL ABNORMALITIES AND IUGR

Ultrasound Findings Present			
IUGR	Anomaly	Hydramnios	**Abnormal Karyotype**
X			12/180 (7%)
X	X		18/57 (32%)
X		X	6/22 (27%)
X	X	X	7/15 (47%)

I.U.G.R.

Management

Antepartum fetal testing

- Doppler ultrasound assessment of fetal blood flow
- Venous circulation
- Cordocentesis

Early IUGR - evaluation

Detailed ultrasound

Amniocentesis (karyotyping, TORCH)

Follow-up every 2 weeks

Antenatal testing at viability (kick count, AFV, NST, BPP, Doppler studies)

I.U.G.R. - surveillance

Fetal kick count

NST

AFI

Biophysical profile (+ modified BPP)

Doppler

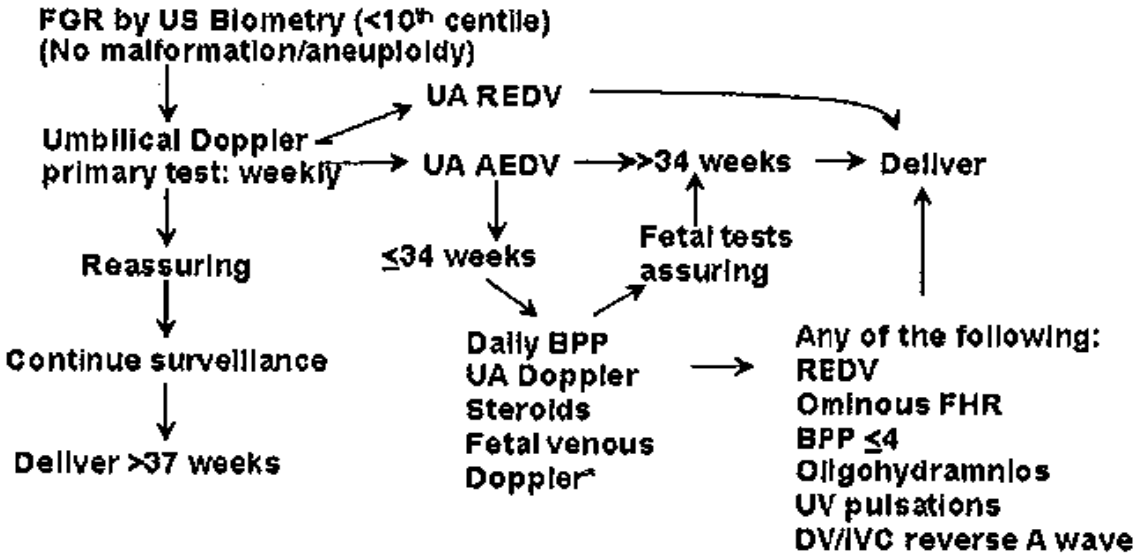


FIGURE 3. FGR: management guideline flow chart. On the basis of the best available evidence, the approach graphically depicted here uses umbilical artery (UA) Doppler sonography as the primary surveillance tool supplemented by biophysical profile (BPP) and other fetal monitoring modalities. ADEV indicates absent end-diastolic flow; DV, ductus venosus; IVC, inferior vena cava; REDV, reverse end-diastolic flow; UV, umbilical vein.

Dev Maulik, Management of Fetal Growth Restriction: An Evidence Based Approach
Clinical Obstetrics and Gynecology, Vol 49, June 2006

I.U.G.R.

Antepartum Therapy

- Maternal hyperoxia (Nicolaides et al 1987) (Battraglia et al 1992)
- Low dose aspirin

  Leitich et al 1997: Meta analysis of 13 randomized studies 13, 234 women decrease I.U.G.R. and pre-eclampsia

  Brazilian report (ECPPA 1996): no ameliorating effect on I.U.G.R.

When to Deliver ?

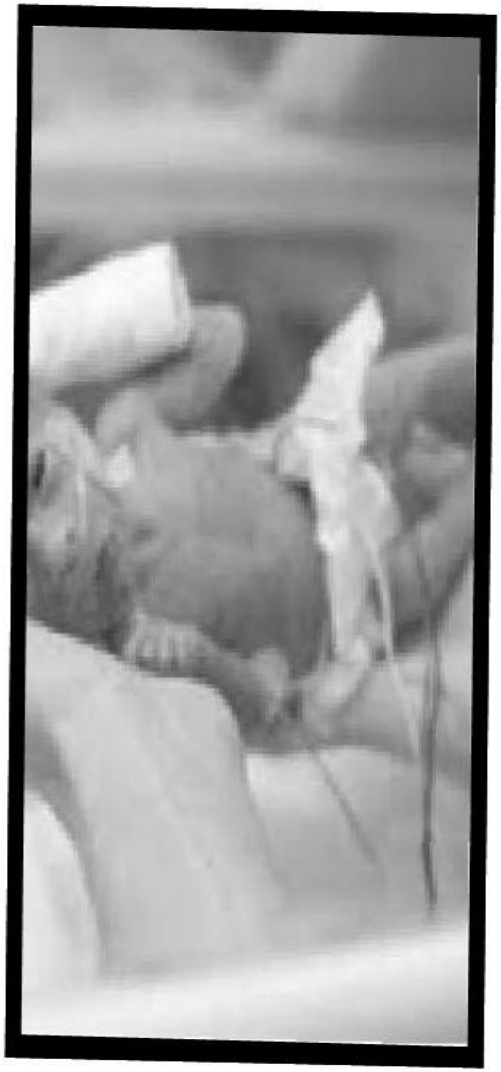
"I AM A FETUS IN THE WOMB

I FEAR IT MAY BECOME MY TOMB

IF ONLY I COULD GIVE A SHOUT

TO MAKE MY DOCTOR GET ME OUT!"

UNKNOWN MEDICAL STUDENT
DUBLIN, UK 1982



I.U.G.R.

Management

Timing of delivery

- Fetal lungs maternity achieved
- Absence or reverse end diastolic flow velocity of umbilical artery wave-form (Karsdorp 1994, Valacmonico et al 1994)
- In case of preterm I.U.G.R. decision should be based on maternal health, fetal function tests, biochemical test of fetal lung maturity

I.U.G.R.
The role of Doppler (1)

Umbilical artery

Uterine artery

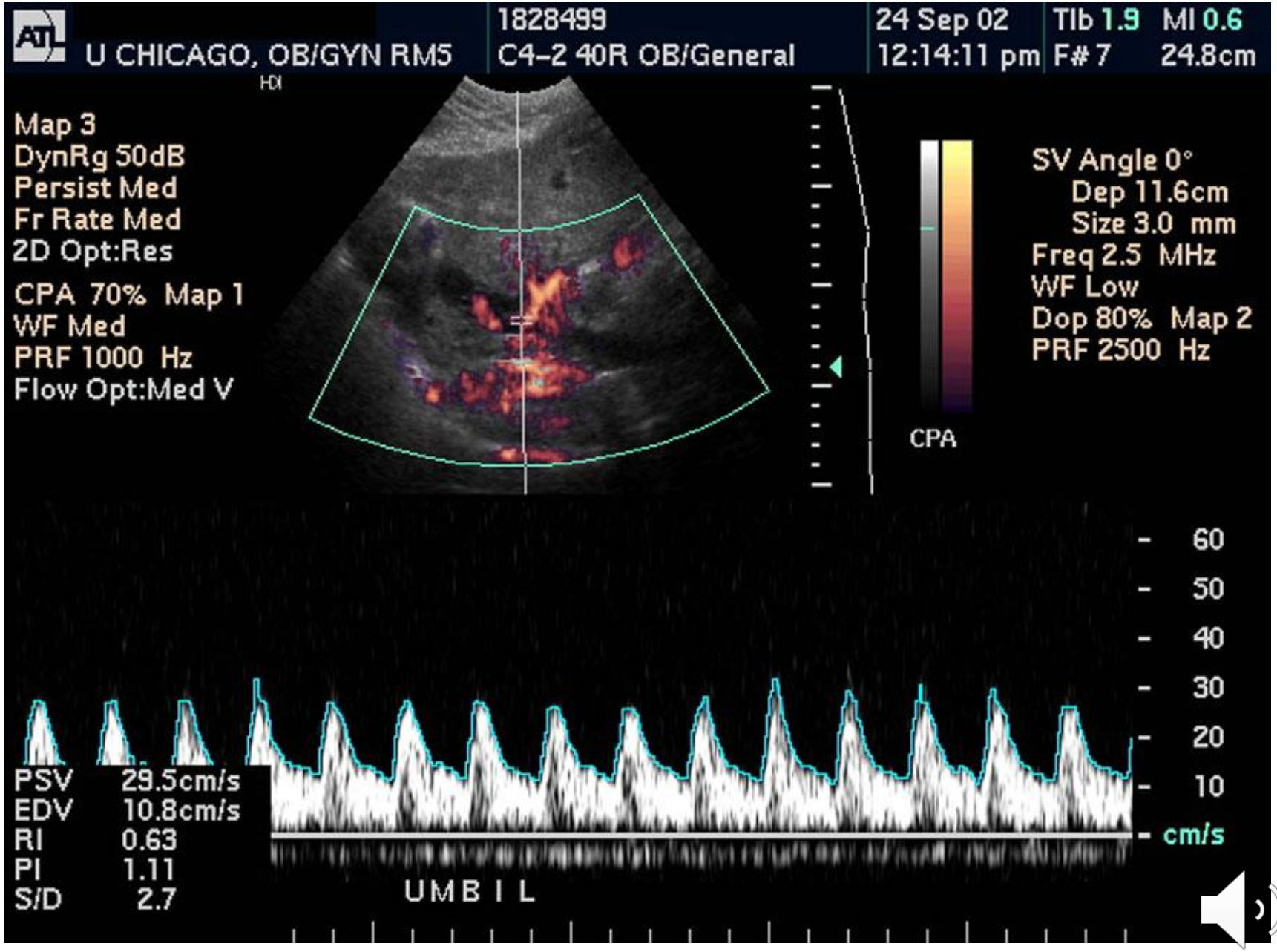
Middle cerebral artery

Ductus venosus

I.U.G.R.
The role of Doppler (2)

Umbilical a: association between high resistance and IUGR (Trudinger, 1985) and placental histology (Voigt, 1992)

Normal: S/D=4 @ 20 w., 3 @ 30 w., 2 @ 40 w.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1828499
C4-2 40R OB/General
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12:14:11 pm
TIb 1.9
MI 0.6
F# 7
24.8cm
HDI
Map 3
DynRg 50dB
Persist Med
Fr Rate Med
2D Opt:Res
CPA 70% Map 1
WF Med
PRF 1000 Hz
Flow Opt:Med V
CPA
SV Angle 0°
Dep 11.6cm
Size 3.0 mm
Freq 2.5 MHz
WF Low
Dop 80% Map 2
PRF 2500 Hz
60
50
40
30
20
10
cm/s
PSV 29.5cm/s
EDV 10.8cm/s
RI 0.63
PI 1.11
S/D 2.7
UMBIL

I.U.G.R.
The role of Doppler (3)

Umbilical a: progression is from normal end-diastolic velocity (EDV) to decreased, to absent to reverse EDV.

PPV in precipitating antenatal asphyxia = 92% (Donner, 1995)

Precedes FHR anomalies by 3 days

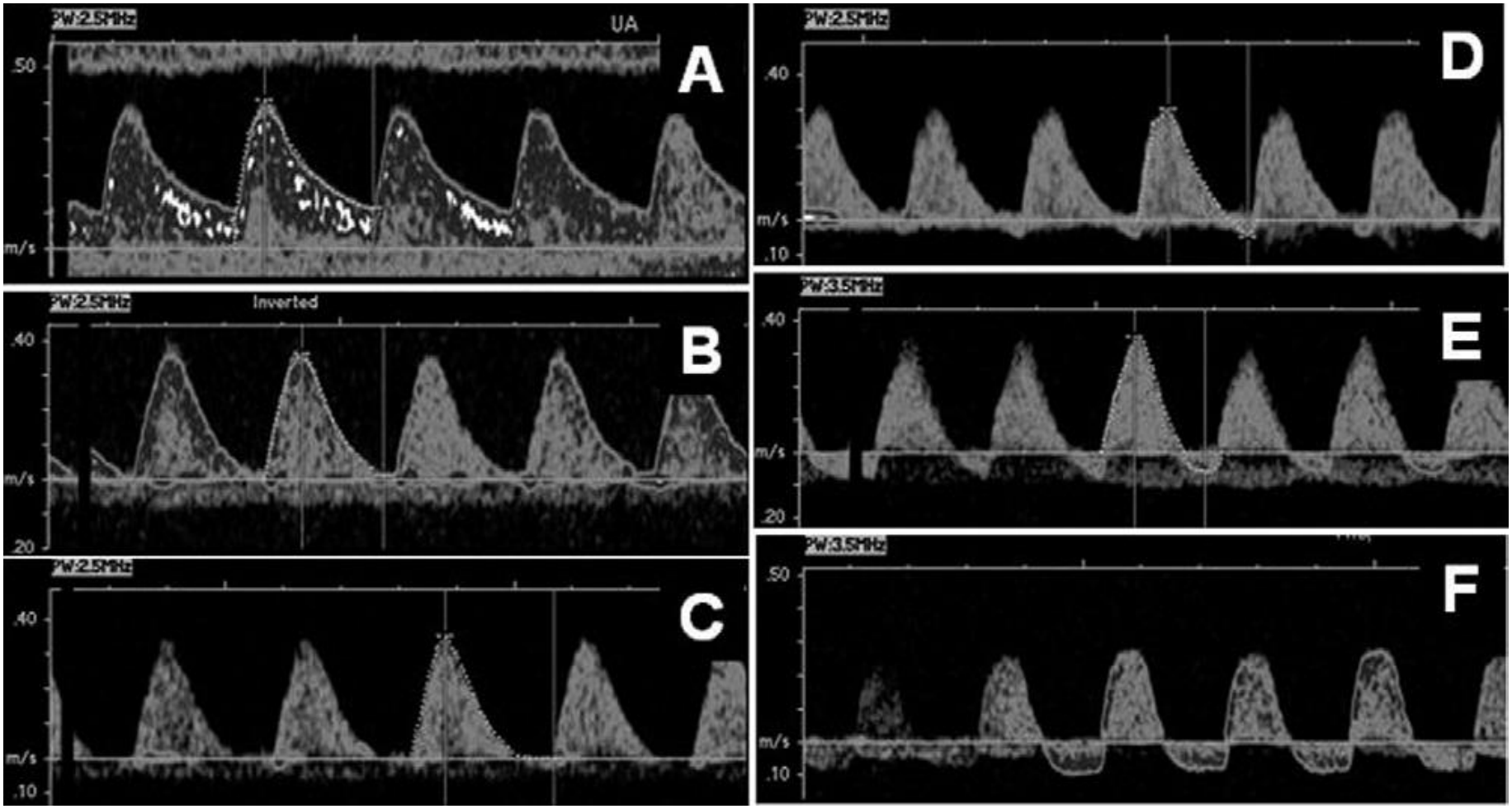


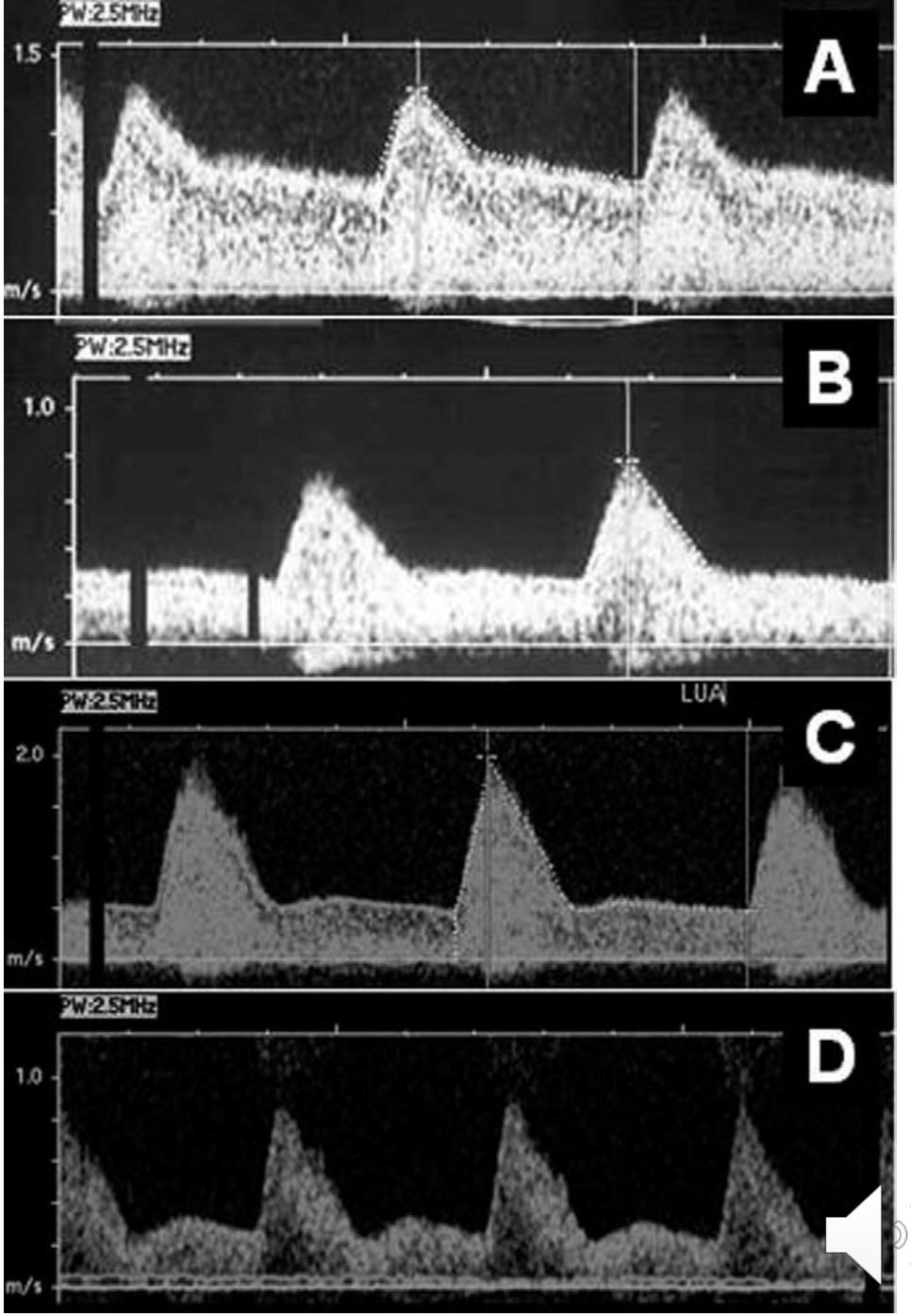
Figure 2 The normal umbilical artery flow velocity waveform has marked positive end-diastolic velocity that increases in proportion to systole toward term (A). Moderate abnormalities in the villous vascular structure raise the blood flow resistance and are associated with a decline in end-diastolic velocities (B). When a significant proportion of the villous vascular tree is abnormal (50-70%), end-diastolic velocities may be absent (C) or even reversed (D). Depending on the magnitude of placental blood flow resistance and the fetal cardiac function, reversal of end-diastolic velocities may be minimal (D), moderate (E), or severe (F). In the latter case precordial venous flows were universally abnormal. (Reprinted with permission.[5])

I.U.G.R
The role of Doppler (4)

Uterine a. :by 20 weeks, low-impedance waveform and no diastolic notch. If still high impedance and diastolic notch, high risk of IUGR and/or preeclampsia. As screening test, 82% sensitivity, 38% specificity (Bewley, 1991)

Value in high-risk patients only.

Figure 1 Flow velocity waveforms obtained from the uterine artery beyond 24 weeks gestation. In the first patient (A) high-volume diastolic flow is established, indicating successful trophoblast invasion. Elevated placental vascular resistance is associated with a decline in diastolic velocities and a subsequent rise in the Doppler index (B). Persistence of an early diastolic notch in the uterine artery flow velocity waveform is evidence of increased spiral artery blood flow resistance. Frequently "notching" is more subtle beyond 32 weeks (C) than in the late second or early third trimesters (D). (Reprinted with permission.36)



I.U.G.R
The role of Doppler (5)

Middle cerebral a. (MCA) : ↓ resistance and ↑ velocity (blood redistribution, brain sparing). Late: ↑ resistance, ? Due to cerebral edema (Hecher, 1995)

Indices: velocity (angle dependent), PI, RI.

PI=2 @ 20w, 2 @ 30w., 1.5 @ 36 w, 1.25 @ 40 w

Fetal Thoracic aorta:

Normal: PI=1.8 → 2.2 from 20 to 40 weeks

In IUGR, increased resistance

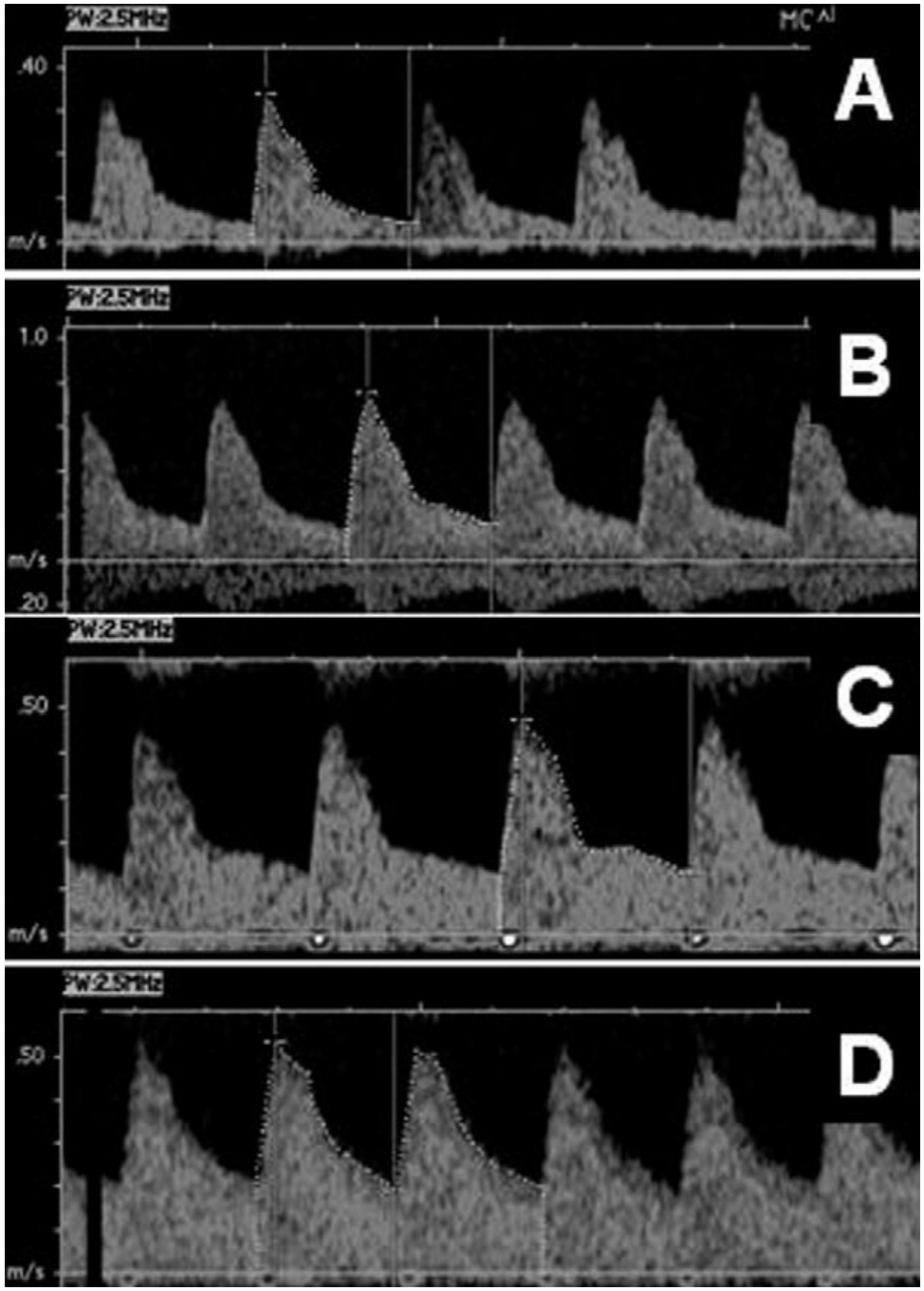
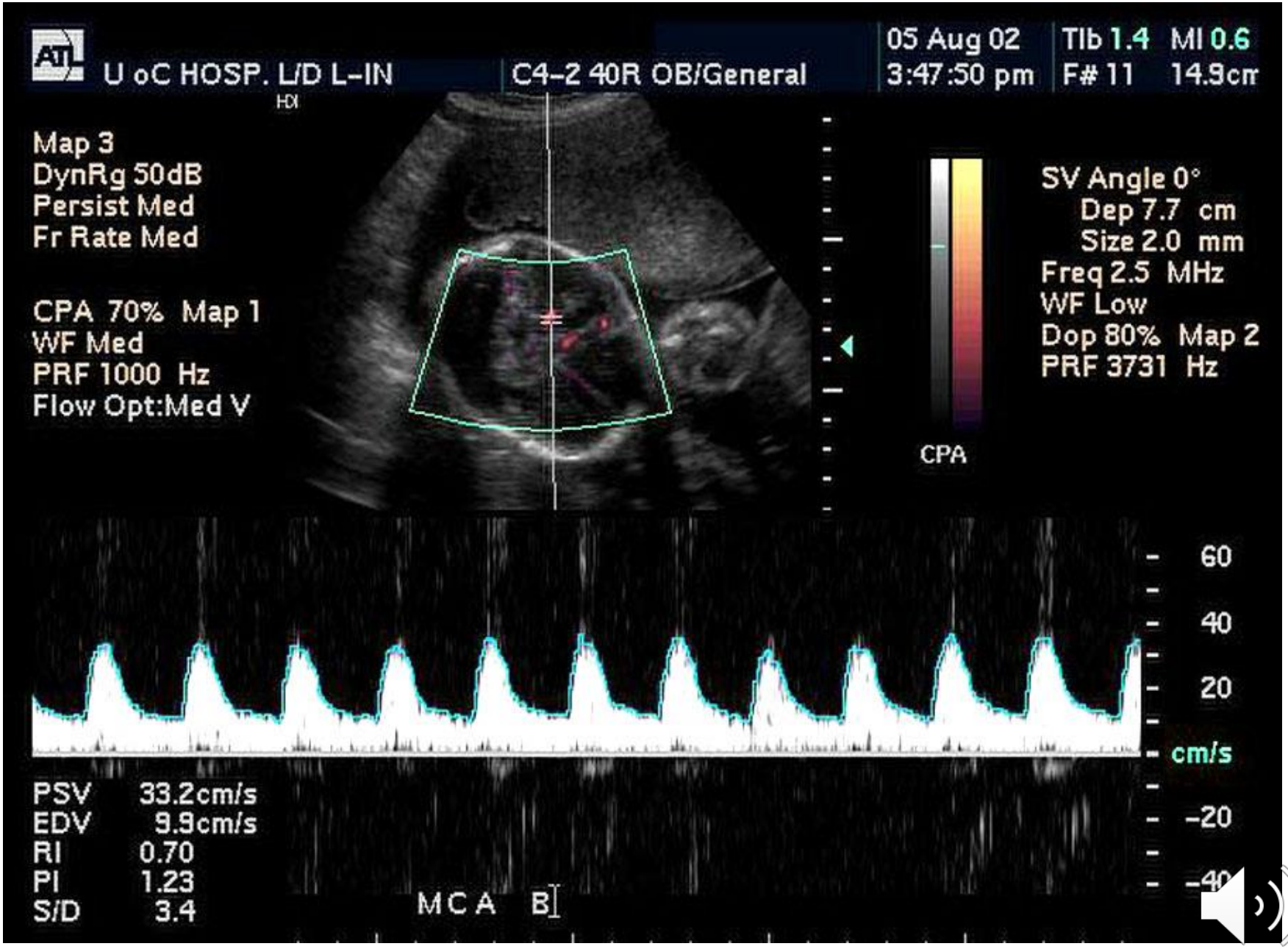


Figure 3 The normal middle cerebral artery flow pattern has relatively little diastolic flow (A). With elevation of placental blood flow resistance the changes in the middle cerebral artery waveform may be subtle, although the cerebroplacental ratio may become abnormal as in fetus B. With progressive placental dysfunction there may be an increase in the diastolic velocity, resulting in a decrease in the Doppler index (Brain sparing, C).With marked brain sparing, the systolic down slope of the waveform becomes smoother so that the waveform almost resembles that of the umbilical artery (D). The associated rise in the mean velocity results in a marked decline in the Doppler index. (Reprinted with permission.36)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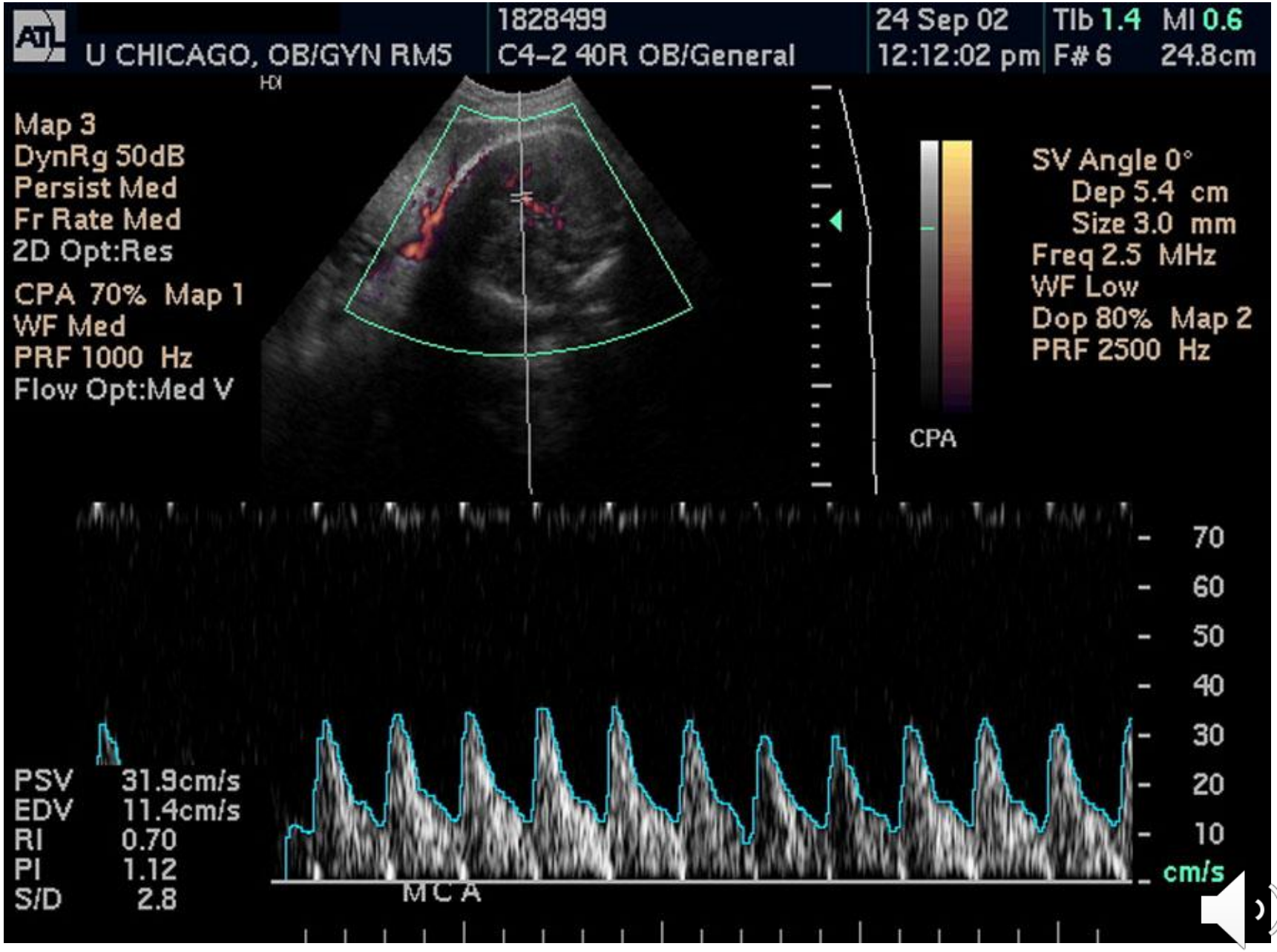
C4-2 40R OB/General
05 Aug 02
3:47:50 pm
TIb 1.4
MI 0.6
F# 11
14.9cm
HDI
Map 3
DynRg 50dB
Persist Med
Fr Rate Med
CPA 70% Map 1
WF Med
PRF 1000 Hz
Flow Opt:Med V
SV Angle 0°
Dep 7.7 cm
Size 2.0 mm
Freq 2.5 MHz
WF Low
Dop 80% Map 2
PRF 3731 Hz
CPA
60
40
20
cm/s
-20
-40
PSV 33.2cm/s
EDV 9.9cm/s
RI 0.70
PI 1.23
S/D 3.4
MCA B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C4-2 40R OB/General
24 Sep 02
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TIb 1.4
MI 0.6
F# 6
24.8cm
HDI
Map 3
DynRg 50dB
Persist Med
Fr Rate Med
2D Opt:Res
CPA 70% Map 1
WF Med
PRF 1000 Hz
Flow Opt:Med V
SV Angle 0°
Dep 5.4 cm
Size 3.0 mm
Freq 2.5 MHz
WF Low
Dop 80% Map 2
PRF 2500 Hz
CPA
70
60
50
40
30
20
10
cm/s
PSV 31.9cm/s
EDV 11.4cm/s
RI 0.70
PI 1.12
S/D 2.8
MCA

IUGR
The role of Doppler (6)

Ductus venosus: normal is forward flow during atrial contraction. IUGR causes ↑ peripheral resistance and eventually rt heart failure with no forward flow. When back pressure reaches umbilical vein, normally minimally pulsatile waveform becomes pulsatile as sign of imminent demise.

Abnormal values associated with 8.2 RR, and pulsations with 18 RR of Perinatal mortality (Gramellini, 2001)

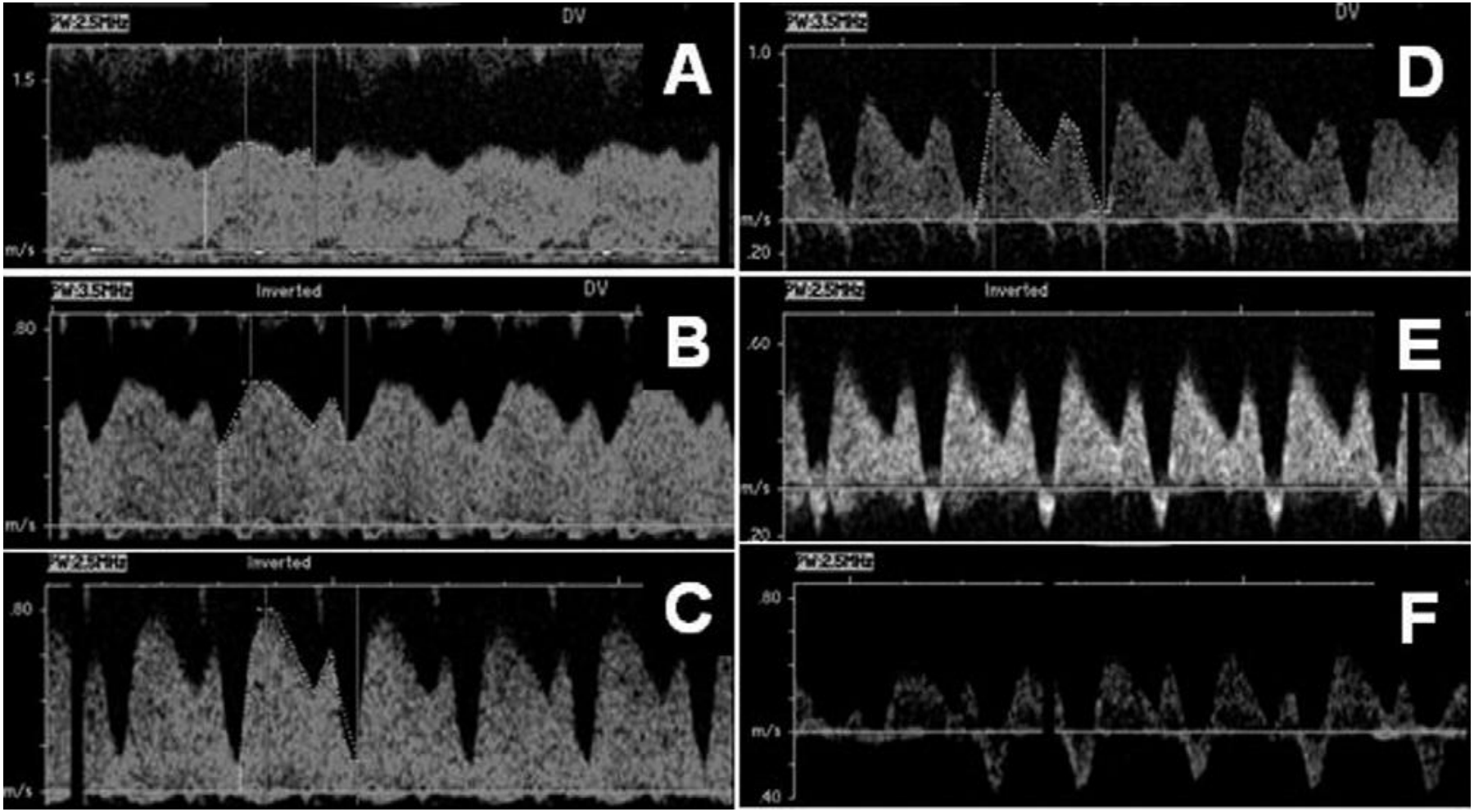


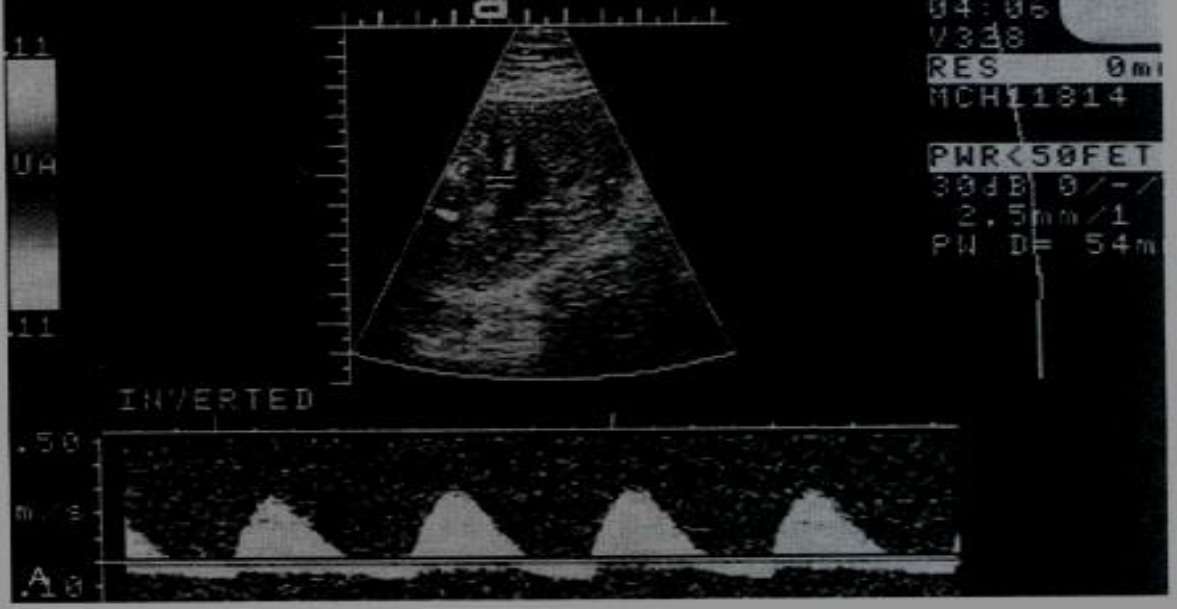
Figure 4 In the ductus venosus blood flow is always antegrade throughout the cardiac cycle under normal circumstances. Pulsatility is less pronounced in waveform patterns obtained at the inlet (A) versus the outlet (B). With impaired cardiac forward function there is a decline in forward flow during atrial systole (C). If progressive atrial forward flow may be lost (D) or reversed (E, F). (Reprinted with permission.36)

I.U.G.R.

Management

Intrapartum

- Adequate oxygenation
- Continuous fetal monitor with scalp electrode
- Cesarean section for deteriorating fetal condition, uncomfortable cervix
- OB- Ped Team approach to decrease of meconium aspiration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11
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.50
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A 0
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V338
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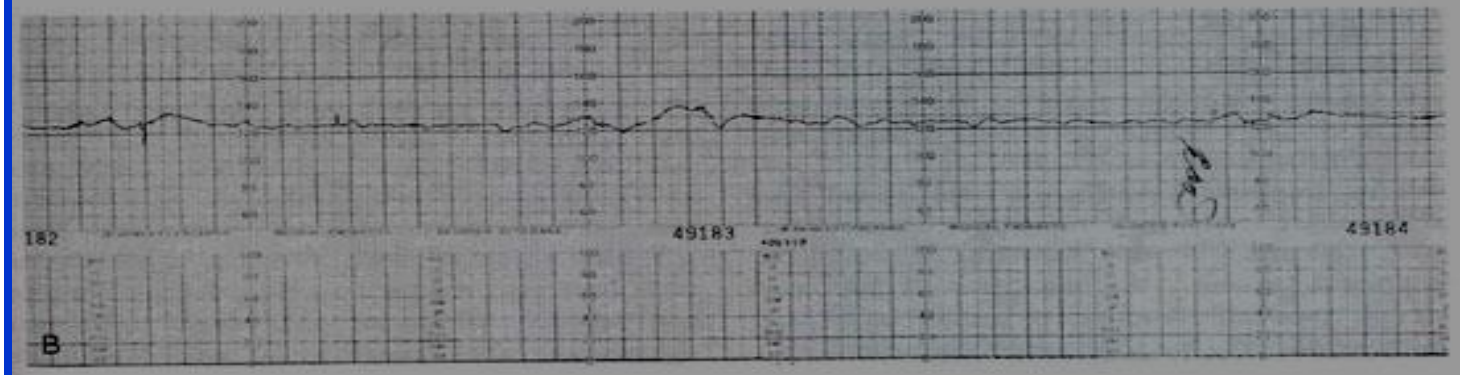
PW D= 54m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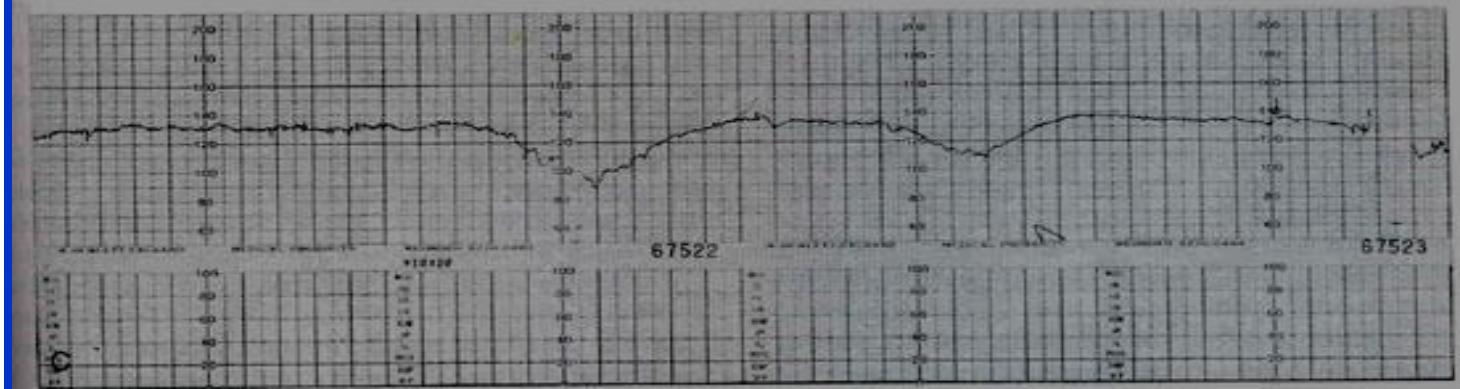
49184
B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C