ABNORMAL CTG

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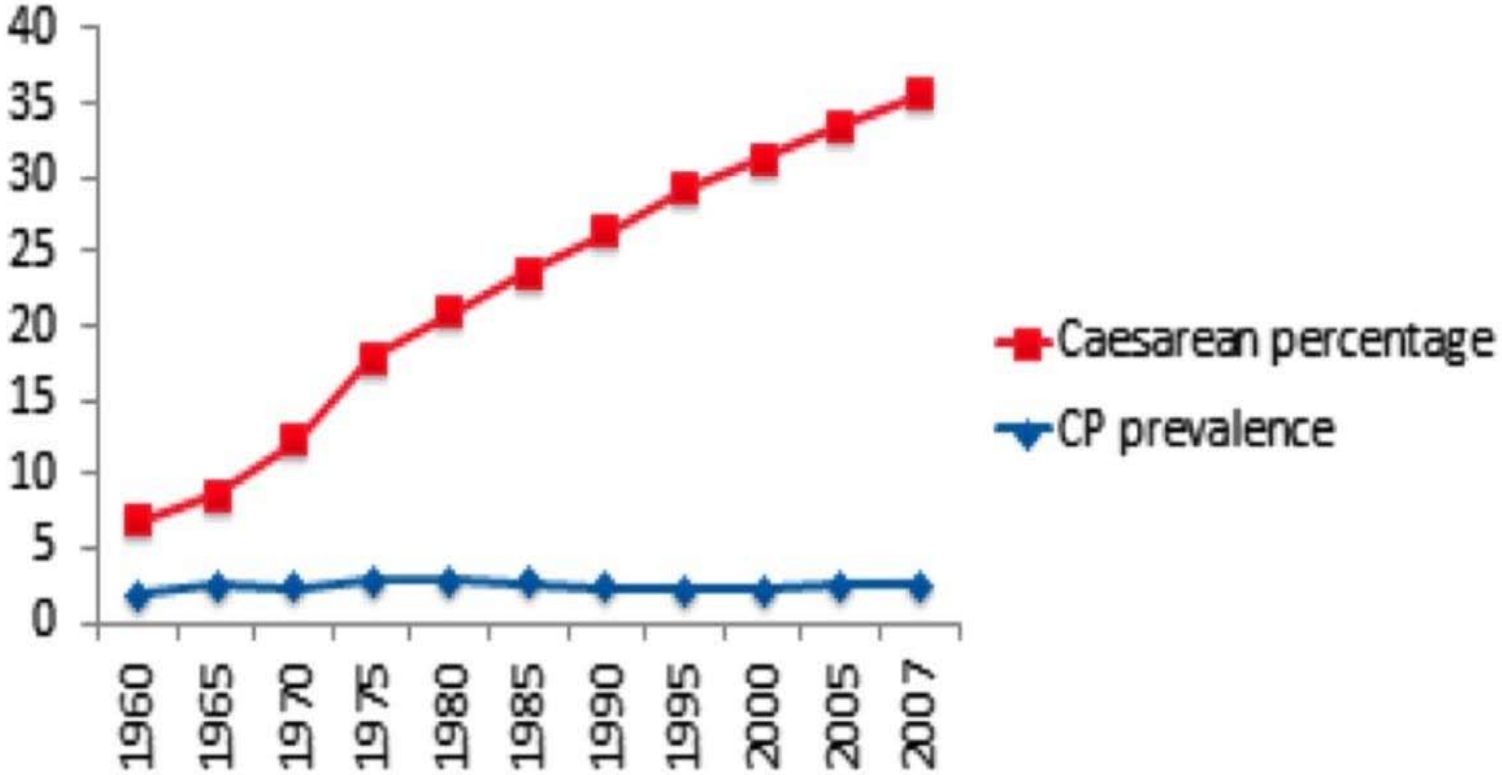
Esfand 1934

- CTG monitoring should never be regarded as a substitute for good clinical observation and judgement, or as an excuse for leaving the mother unattended during labour

- CTG not only has a 60% false-positive rate , but also has a high inter & intra-observer variability

FIGURE 1

Six-fold increase in cesarean delivery without change in CP prevalence



Prevalence of CP/1000 births compared with cesarean rates over the past 50 years.[68]

CP, cerebral palsy.

MacLennan. Cerebral palsies: new insights and causes. Am J Obstet Gynecol 2015.

Intrapartum indications for CTG

Risk factor	Potential physiological basis
Abnormal auscultation or CTG	Inability to assess compromise by auscultation / potential fetal compromise
Induction of labour with prostaglandin / oxytocin	Uterine hyperstimulation / fetal asphyxia
Oxytocic augmentation	Uterine hyperstimulation / fetal asphyxia
Regional anaesthesia i.e. epidural or spinal, paracervical block (pre epidural CTG should be considered)	Maternal hypotension, fetal hypoxia, CTG changes,
Abnormal vaginal bleeding in labour	Reduced placental function
Maternal pyrexia ≥ 38°C	Increased fetal O_2 requirements
Meconium or blood stained liquor	Prior fetal compromise
Absent liquor following amniotomy	Reduced placental function / ↑ cord compression / ↓ fetal reserves
Prolonged first stage, defined by referral guidelines	Reducing fetal reserves
Prolonged second stage, defined by referral guidelines	Reduced fetal reserves, ↑ maternal and fetal acidosis
Pre term labour < 37 weeks	Limited reserves
Tachysystole i.e. > 5 contractions over 10 mins without FHR abnormalities	Risk of fetal asphyxia
Uterine hypertonus i.e. contractions > 2 min or within 60 sec of each other, without FHR abnormalities	Risk of fetal asphyxia
Uterine hyperstimulation i.e. tachysystole or uterine hypertonus with FHR abnormalities	Fetal asphyxia

Considerations for intrapartum CTG

The following are conditions where intrapartum cardiotocography is not indicated where the condition occurs in isolation, but should be considered if multiple conditions are present, due to the synergistic effect of the conditions.

Antenatal risk factor	Intrapartum risk factors
Pregnancy gestation 41.0 - 41.6 weeks gestation	Maternal pyrexia ≥ 37.8 and < 38C
Gestational hypertension	
Gestational diabetes mellitus without complicating features	
Maternal obesity (BMI 30 - 40)	
Maternal age ≥ 40 and < 42 years	

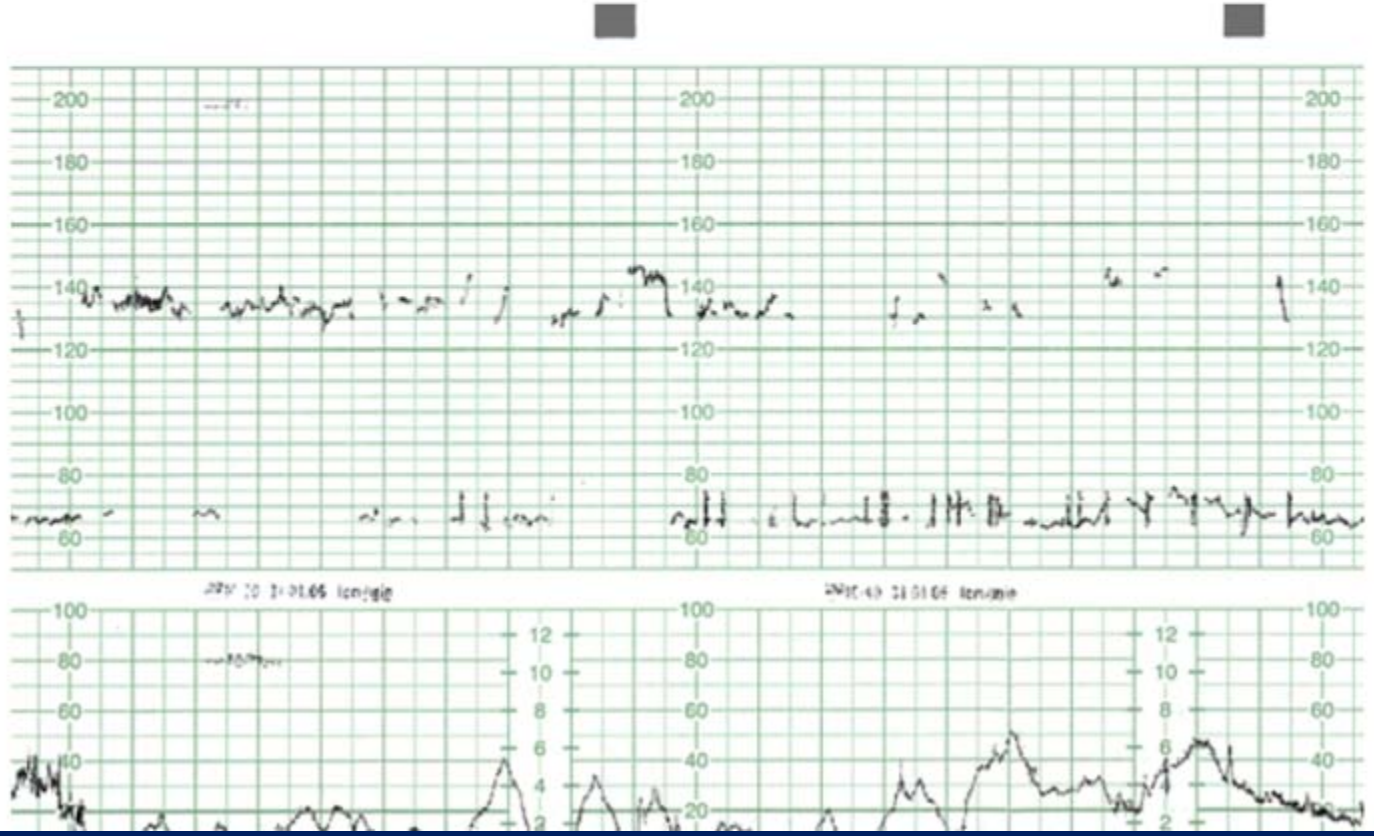
TRACING ACQUISITION

- Maternal position for CTG acquisition :
  - The lateral recumbent, half-sitting, and upright positions
- Paper scales for CTG registration and viewing :
  - "paper speed" are usually 1, 2 or 3 cm/min-in North America and Japan it is almost exclusively 3 cm/min
- External versus internal FHR monitoring
- External versus internal monitoring of uterine contractions
- Simultaneous monitoring of the maternal heart rate
- Storage of tracings

- External Monitoring is more prone :
  - to signal loss,
  - to inadvertent monitoring of the maternal heart rate
  - to signal artifacts such as double-counting and half-counting , particularly during the second stage of labor
  - It may also not record fetal cardiac arrhythmias accurately
  - needs repositioning of the probe should be carried out during the second stage of labor

This second trace was recorded antenatally and again reflects a fetus with an irregular heart beat, but this time is recorded by the ultrasound transducer. It should be noted that the lower recording is exactly half that of the upper heart rate recording and was noted not to be maternal. The CTG machine struggles to document a fetal heart rate pattern which is irregularly irregular; because it works by averaging the fetal heart rate before printing it. Again, this irregular heart rate would be audible and fetal well-being can be assessed by fetal movement. Antenatally a cardiac ultrasound scan should be performed to ensure there are no structural abnormalities of the fetal heart.

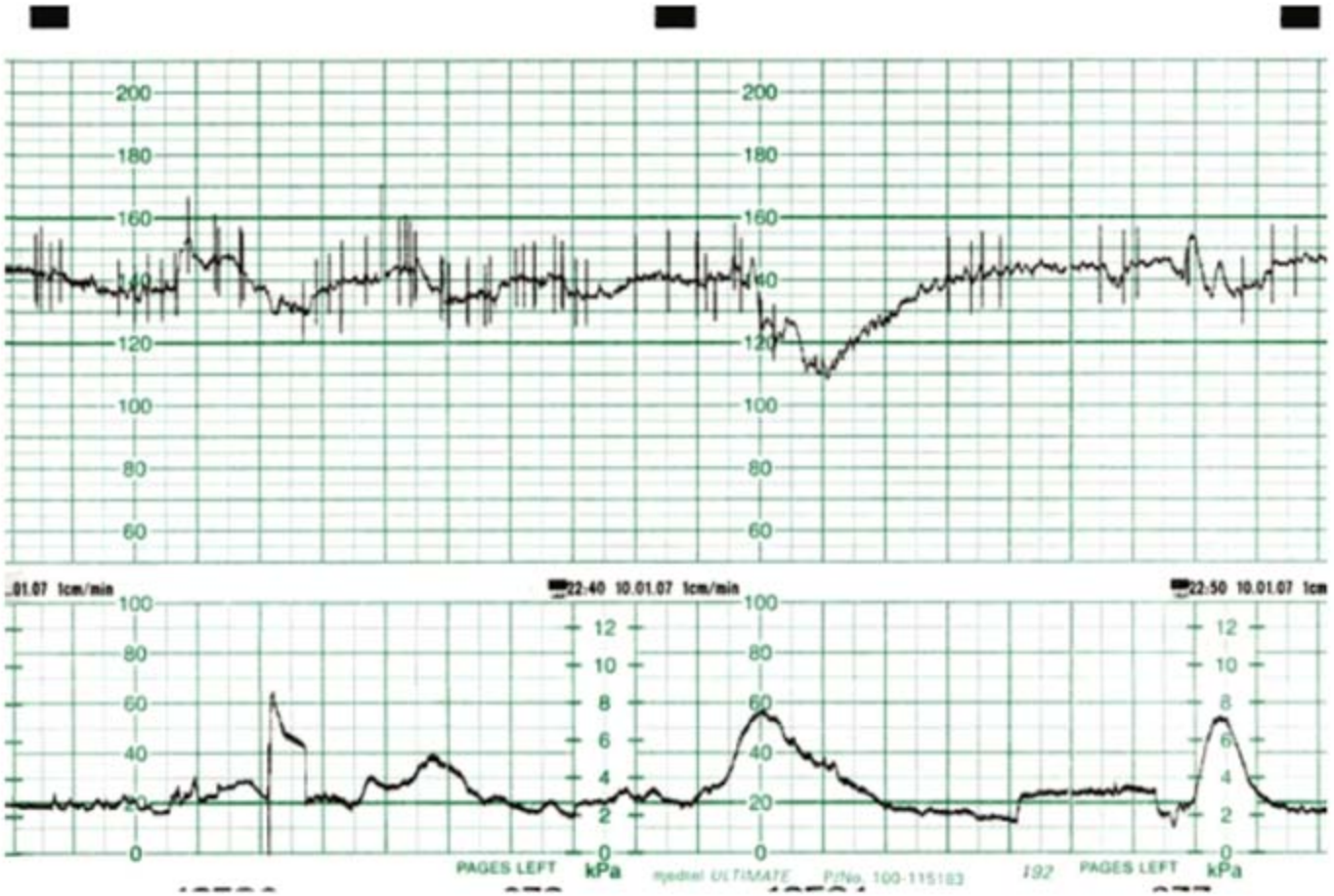
DR C Br V A D O



Fetal arrhythmias are uncommon and their aetiology typically complex.

The following CTG was from a fetus with an irregular heart beat i.e. atrial or ventricular ectopics and was recorded with a fetal scalp electrode. The irregular beat would typically be audible and will likely disappear upon delivery. The spikes on the recording reflects the rapid brief heart rate changes with the ectopic beats.

DR
C
Br
V
A
D
O



- In term fetuses, ‘cycling’ on a CTG is often seen as a hallmark of a fetal wellbeing and refers to alternating episodes of fetal activity (increased variability with or without accelerations) and quiescence

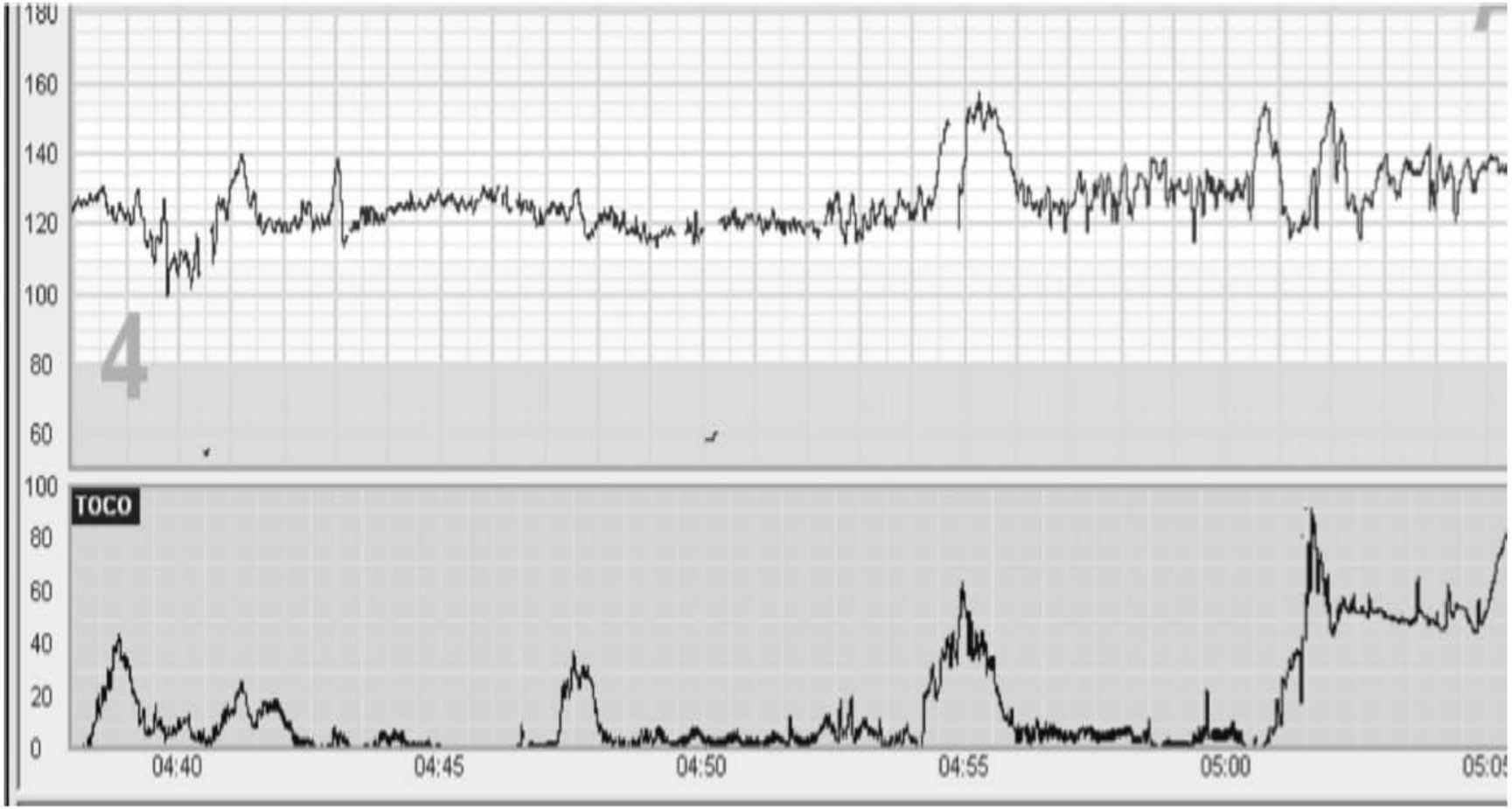
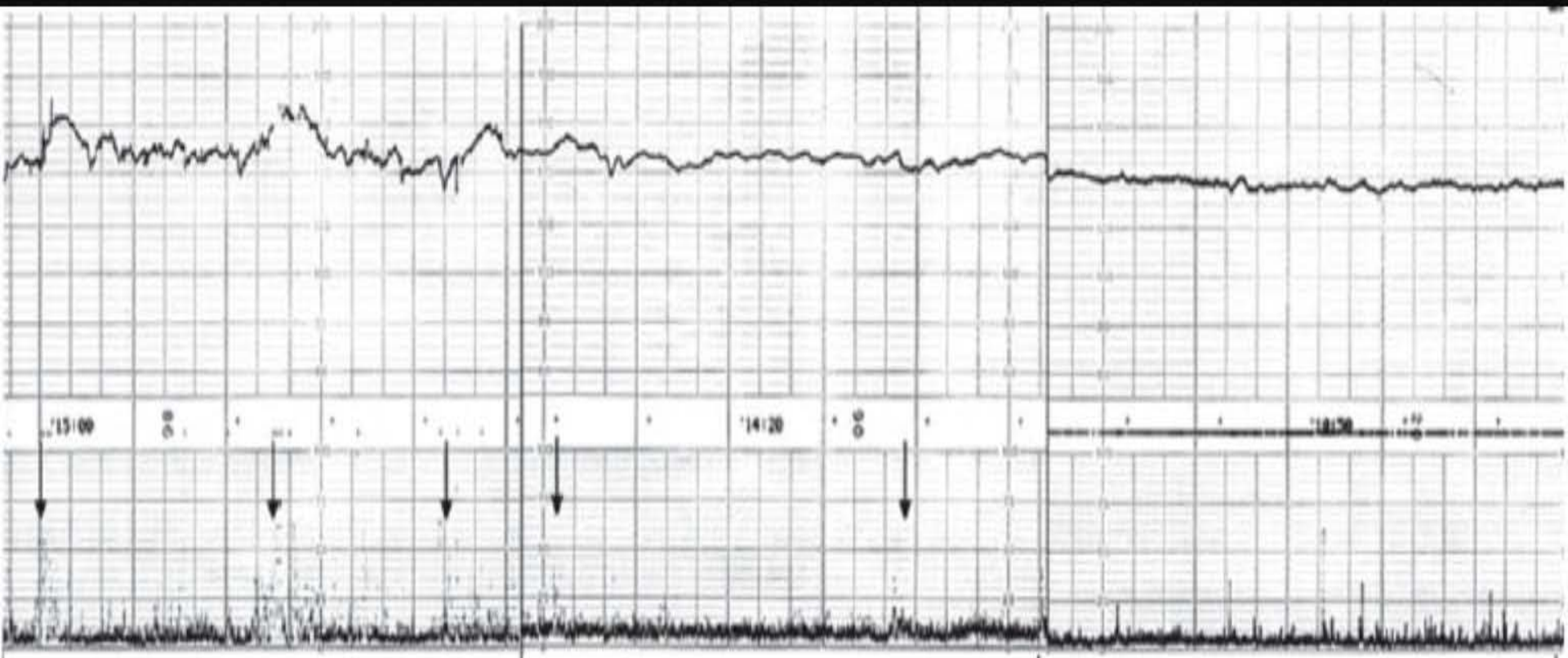


Fig. 1. CTG showing 'cycling': alternating periods of quiescence (reduced variability) and periods of activity (accelerations, variability of 5–25 bpm).



Frequent accelerations, high baseline, rich variability and frequent bursts.	Small and rare acceleration, high baseline, moderate variability, small and sporadic bursts	No acceleration, low baseline, minimal variability, no movement burst

The literature is consistent in :

- That for any individual fetus, baseline variability and accelerations will reliably be "depressed " before the pH has reached a level of acidemia associated with neurologic injury for that fetus, regardless of its quantitative value
- That no single quantitative value of fetal arterial pH serves to define a point of hypoxia-induced damage applicable to all fetuses

- There are data to suggest that in spontaneous labor it takes up to 90 seconds after a contraction for fetal oxygenation to be restored
- In oxytocin-augmented labors this recovery period averages 138 seconds
- Excessive uterine activity is often responsible for decreased fetal oxygenation, and where possible, should be avoided irrespective of fetal heart rate changes
- Spontaneous or iatrogenic in nature, excessive uterine activity can usually be reversed by reducing or stopping oxytocin infusion and/or starting acute tocolysis with beta-adrenergic agonists (salbutamol, terbutaline, ritodrine) - atosiban or nitroglycerine

The fetal heart rate, is controlled by both the autonomic and somatic nervous sytems

- ***Baroreceptors and the Parasympathetic Nervous System :*** Both fetal head and cord compression cause activation of the parasympathetic nervous system and this can often be seen as an early or variable deceleration, respectively, on the CTG trace
- ***Chemoreceptors and the Parasympathetic Nervous System :*** 'chemoreceptor – mediated' decelerations take longer to recover back to the original baseline and therefore, they may reflect an ongoing metabolic acidosis
- ***The Fetal Adrenal Glands :*** Fetal adrenal glands produce catecholamines (adrenaline and noradrenaline) in response to hypoxia over a period of time (usually over a number of hours)
- ***The Somatic Nervous System*** : A hypoxic fetus conserves energy by reducing its movements and hence accelerations would not be identified
- ***Other factors***

Acute Hypoxia

- Prolonged decelerations of more than three minutes require immediate assessment to exclude:
- **The three major 'accidents' during labour – placental abruption, cord prolapse or caesarean scar rupture**
- Iatrogenic causes include hypotension and uterine hyperstimulation
- The metabolic acidosis that ensues in such circumstances develops rapidly, with the fetal pH dropping by 0.01/minute
- In the case of hyperstimulation, the infusion of oxytocin must be stopped and consideration given to tocolysis, most commonly Terbutaline 250mcg subcutaneously or Nitroglycerin 50-100 mcg IV

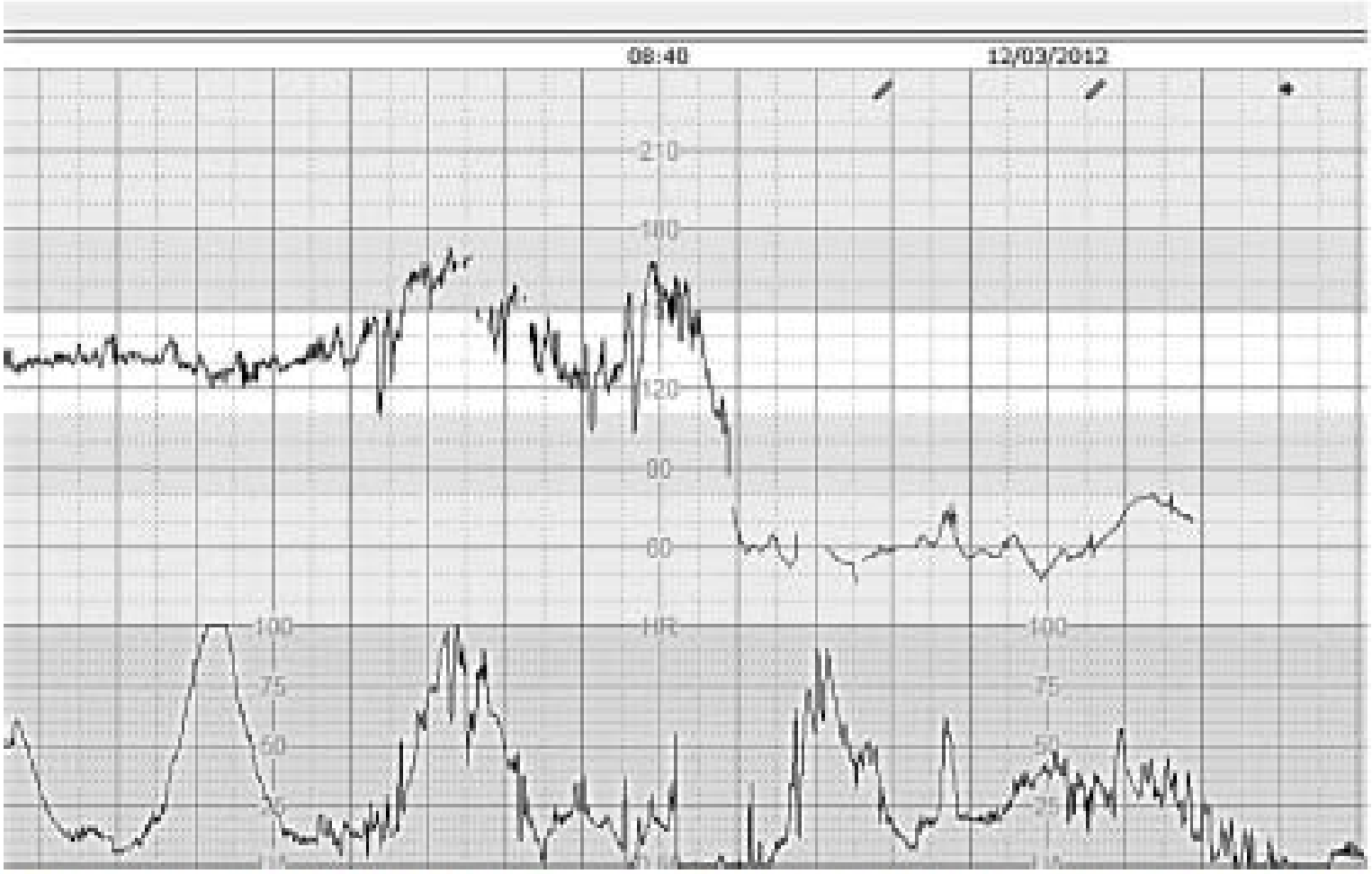


Fig. 5. Acute hypoxia due to uterine rupture. Note the sudden drop in the fetal heart rate below 80 bpm with total loss of baseline variability within 3 min of the deceleration.

- In more than 50% of prolonged decelerations no cause can be identified and these are likely to be secondary to prolonged umbilical cord compression.
- If after 6 minutes, there are no signs of recovery, preparations need to be made to be in theatre by 9 minutes with surgery starting within 12 minutes of the start of the prolonged deceleration.
- Delivery should ideally occur within 15 minutes.
- The rationale for this '3-6-9-12' minute rule is based on the observation that 90% of CTGs with a prolonged deceleration will recover within 6 minutes and 95% within 9 minutes

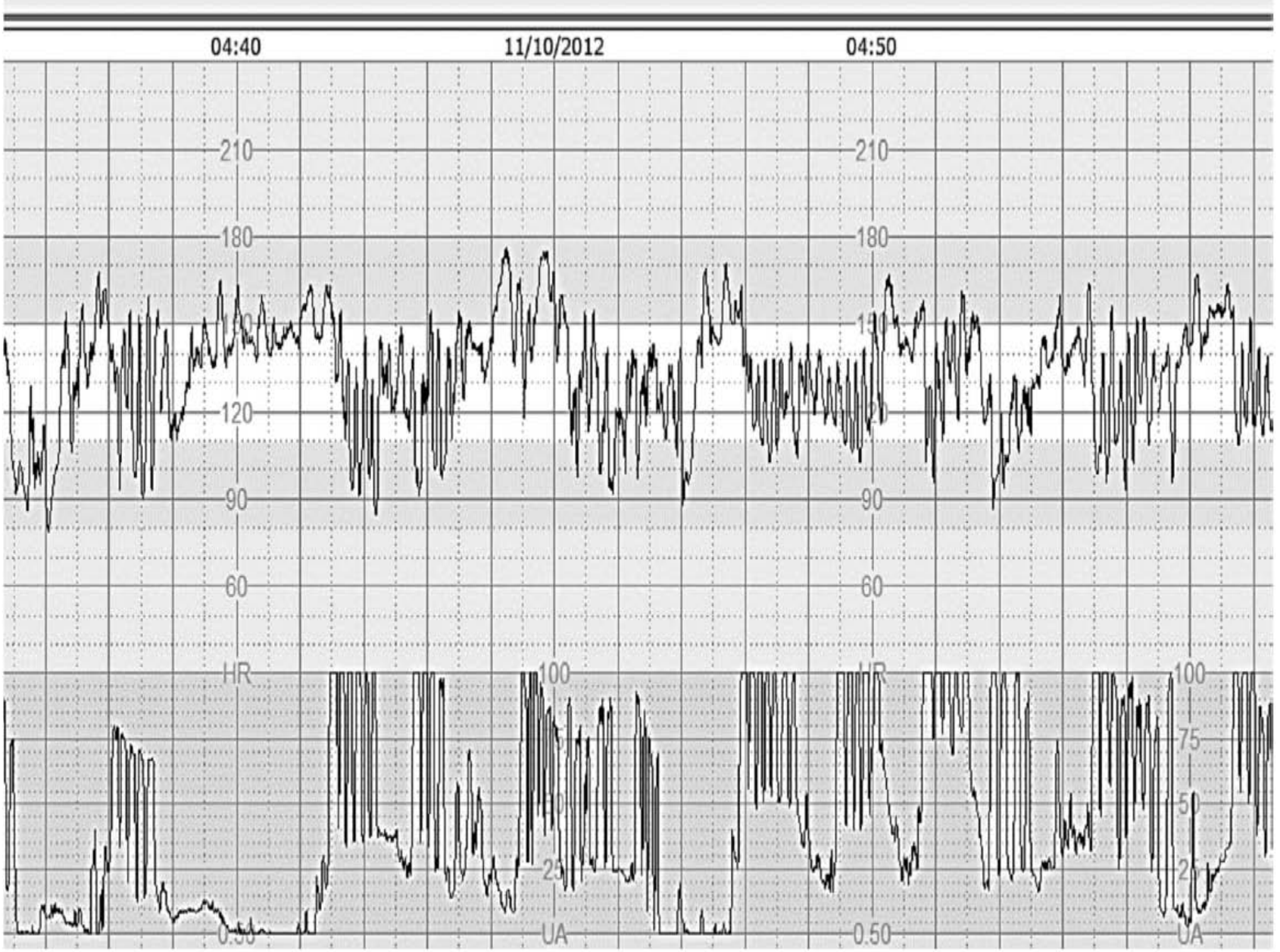


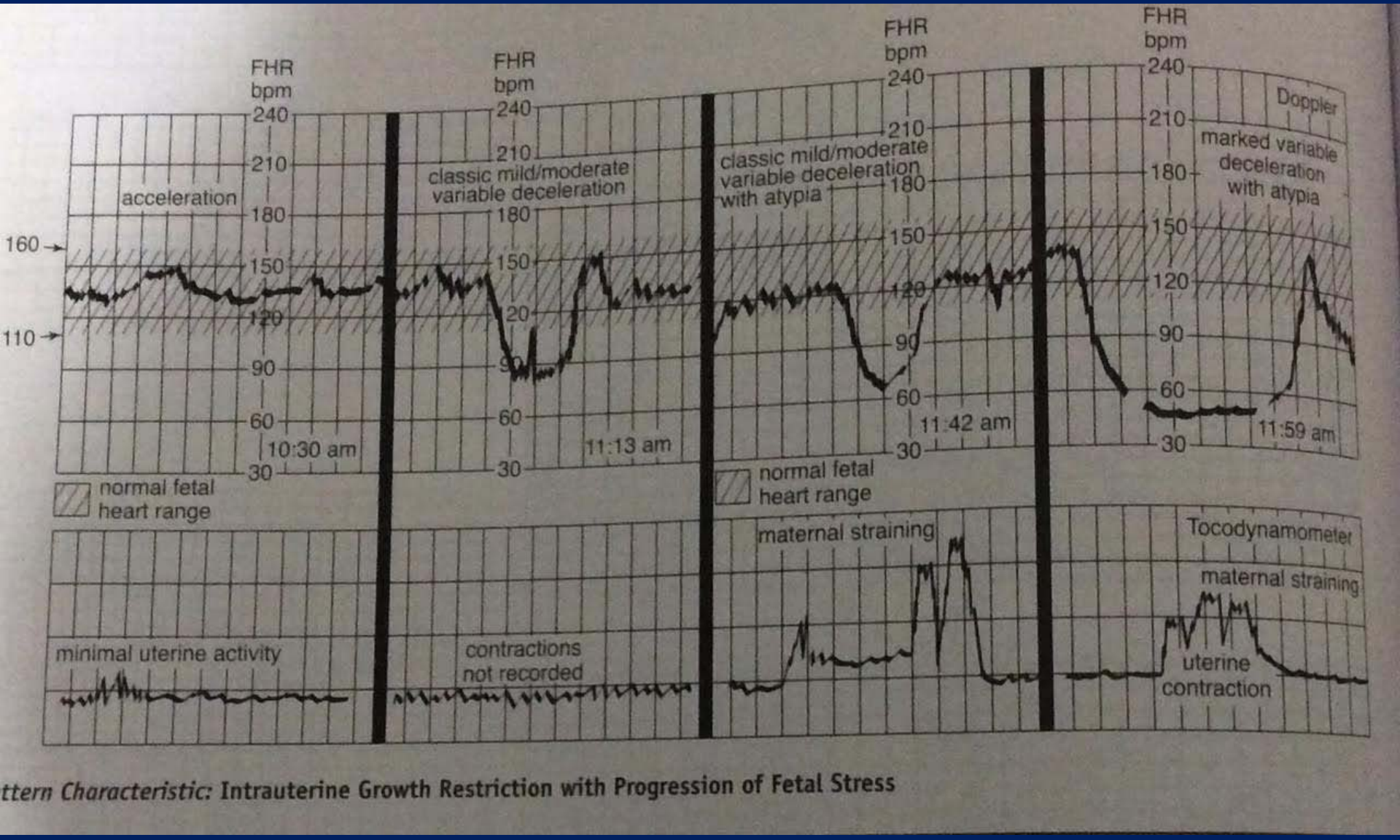
Fig. 2. Saltatory pattern suggestive of rapidly evolving hypoxia to the central nervous system, and this is usually seen with injudicious use of oxytocin or during active maternal pushing as was in this case.

Special Situations

- ***Preterm Infants***
- The CTG of a fetus at more than 32 weeks is likely to be similar to that of a term fetus and should be interpreted as such
- Fetus < 30 : Fetal tachycardia, reduced variability reduced duration and amplitude of accelerations
- Decelerations without uterine activity are not uncommon in a fetus of less than 30 weeks gestational age, with variable decelerations seen in up to 75% of intrapartum CTG tracing, compared to 30-50% in that of a term fetus

Growth Restriction :

- Restricted fetus will have less reserve than a non growth restricted fetus.
- In placental insufficiency, there is less uteroplacental circulation – more opportunity for late decelerations and less physiological reserve to combat the ensuing hypoxia
- Poor placental perfusion results in accumulation of carbon dioxide and hydrogen ions with progression to metabolic acidosis



ttern Characteristic: **Intrauterine Growth Restriction with Progression of Fetal Stress**

3cm/min

Meconium :

- The finding of meconium stained liquor is a common occurrence with between 15-20% of term pregnancies and 30-40% of post-term pregnancies affected
- In the preterm fetus, the presence of meconium in the amniotic fluid may be associated with a higher adverse perinatal outcome
- In term fetuses there is no significant correlation between meconium and adverse outcome with normal heart rate monitoring.
- However, fetal heart rate abnormalities in the presence of meconium compared to clear amniotic fluid are associated with a lower umbilical artery pH and lower apgar scores

Useful ancillary tests for intrapartum fetal evaluation

- **FHR response to stimulation:**

  This should be performed when the FHR is at its baseline rate – "Performance during a deceleration is not likely to terminate the deceleration, is not predictive of fetal acid-base status, and might exacerbate fetal compromise if parasympathetic tone increases in response to the stimulus"

- When accelerations are induced in this setting, the fetal pH is >7.20 in over 90 percent of cases, and when no accelerations occur, pH is <7.20 in approximately 50 percent of cases

Management of intrapartum fetal heart rate tracings

Fetal heart rate tracing	Possible etiologies and interpretation	Management
Category I		
Baseline 110 to 160 beats per minute with moderate variability and no late or variable decelerations. Accelerations and early decelerations may be present or absent.	This is a normal tracing.	Intermittent or continuous fetal monitoring based on clinical status and underlying risk factors. Review every 30 minutes in the first stage and every 15 minutes in the second stage of labor.
Category II		
Intermittent variable decelerations (<50 percent of contractions)	Common finding usually associated with normal outcome.	No intervention required.
Recurrent variable decelerations (>50 percent of contractions)	Umbilical cord compression. May be associated with impending acidemia, especially if progressive increase in depth, duration, and frequency. Moderate variability and/or accelerations suggest fetus is not currently acidemic.	Reposition mother to left or right lateral. Amnioinfusion is an option. Adjunctive measures to promote fetal oxygenation (oxygen supplementation, intravenous fluid bolus, reduce uterine contraction frequency) may be useful. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidotic.
		Delivery is indicated if tracing does not improve and acidemia suspected.
Recurrent late decelerations	Transient or chronic uteroplacental insufficiency, such as from hypotension, tachystole, or maternal hypoxia. Accelerations and/or moderate variability suggest fetus is not currently acidemic.	Reposition mother to left or right lateral. Adjunctive measures to promote fetal oxygenation include oxygen supplementation, intravenous fluid bolus, reduce uterine contraction frequency. Persistent late decelerations with minimal variability and absent accelerations suggest fetal acidemia; this is even more likely if variability is absent (category III). Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidotic.
		Delivery is indicated if tracing does not improve.
Fetal tachycardia (baseline heart rate greater than 160 beats per minute for at least 10 minutes)	Infection, medication, maternal medical disorders, obstetric complications, fetal tachyarrhythmia (typically rate over 200 beats per minute). Fetal acidemic more likely when associated with minimal or absent variability, absent accelerations, and/or recurrent decelerations.	Treat underlying cause, if known. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidotic.
		Delivery is indicated if tracing does not improve and acidemia suspected.
Bradycardia (baseline heart rate less than 110 beats per minute for at least 10 minutes)	Acute onset may be due to hypotension, umbilical cord occlusion, rapid fetal descent, tachysystole, abruption, uterine rupture. Fetal acidemic more likely when associated with minimal or absent variability and absent accelerations during baseline periods.	Treat underlying cause, if known. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidotic. Delivery is indicated if tracing does not improve and acidemia suspected.
Prolonged decelerations (15 beats per minute drop below baseline for more than 2 and less than 10 minutes)		
Minimal variability	Fetal sleep, medication, fetal acidemia. If due to fetal sleep, should recover in 20 to 60 minutes. If due to maternal medication, should recover as medication wears off.	If decreased fetal oxygenation suspected, reposition mother to left or right lateral. Adjunctive measures to promote fetal oxygenation include oxygen supplementation, intravenous fluid bolus, reduce uterine contraction frequency. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidotic.
		If no improvement and no accelerations, delivery is indicated if acidemia suspected or confirmed by scalp pH.

Tachysystole (more than 5 contractions in 10 minutes, averaged over 30 minutes) with fetal heart rate changes. Tachysystole that is spontaneous and associated with a normal fetal heart rate pattern does not require treatment, but the possibility of placental abruption as the underlying etiology should be considered.	Spontaneous labor: Tachysystole may be associated with fetal acidemia if accompanied by recurrent fetal heart rate decelerations.	Reposition mother to left or right lateral, oxygen supplementation, intravenous fluid bolus. If ineffective, reduce uterine contraction frequency with a tocolytic. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidotic.
	Induction or augmentation.	Decrease or stop uterotonic medications. Reposition mother to left or right lateral, oxygen supplementation, intravenous fluid bolus. If ineffective, reduce uterine contraction frequency with a tocolytic. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidotic.
Category III		
Absent baseline variability and recurrent late decelerations, recurrent variable decelerations, or bradycardia	Increased risk of fetal acidemia.	Prepare for delivery and reposition mother to left or right lateral, oxygen supplementation, intravenous fluid bolus. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidotic. If no improvement after conservative measures and scalp stimulation does not result in acceleration, delivery is advisable.
Sinusoidal	Increased risk of hypoxemia. Risk of acidemia increased if prolonged or amplitude of 15 beats per minute or more.	Prepare for delivery and reposition mother to left or right lateral, oxygen supplementation, intravenous fluid bolus. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidotic. If no improvement after conservative measures and scalp stimulation does not result in acceleration, delivery is advisable.

This chart represents a suggested approach to the interpretation and management of fetal heart rate patterns. It is not intended as a standard of care. Patient-specific factors need to be considered in the evaluation and management of individual patients.

Modified from: American College of Obstetricians and Gynecologists Practice Bulletin. Management of intrapartum fetal heart rate tracings. Obstet Gynecol 2010; 116:1232.

Dr. C. BraVADO

- **D**efine **R**isk
- **C**ontractions (in 10 mins)
- **B**aseline **Ra**te (should be 110-160)
- **V**ariability (should be greater than 5)
- **A**ccelerations
- **D**ecelerations
- **O**verall (normal or not)

Factors influencing CTG interpretation

Previous antenatal traces might provide very useful information, particularly where the trace is unusual. Some fetuses do have slightly unusual traces.

The parity might be important in terms of anticipating rates of progress in labor.

The clinical history is important, such as previous deliveries and investigations like AFI, Doppler flow studies or biometry.

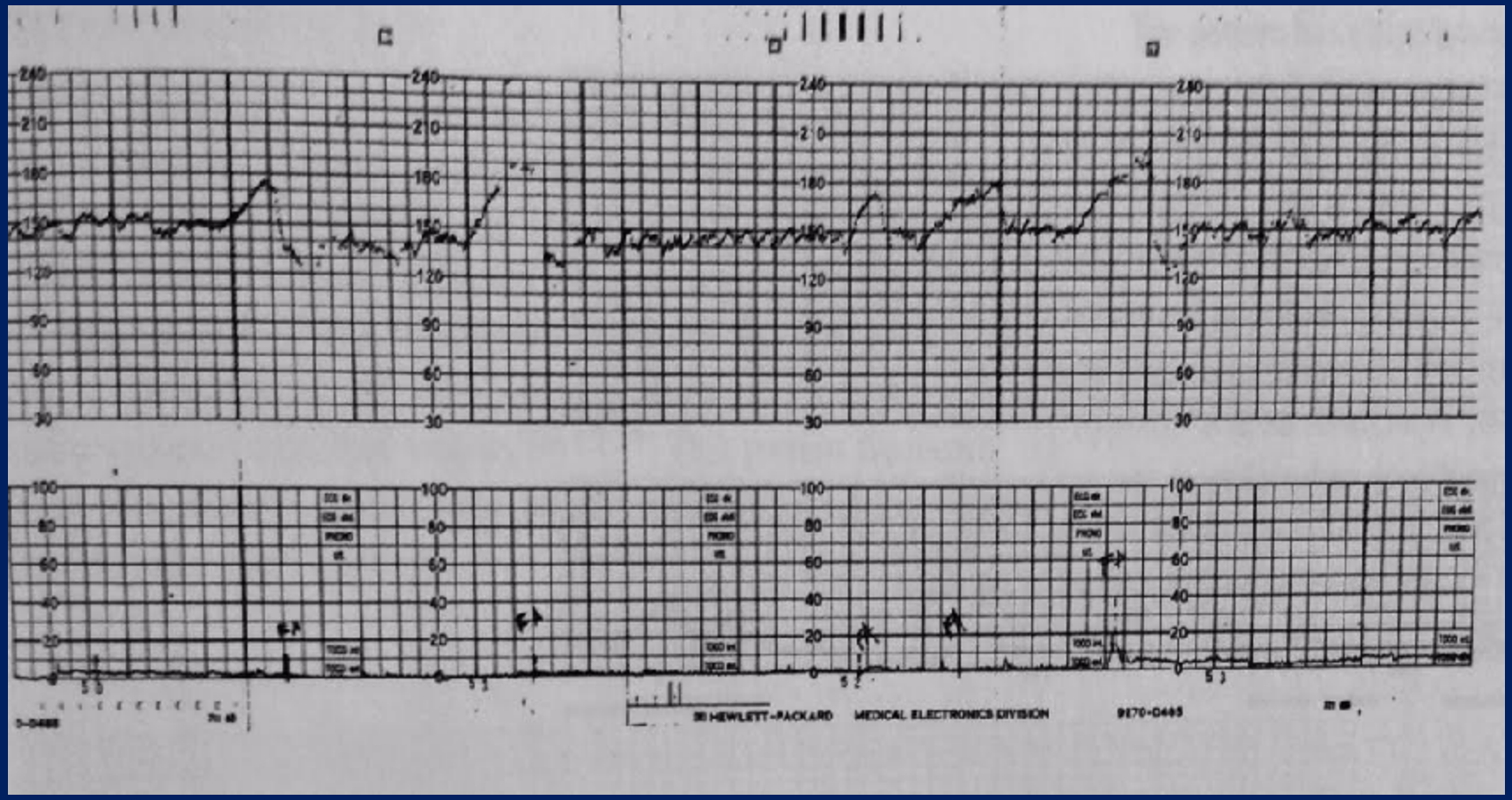
Any infusions that are running, **medications** being taken or **illicit drug use** is important. If it affects the mother it will affect the fetus and therefore potentially, the CTG.

The stage of labor is important. There are abnormal traces you might accept in second stage, accompanied by rapid progress, which you would not find acceptable early in labor.

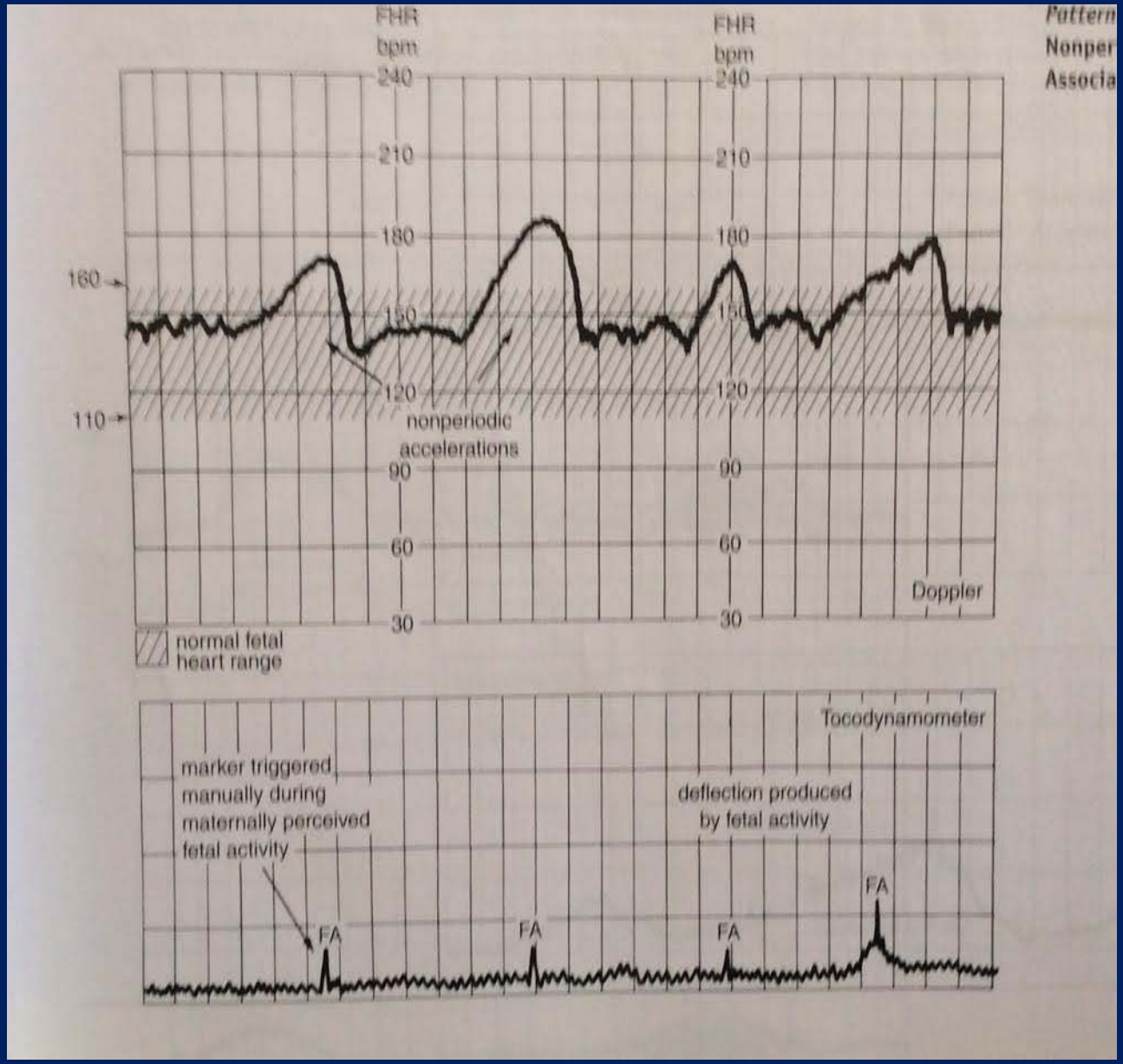
The trace prior to and following the abnormality is also important. Often our focus is on the abnormal component, when it is the reassuring information either side of the abnormality that is informative in terms of fetal wellbeing.

+ Maternal fever, hypovolenia and dehydration

DR : G1-PROM – 40 wks – Admission test
C: non
Bra : 150/min
V: mod
A: +
D: -

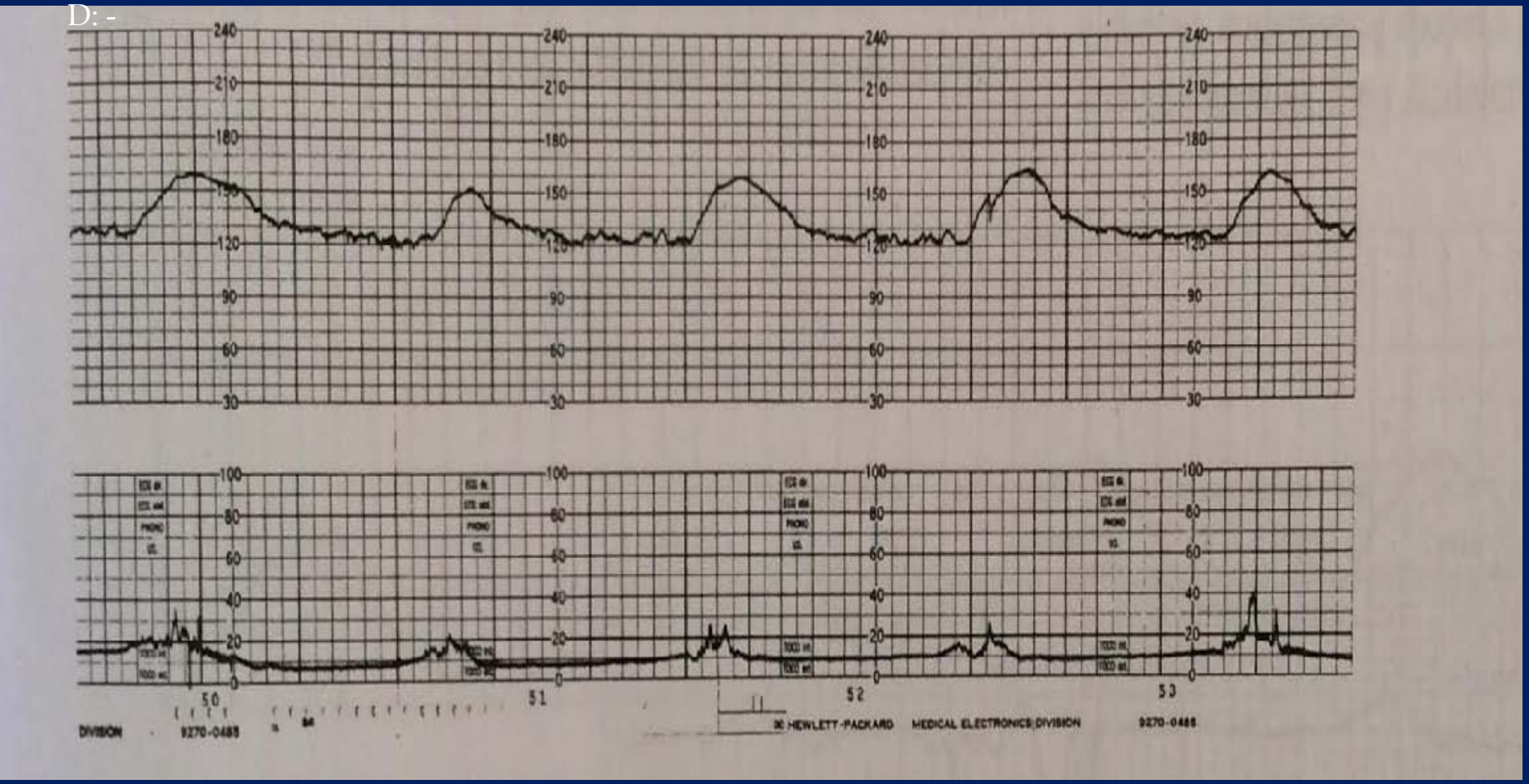


3cm/min

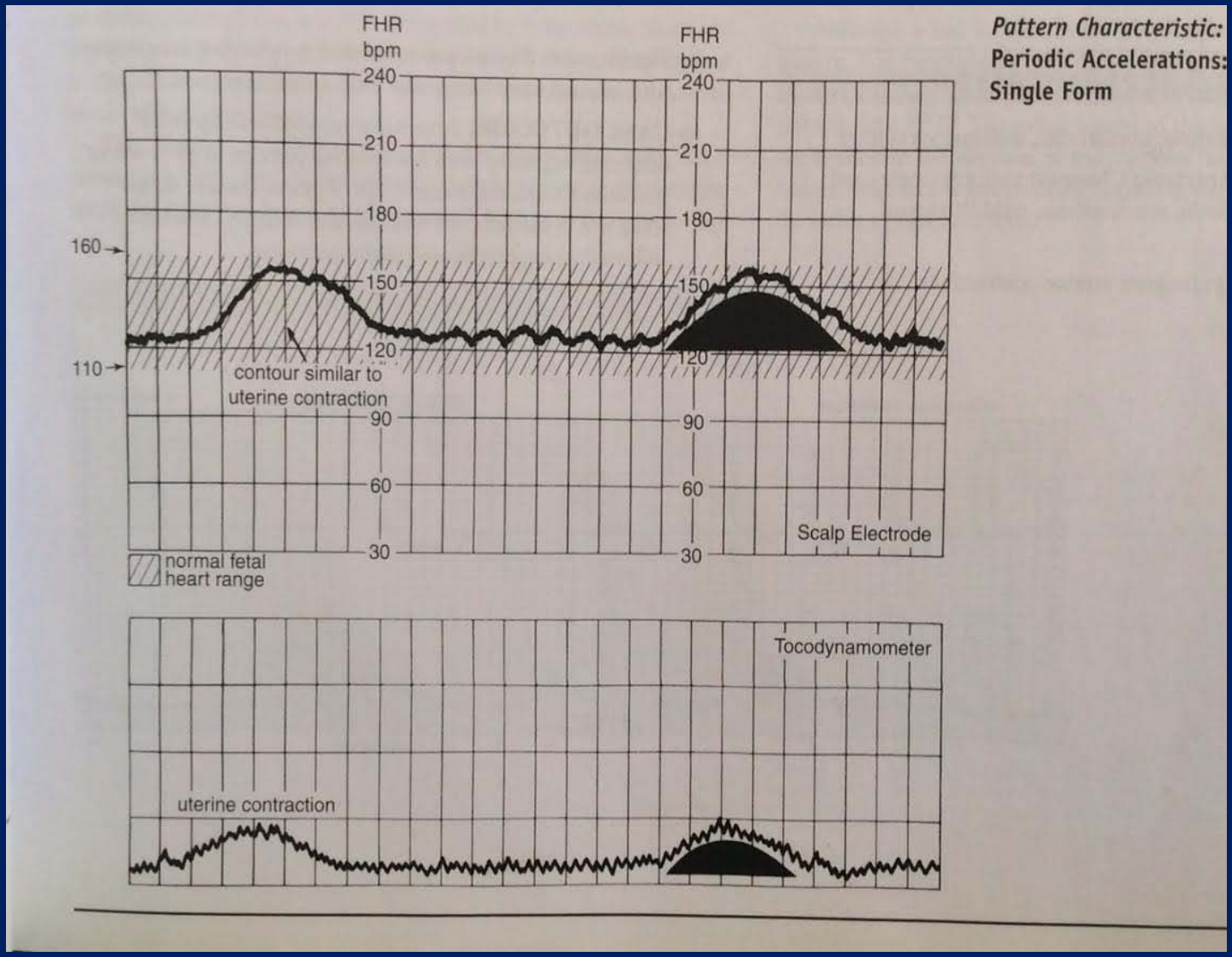

FHR
bpm
240
210
180
150
120
90
60
30
160
110
nonperiodic
accelerations
normal fetal
heart range
Doppler
Tocodynamometer
marker triggered
manually during
maternally perceived
fetal activity
deflection produced
by fetal activity
FA
FA
FA
FA

3cm/min

The same Pt an hour after commencement of induction
C: 2-3/10min
Bra: 125/min
V: minimal
A : +
D: -



3cm/min



Pattern Characteristic:
Periodic Accelerations:
Single Form

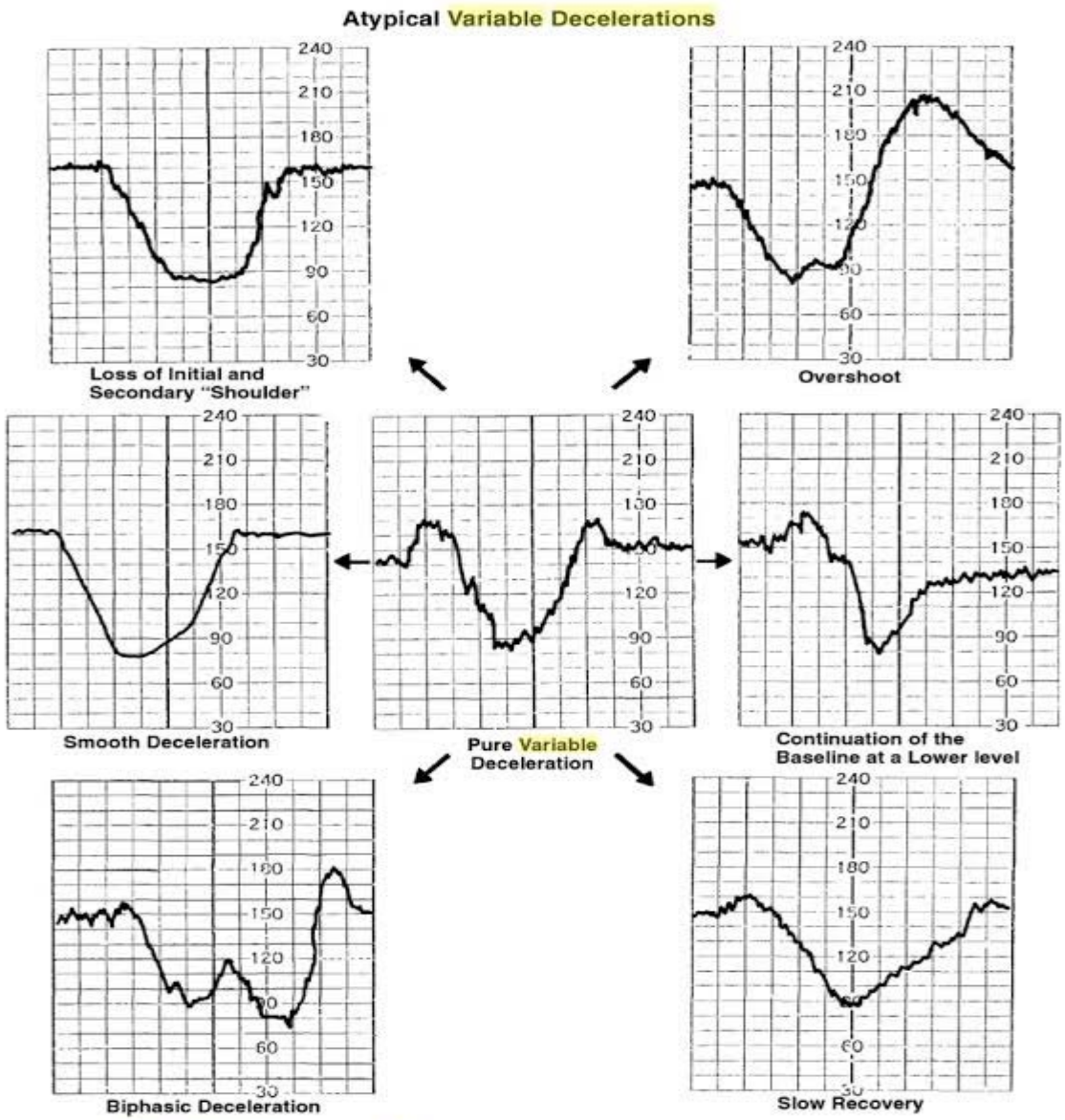


Figure 2.65: Atypical variable decelerations. (Reproduced with permission of the C. V. Mosby Company from Krebs, H. B., Petres, R. E., & Dunn, L. J. (February 1, 1983). Intrapartum fetal heart rate monitoring. VIII. Atypical variable decelerations. *American Journal of Obstetrics and Gynecology, 145*(3), 298). Labels have been modified to conform with this text.

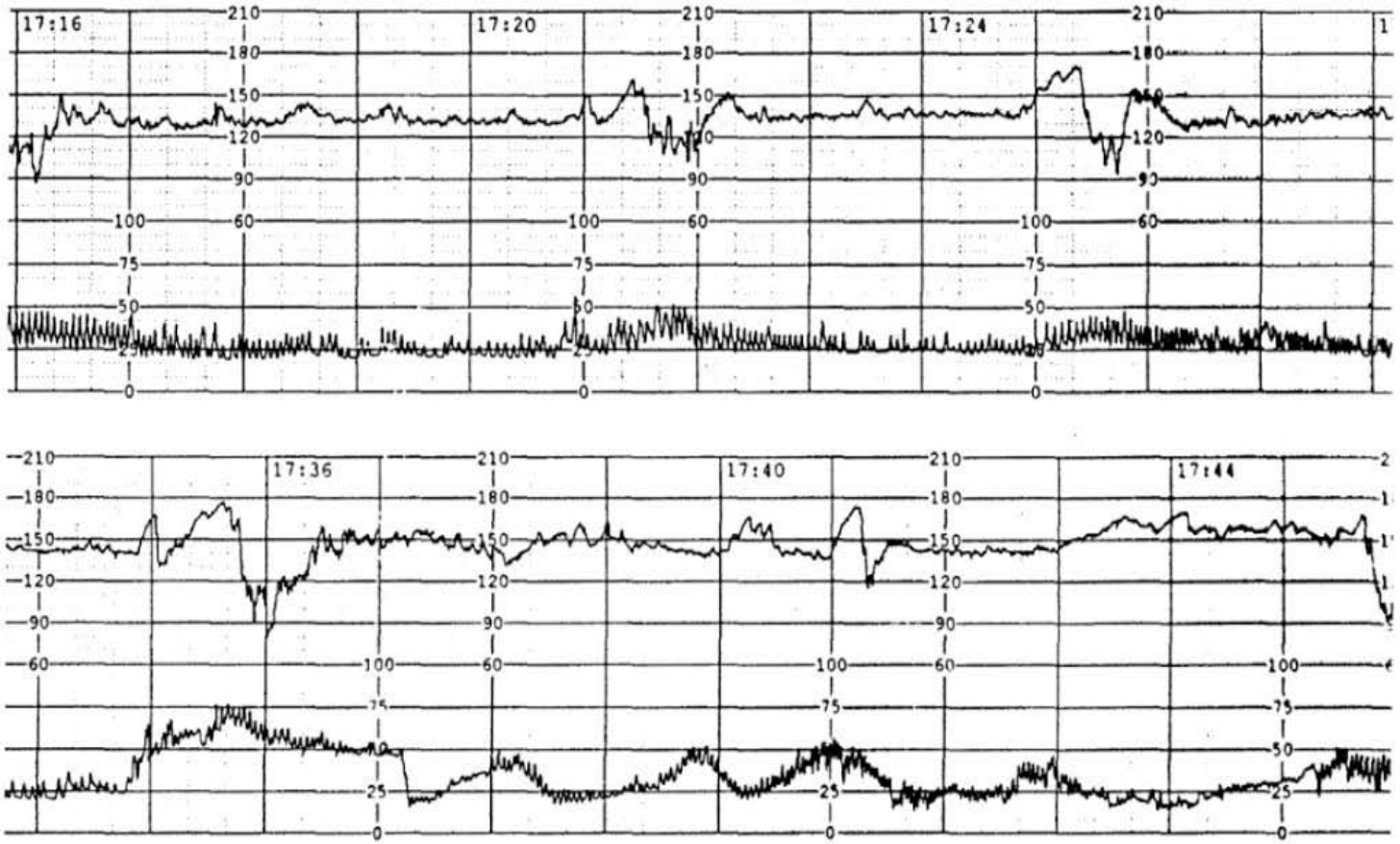


Figure 2.60: Variable decelerations with "shoulders" in a 41 week fetus. The variable decelerations in the top panel have primary and secondary acceleratory phases or shoulders. The variable decelerations in the bottom panel only have primary shoulders. There was a "very short cord." Delivery was by cesarean section. Apgar scores were 8 and 9 at 1 and 5 minutes.

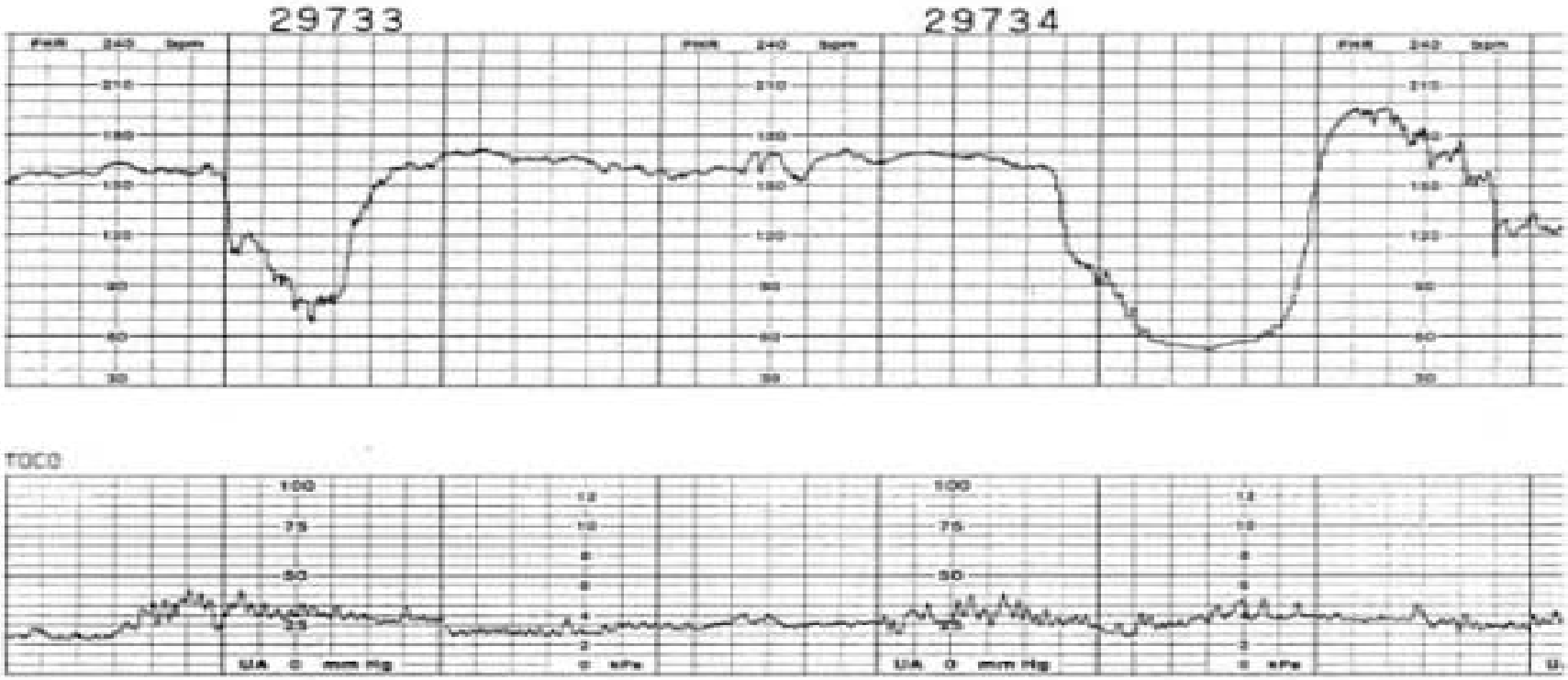


Figure 2.62: Moderate variable deceleration followed by a severe variable deceleration in the fetal heart rate of a term fetus in a G_1P_0 woman who smokes 1/2 pack per day, is 5 ft 4 inches tall, weighs 235 lbs, and has a blood pressure at the time of this pattern of 170/90. A cesarean section was performed for failure to progress. Apgar scores were 6, 9, and 9 at 1, 5, and 10 minutes. Note the overshoot after the severe variable deceleration indicating the fetus has released catecholamines as a compensatory response to hypoxia.

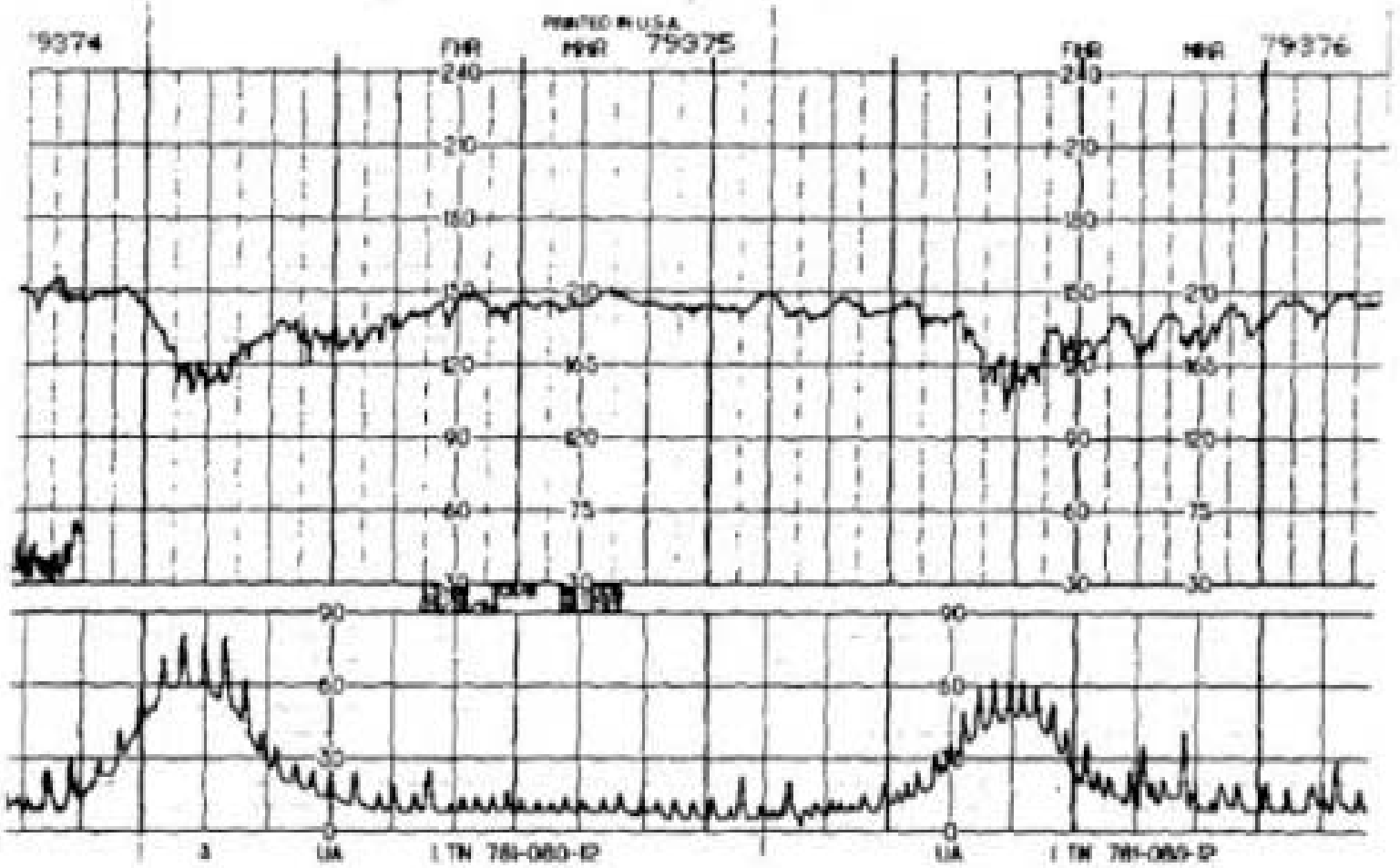


Figure 2.63: Atypical variable decelerations with a "late component." The

DR: G2 – ROM- Augmentation – AF clear- Previous tracing :reassuring
C: 4/10min
B : 120/min
V: absent
A: -
D: -
O :

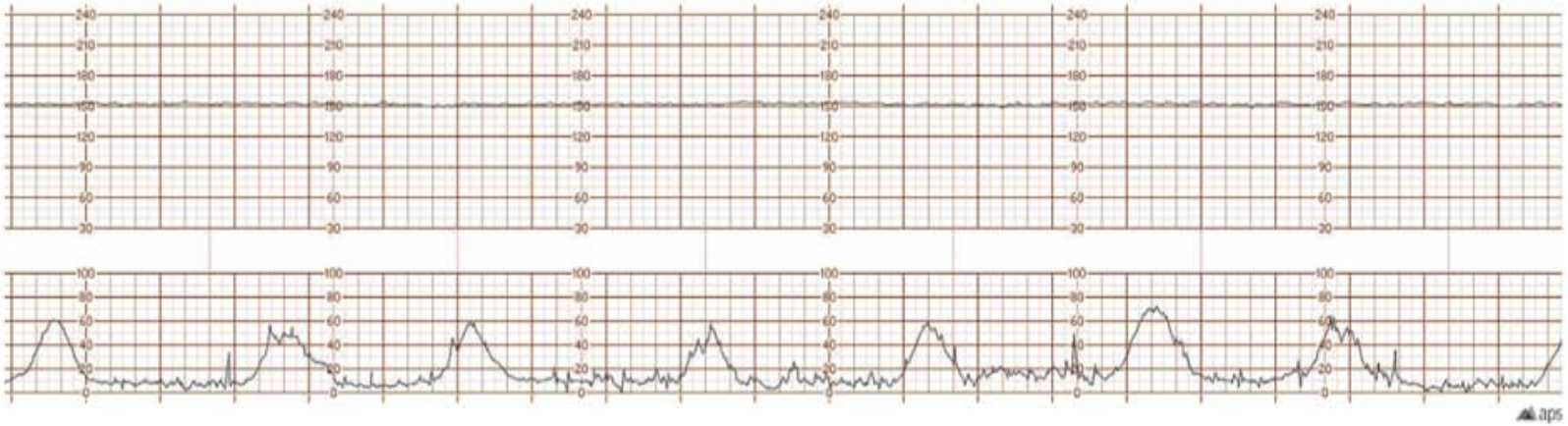


Fig. 5. The absence of decelerations excludes ongoing hypoxia in a neurologically intact fetus, but this fetus may not tolerate labor; delivery should be considered if the pattern persists for 1 hour. (*Courtesy of* Advanced Practice Strategies Inc, Boston, MA; with permission.)

3cm/min

DR:G1- Active phase – receiving epidural top -up
C: 4/10min
B: 135/min
V: mod
A: -
D: prolonged
O:

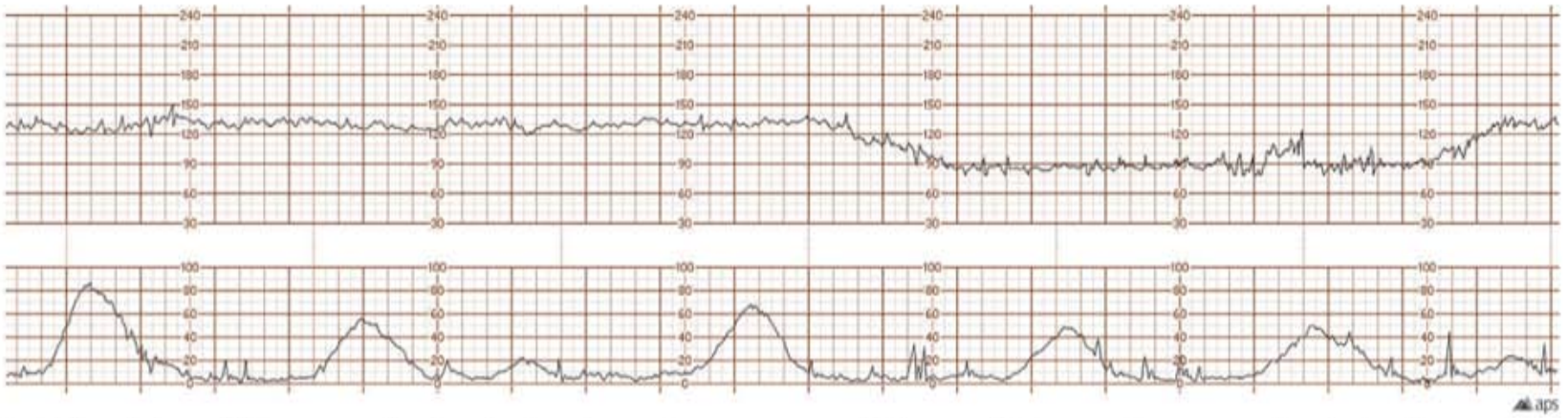


Fig. 4. The etiology of this prolonged deceleration is unknown. The management of this type of FHR pattern is not covered by the algorithm and needs to be tailored to the clinical picture. (*Courtesy of* Advanced Practice Strategies Inc, Boston, MA; with permission.)

3cm/min

Dr : G3P2L2 -low risk –second stage
C: 4/10min
B: 140/min
V: mod
A: +
D: variable
O:

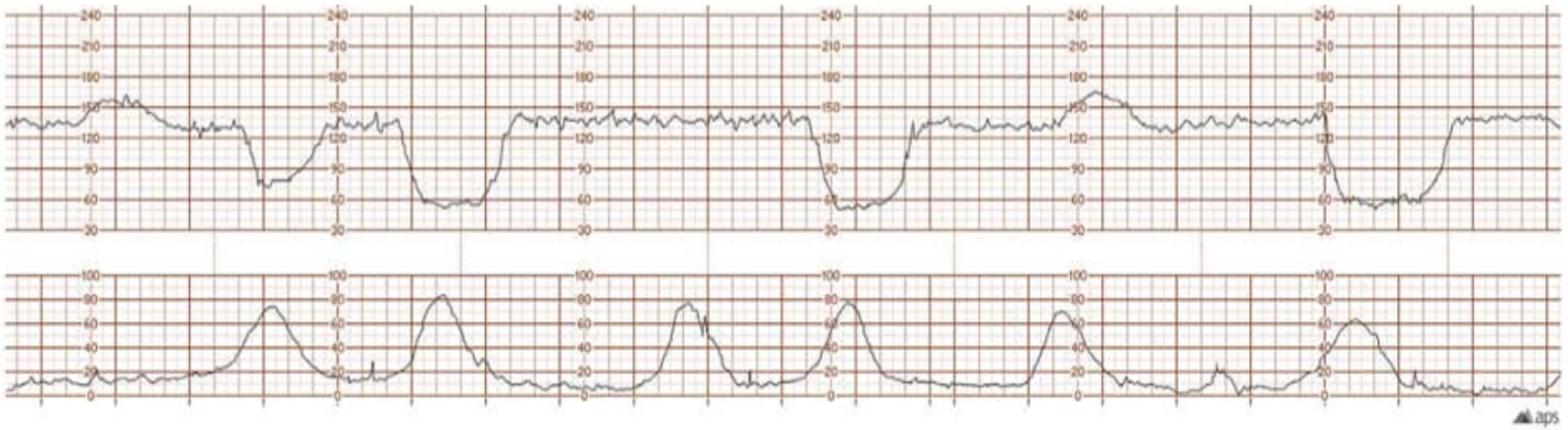


Fig. 3. When close to delivery, with normal progress in the second stage, this fetus may be watched or considered for operative vaginal delivery if the pattern persists. When remote from delivery, cesarean section should be done per the algorithm. (*Courtesy of* Advanced Practice Strategies Inc, Boston, MA; with permission.)

3cm/min

DR:G1 – severe preeclampsia – suspected IUGR – active phase (6cm)
C: 3-4/10min
B:120/min
V:
A:
D:
O:

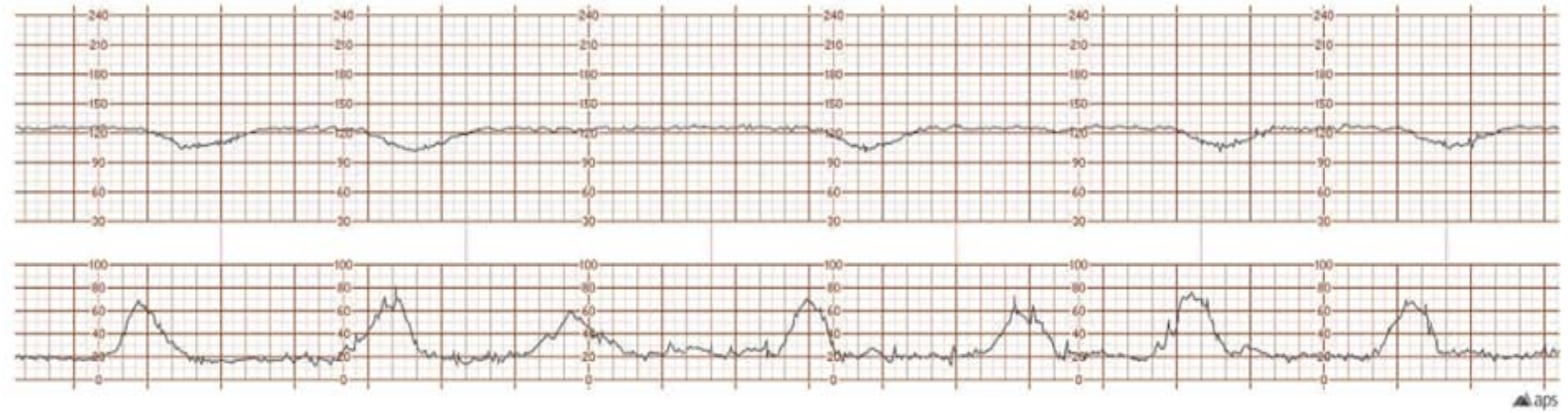


Fig. 1. FHR tracing exhibiting minimal variability with recurrent late decelerations. Expedited delivery is indicated. (*Courtesy of* Advanced Practice Strategies Inc, Boston, MA; with permission.)

3cm/min

How to Approach Intrapartum Category II Tracings

DR

G1-m-PIH Induced: MISO

C

6/min

Br

135-140/min

V

mod

A

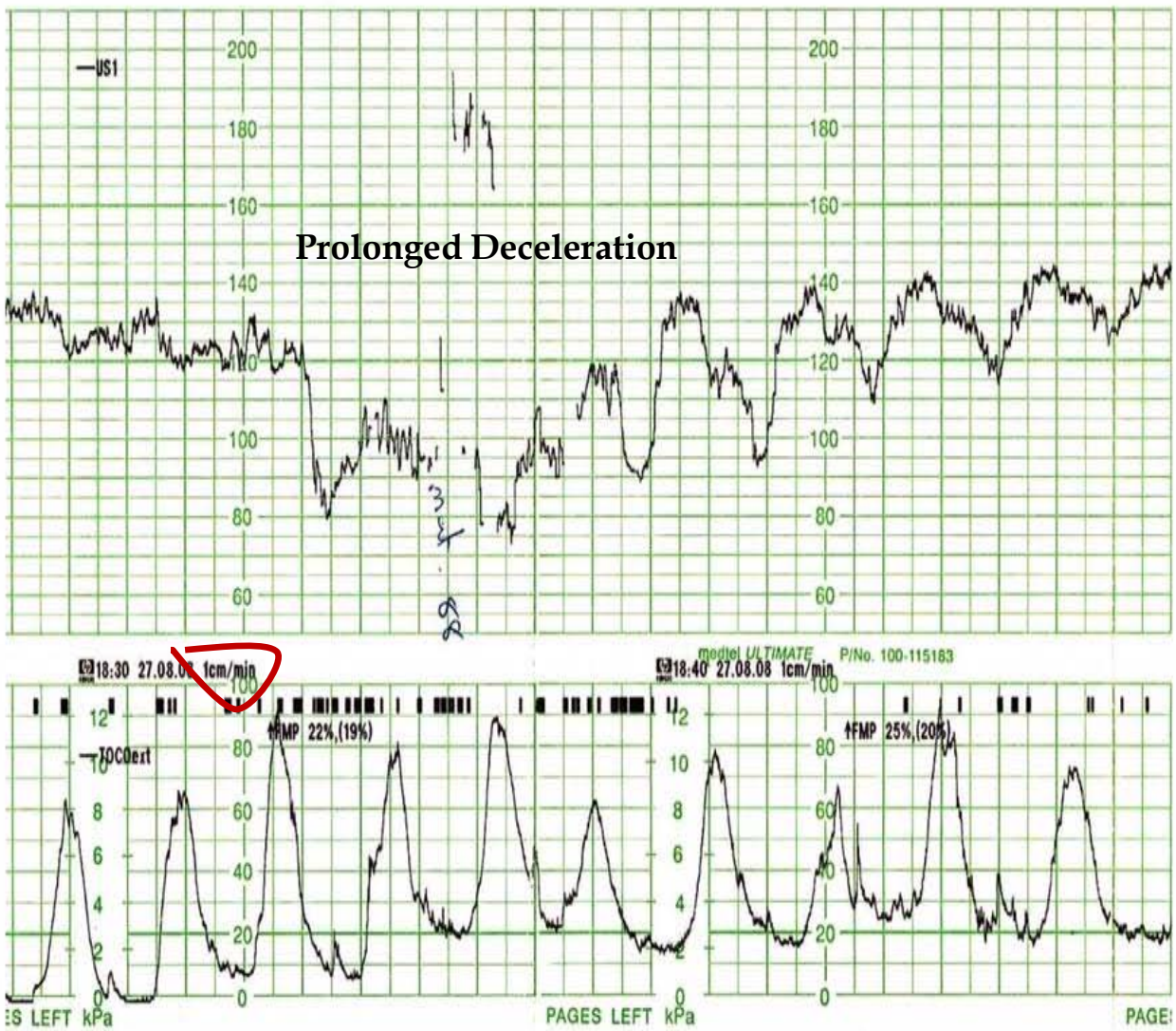
-ve

D

Prolonged late

O

The following CTG is an example of uterine hyperstimulation, where a previous uterine hypertonus, after a prostin induction at 38 weeks, was not actively managed. The bradycardia and subsequent late decelerations below was the result. An emergency LUSCS was performed with the fetus being delivered in reasonable condition. Tocolysis could also have been considered, particularly in the setting of limited theatre access.



The following CTG clearly demonstrates a compromised fetus. With a baseline rate of 165-170 bpm, absent baseline variability, no accelerations and persistent late decelerations, which are also prolonged, there is nothing physiologically reassuring about this trace. This is supported by the clinical picture of a 34 week primigravida, with a growth restricted fetus and prolonged ruptured membranes and warrants urgent delivery.

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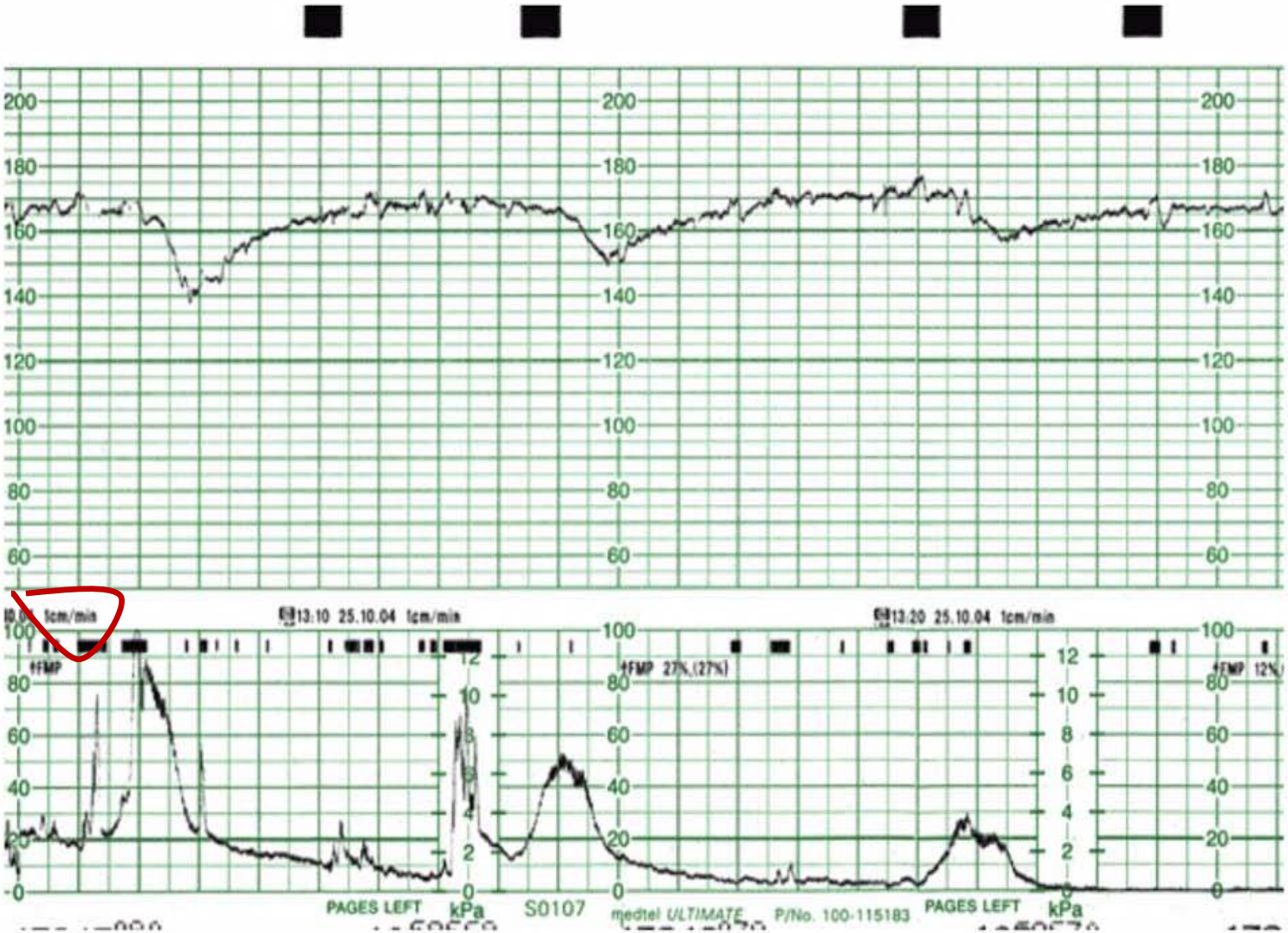

The following CTG demonstrates such an effect, with high dose beta blockers causing a baseline bradycardia in the fetus of this 36 week gestation labouring primigravida with moderate pre-eclampsia. The reduced baseline variability in this case was the result of an epidural. Despite the abnormalities in this CTG, this is clearly a well oxygenated fetus.

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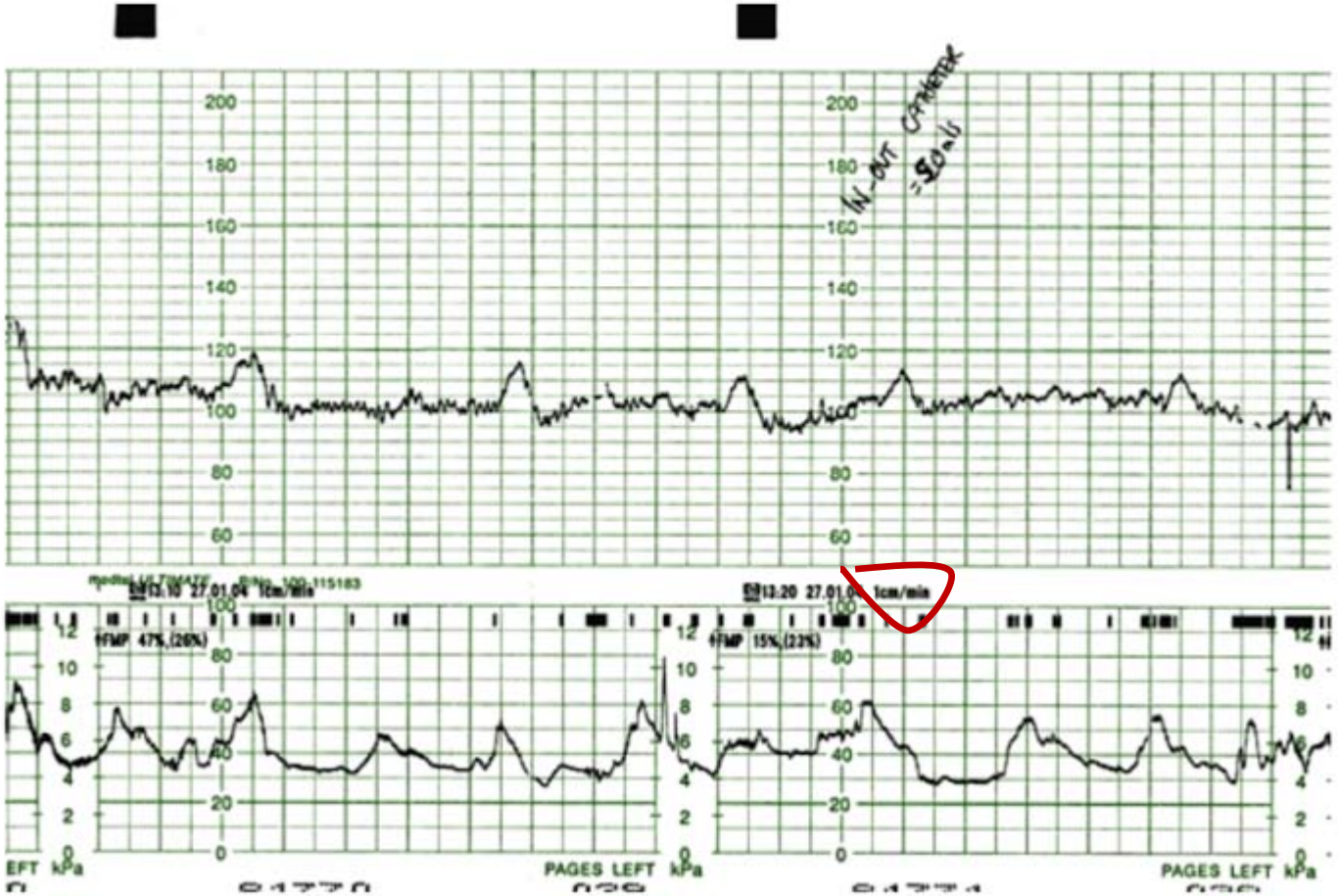
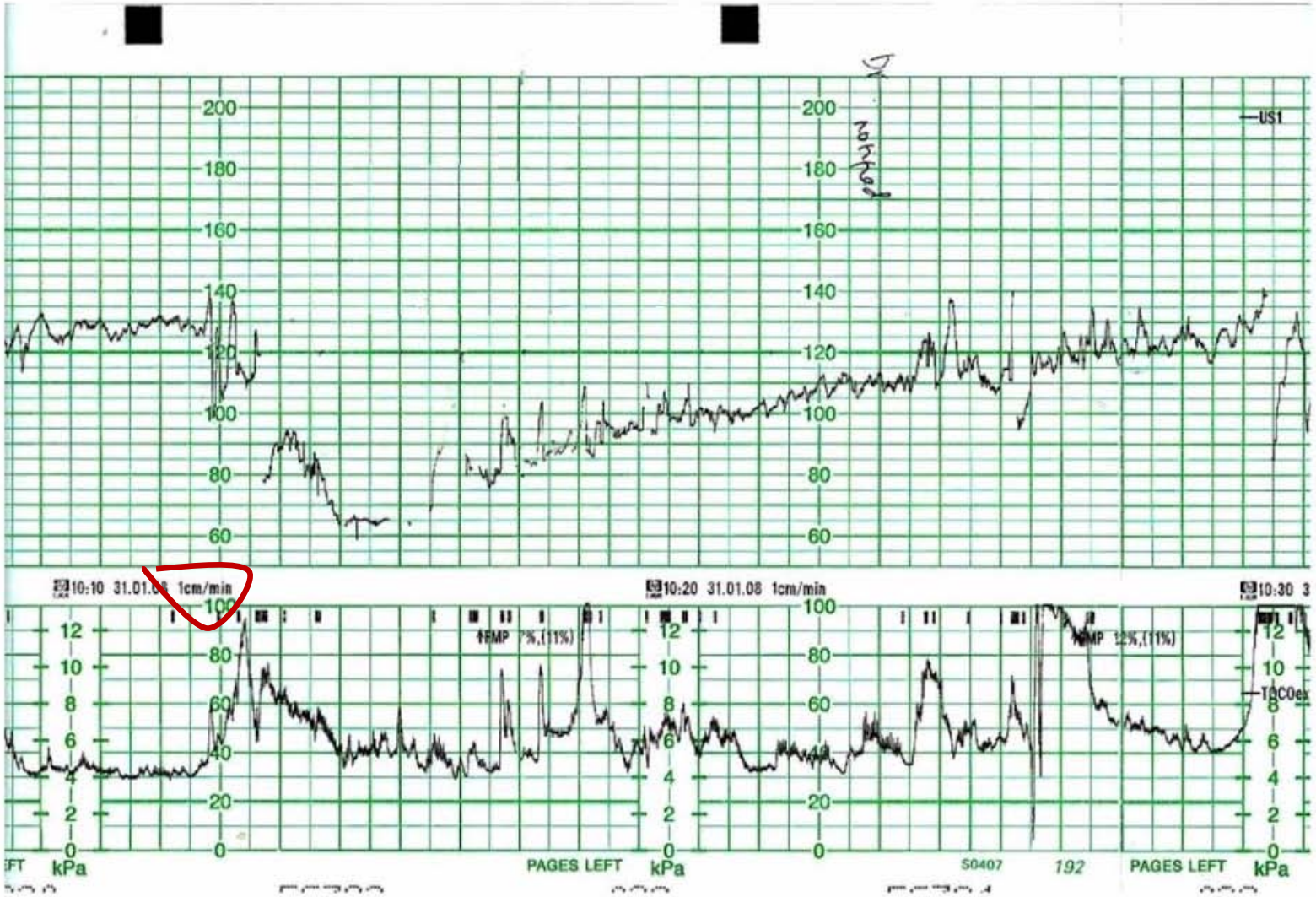


The following CTG, with a baseline rate of 125 bpm and normal baseline variability, shows the effect maternal hypotension can have on the fetal heart rate. The hypotension followed the insertion of an epidural, resulting in fetal hypoxia from reduced uterine artery blood flow. The subsequent fetal hypoxia is reflected in the bradycardia. As the maternal blood pressure is corrected, there is a slow return to the baseline.



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The following CTG is a baseline bradycardia associated with a mature parasympathetic nervous system in a fetus at 41+6 weeks gestation. With a baseline rate of 100-105 bpm, normal baseline variability and accelerations, this is clearly a well fetus, but is not a normal trace.

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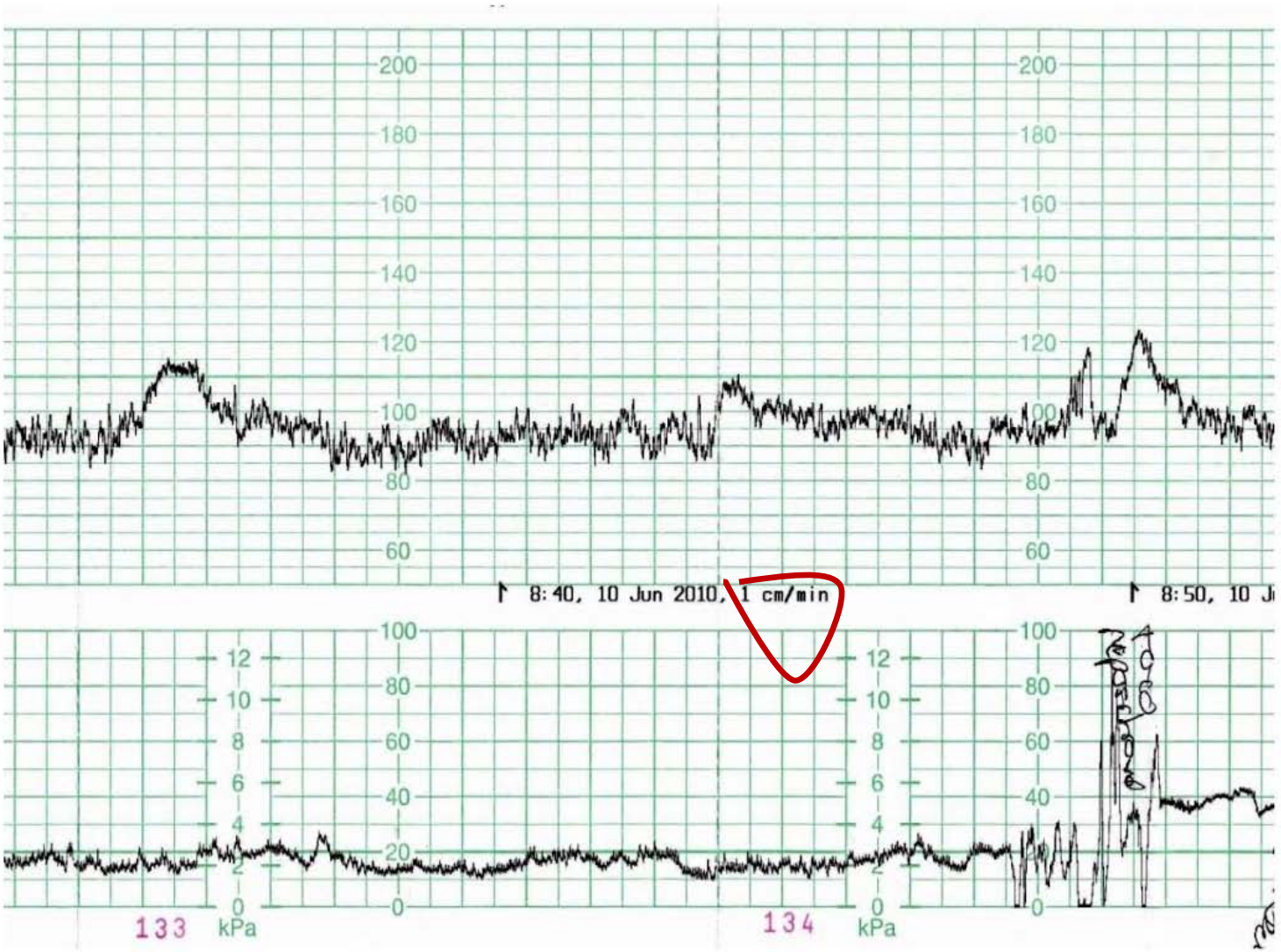

The following CTG is from a fetus whose mother is febrile with pyelonephritis (note the maternal heart rate recorded on the CTG via an O_2 saturation probe). With a baseline of 200-205 bpm and no decelerations, despite uterine activity, this fetus is unlikely to be hypoxic at this time but its reserves will be very quickly depleted. The increase in O_2 requirements resulting from the maternal temperature means a significant increase in sympathetic stimulation in order to maintain the O_2 delivery required to meet its metabolic needs. With maternal hydration, IV antibiotics and PR paracetamol, the maternal temperature was reduced and the fetal heart rate quickly improved.

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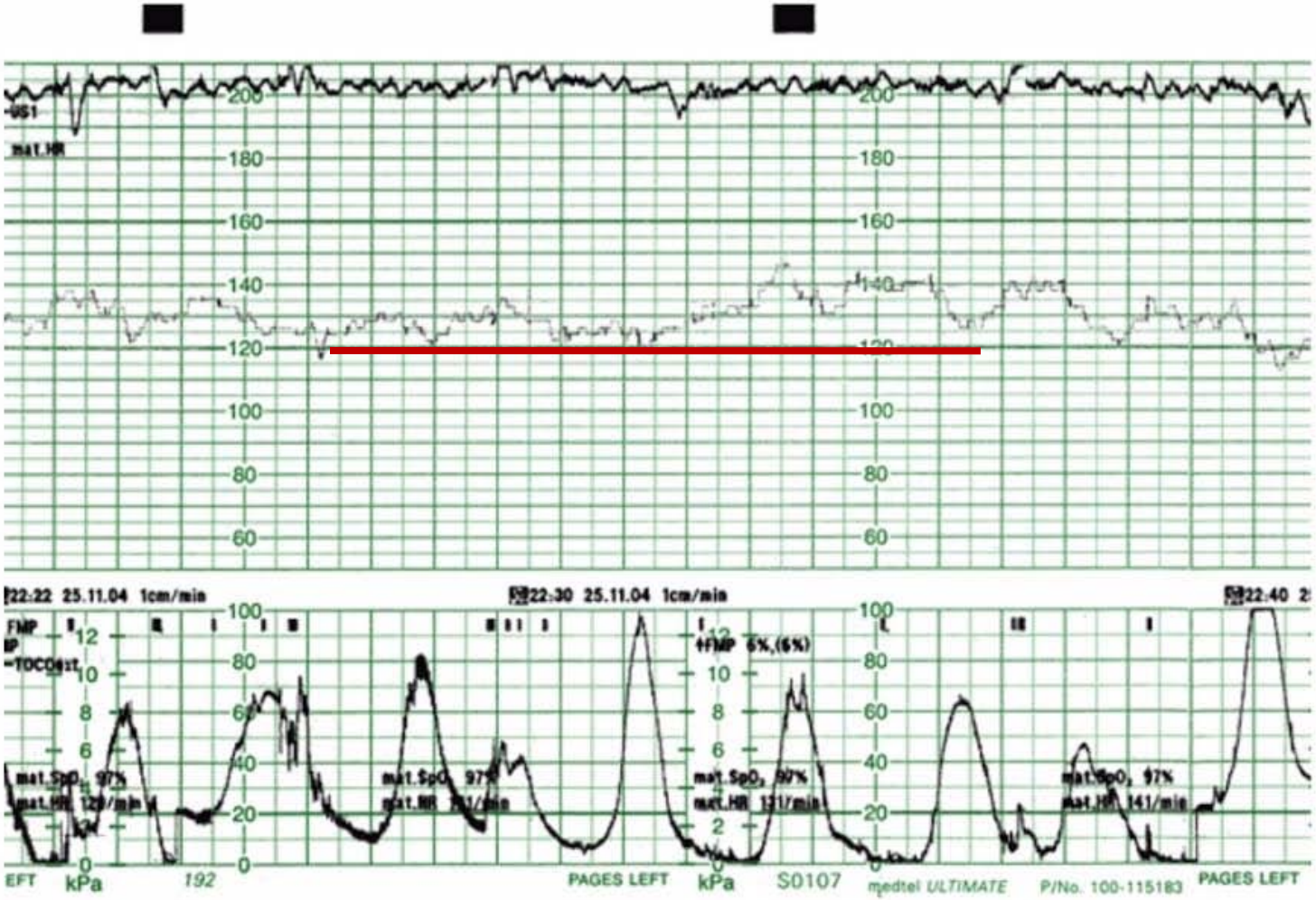

The next CTG is also a baseline tachycardia, but with a very different cause. This primigravida was being augmented with oxytocin and is in early second stage. With a baseline rate of approximately 175-180 bpm and late decelerations, which are also prolonged, this fetus is likely to be hypoxic. The tachycardia represents an increase in sympathetic stimulation in an attempt to increase its cardiac output and improve its oxygenation. Active management is required and ceasing the oxytocin infusion and notifying senior staff would be an appropriate start. Depending on the clinical progress, consideration should be given to tocolysis or delivery.

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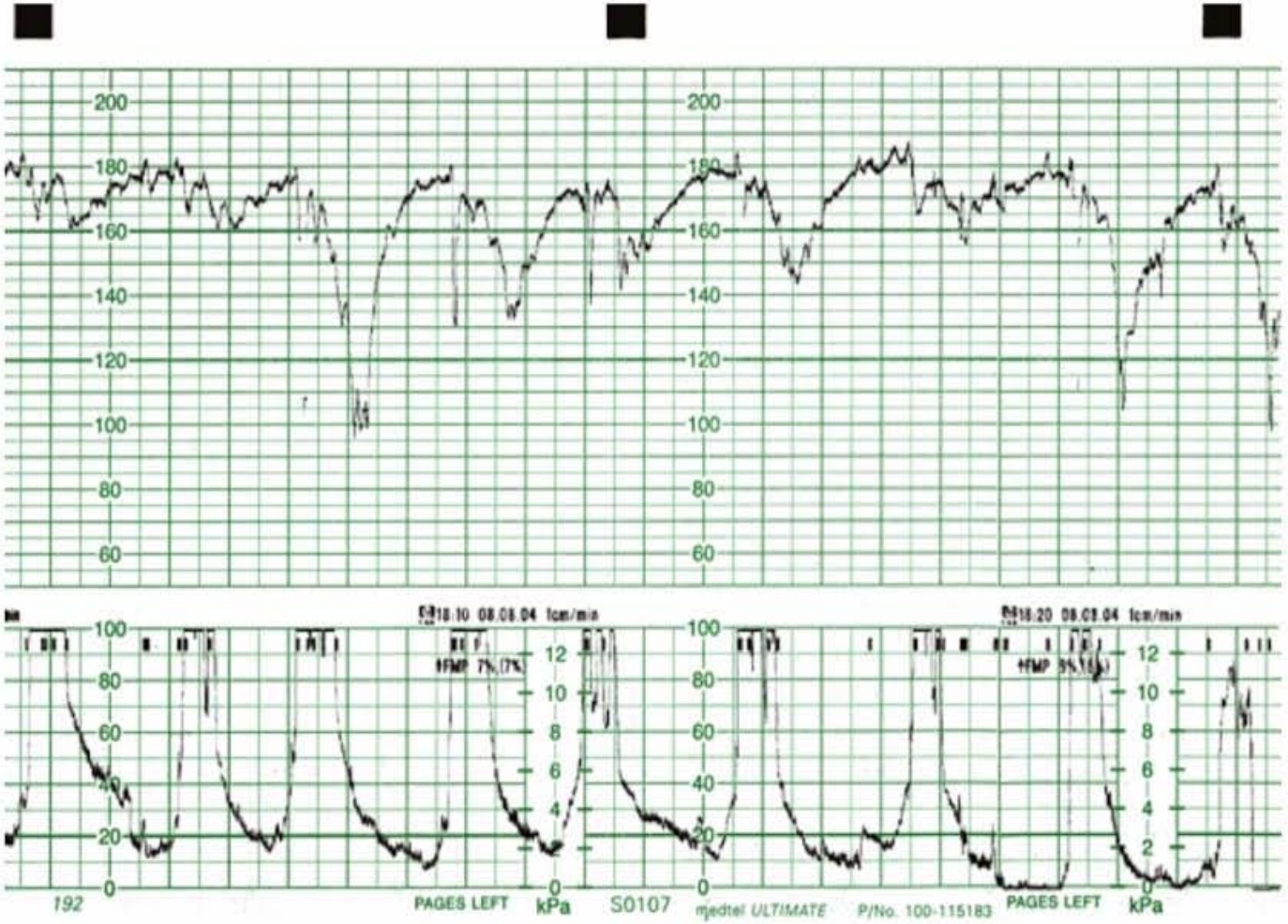

The following CTG demonstrates a baseline initially at 140bpm but rising to 150bmp over just 20 minutes. Though the baseline variability and variable decelerations reflect a well oxygenated fetus, the rising baseline indicates a fetus which is having to increase sympathetic innervation in an attempt to increase its oxygenation and is therefore at risk. In this circumstance it is likely that the lack of uterine rest between contractions (uterine hypertonus) is contributing to the fetuses' needs to increase oxygenation. Management therefore at this time would include maternal repositioning, maternal observations, reducing the oxytocin and notifying senior staff.

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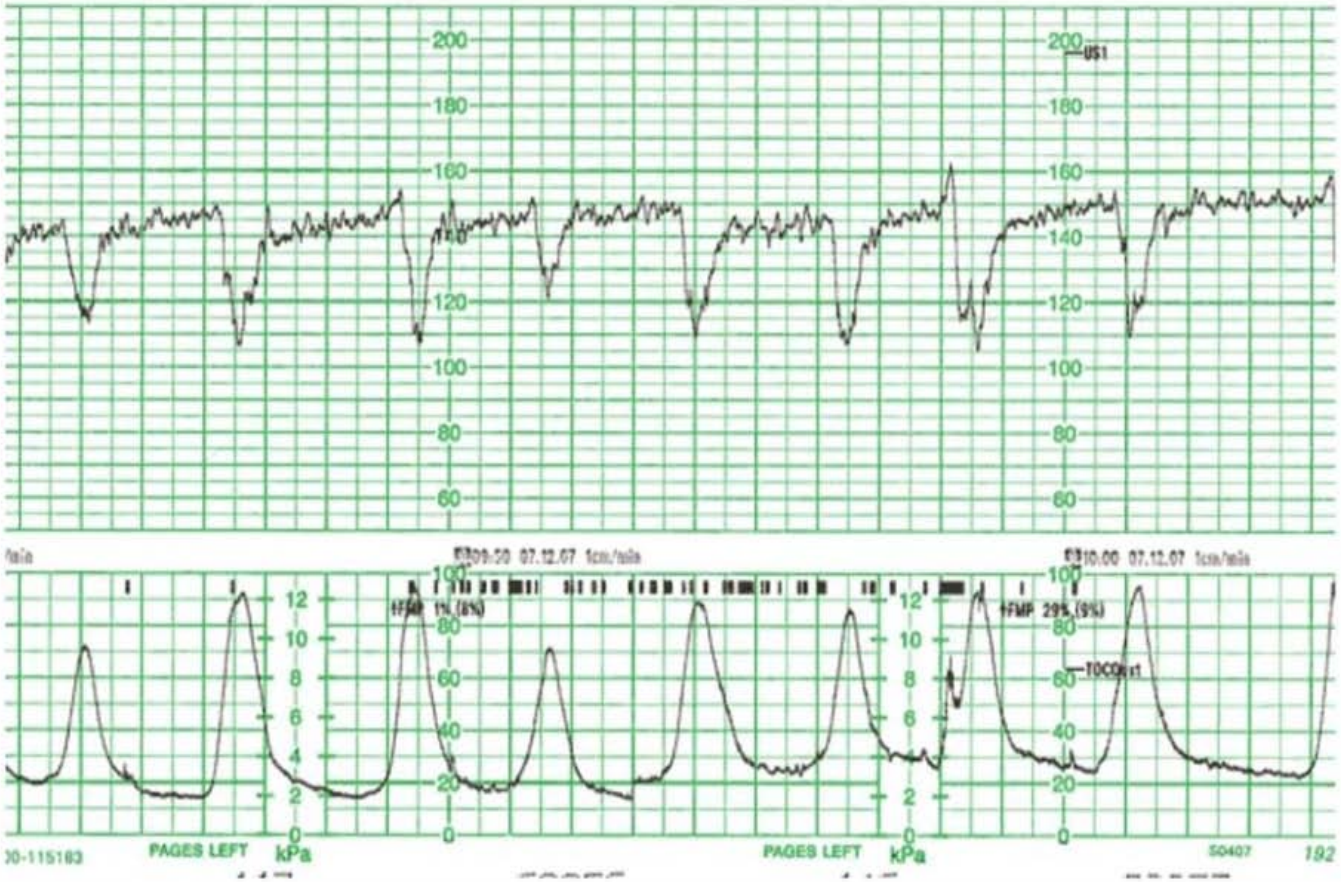

The following traces demonstrate prolonged decelerations. The first is in an antenatal setting where maternal repositioning (and possibly uterine activity) have resulted in a temporary cord compression with acute hypoxia and an isolated prolonged deceleration is the result. The trace quickly recovers.

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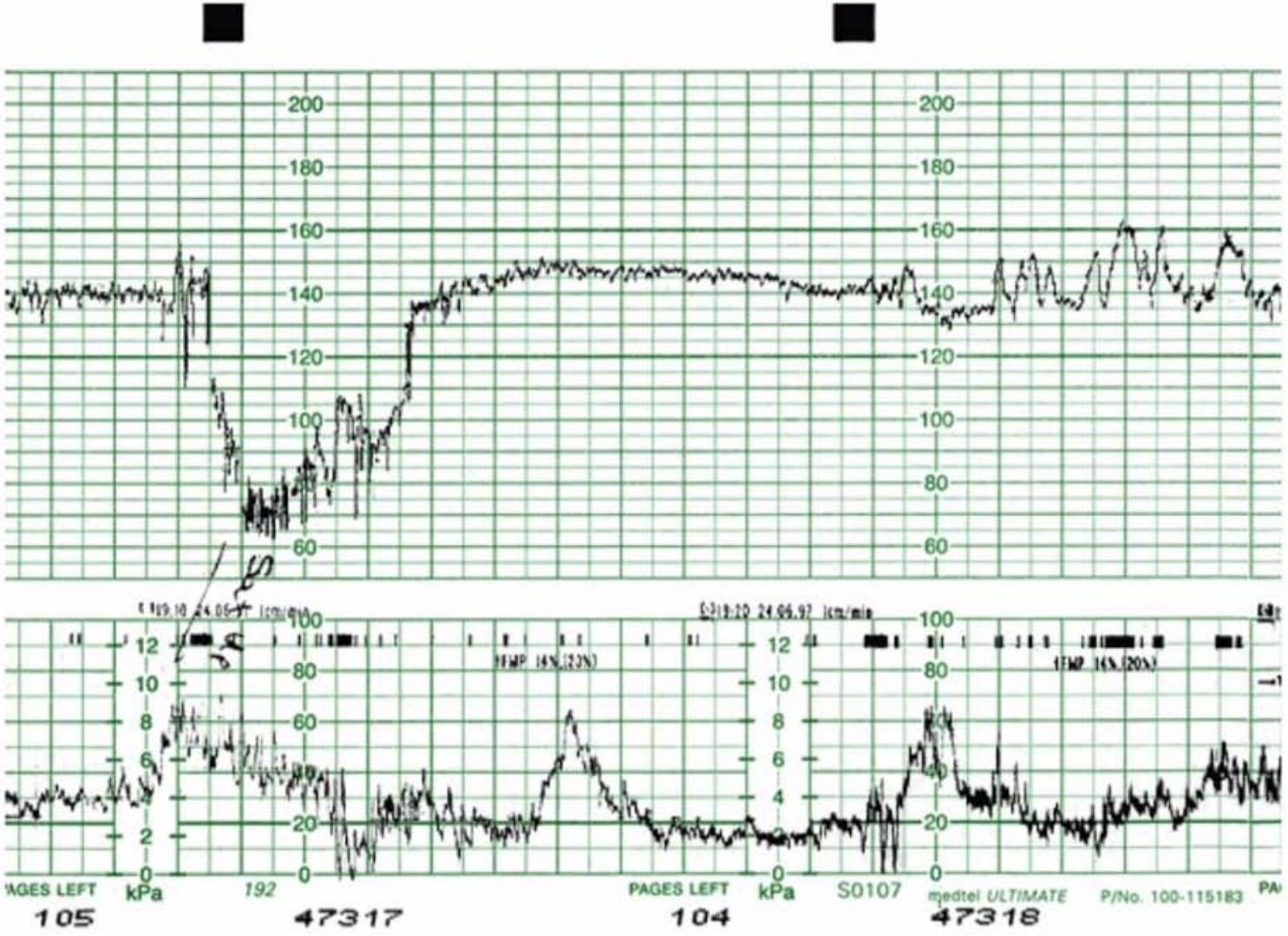

In this second trace, the decelerations are prolonged and reflect the fetal environment, that is the prolonged contractions, rather than the fetal condition. With a stable baseline and normal baseline variability, fetal well-being is assured. If the fetal metabolic requirements are not being met, a rising baseline fetal heart rate would be expected. Because of the uterine hyperstimulation, the fetus is at risk and close observation is required.

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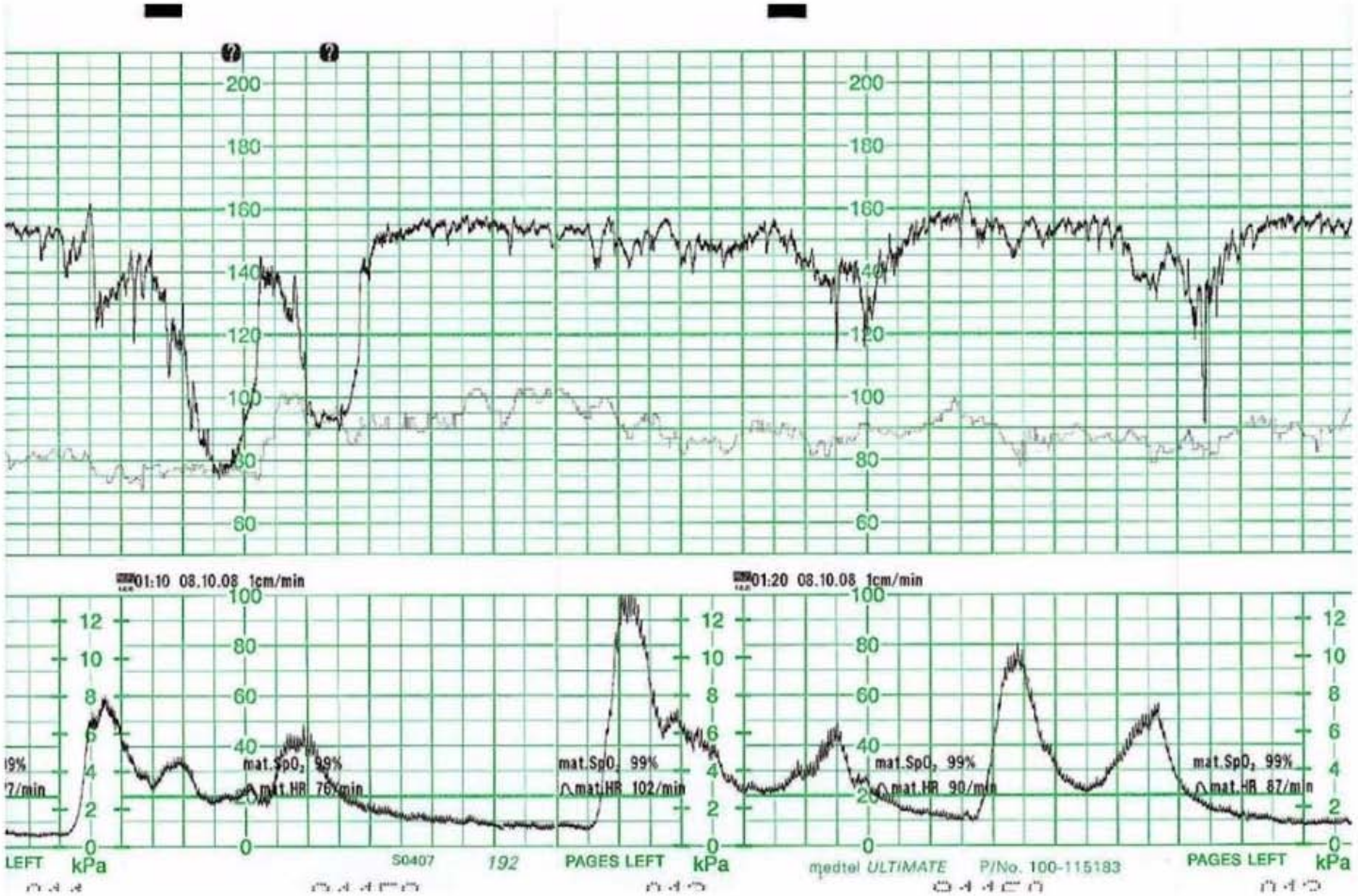

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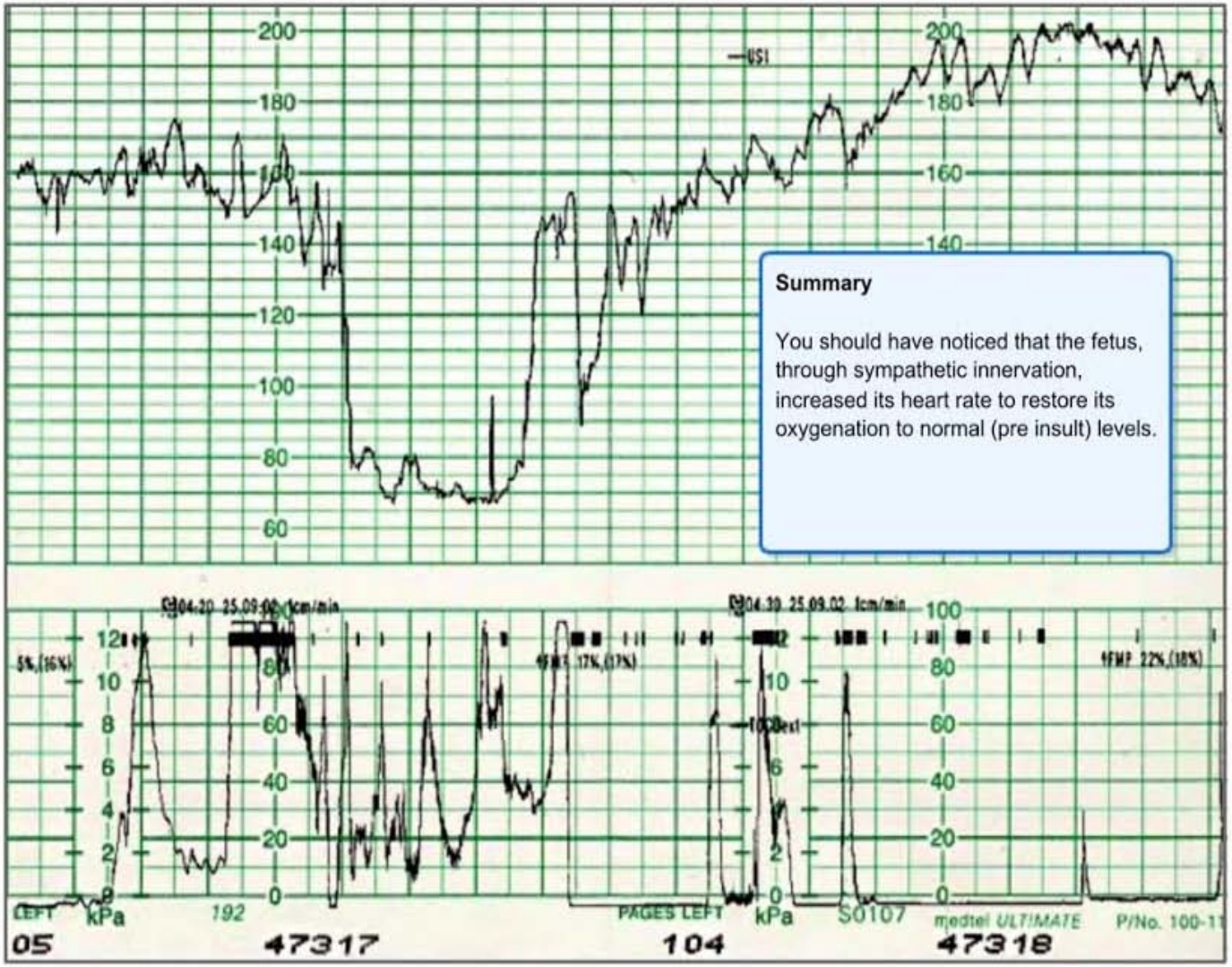
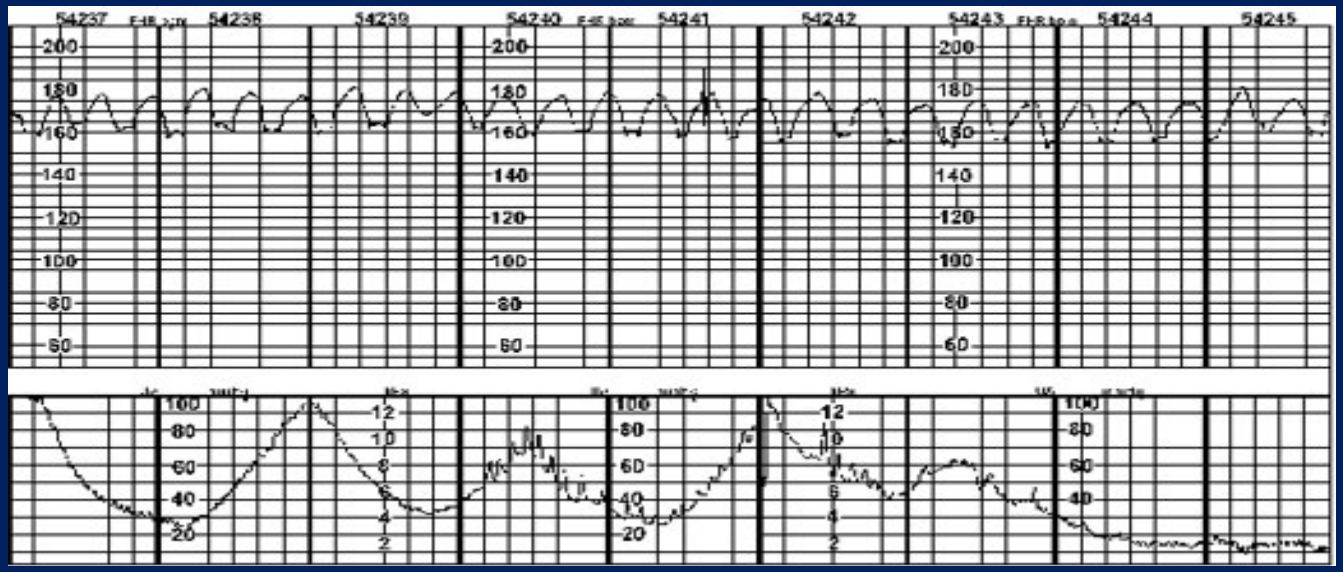
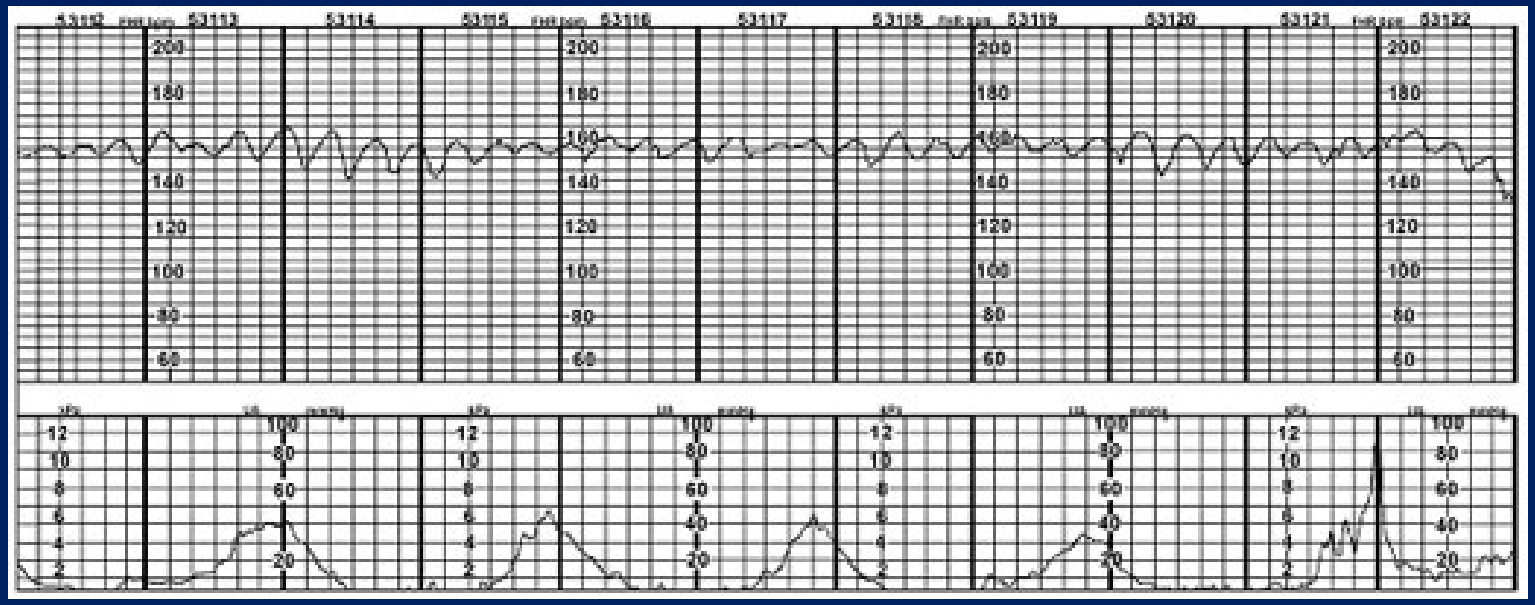


Trouble viewing the animation? CLICK HERE for an alternative version

This is a CTG where maternal hypoxia resulted from an epileptic fit. The increased maternal O_2 usage and reduced O_2 input has quickly impacted on the concentration gradient the fetus requires to take up the O_2 it needs. The fetal heart rate then drops rapidly in order to reduce the myocardial work load and prevent myocardial damage.



Pseudosinusoidal Fetal Heart Rate Pattern
An undulating pattern often appears within five minutes of intravenous narcotic administration and spontaneously resolves without intervention within 30 minutes. The pattern of undulation usually is interspersed with an occasional acceleration or period of moderate variability



Sinusoidal FHR pattern, demonstrated by a fetus with known erythroblastosis fetalis, continued to be present in the neonatal period and did not resolve with neonatal oxygen administration

CTG Interpretation: The normal baseline variability and stable fetal heart rate tells us that the fetus is well oxygenated. However, management is warranted because during the contractions the cord is being compressed and delivery of oxygen to the fetus compromised. Management should include maternal repositioning, assessment of maternal observations and notification of senior staff.

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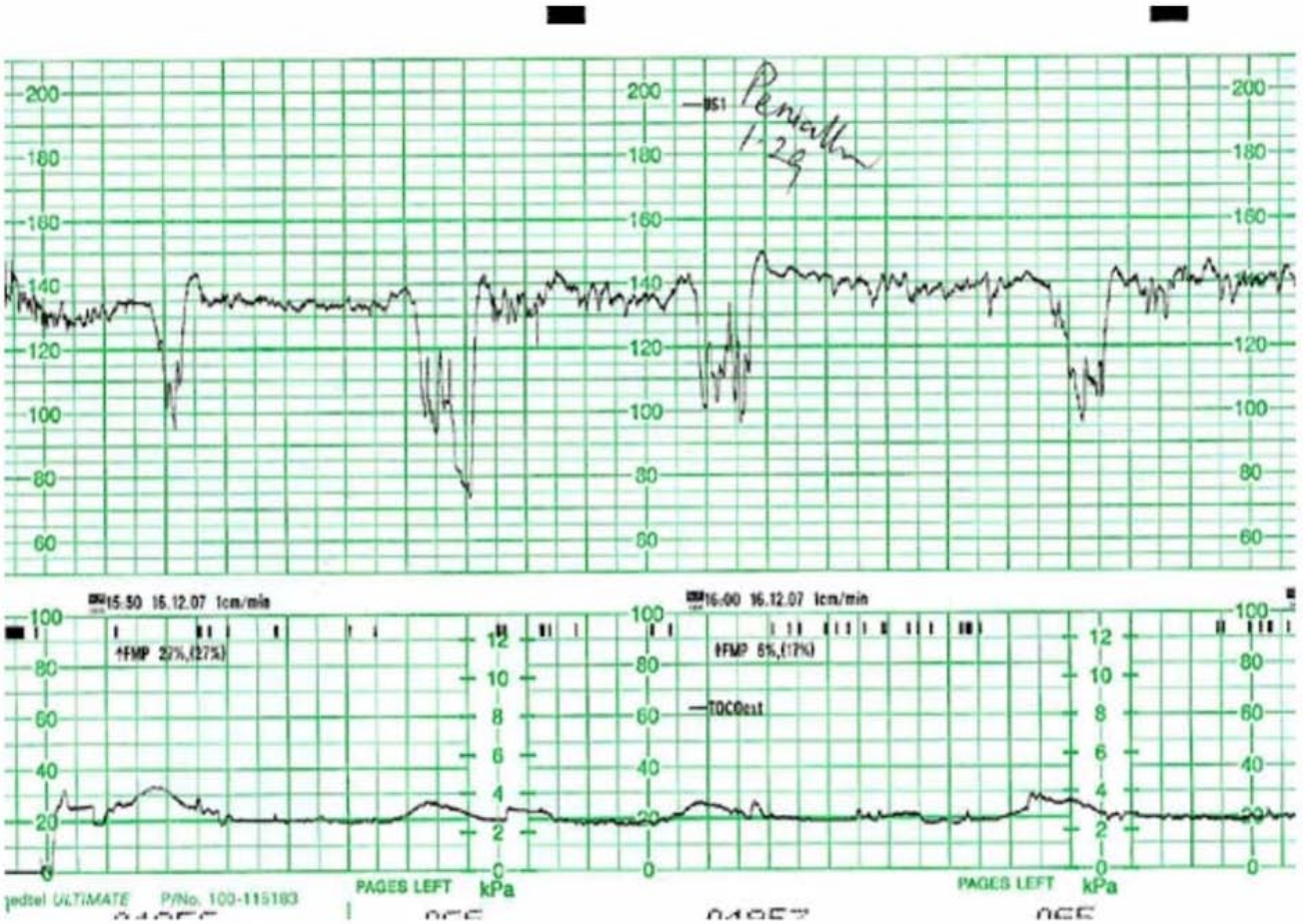

The following trace is the one from which the above animation was drawn. This a second stage trace and oxytocin is being used for augmentation. Maternal repositioning, reducing the oxytocic infusion and notifying senior staff would be appropriate in the first instance. Consideration may be given to tocolysis or delivery, depending on the overall clinical circumstance.

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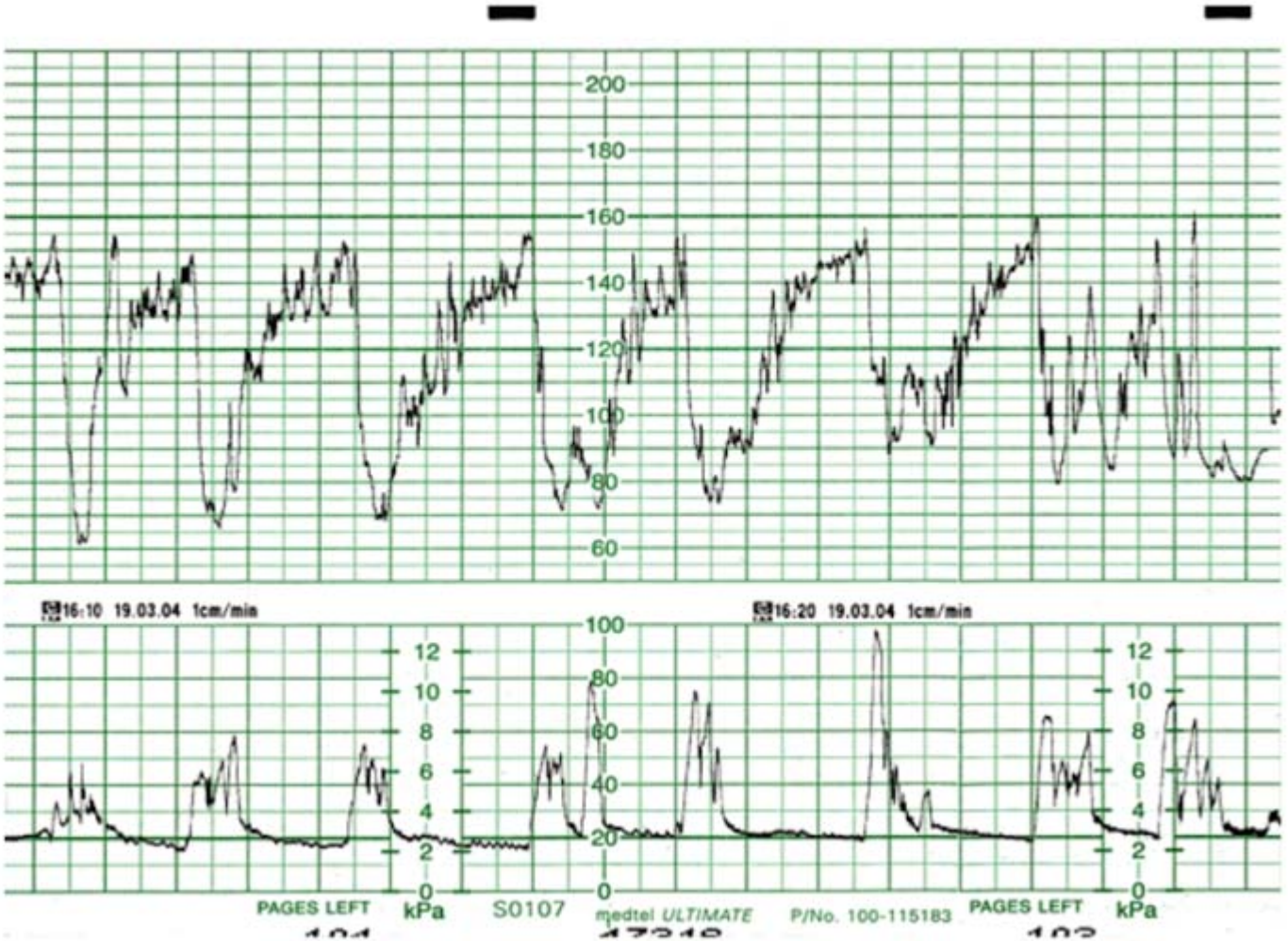

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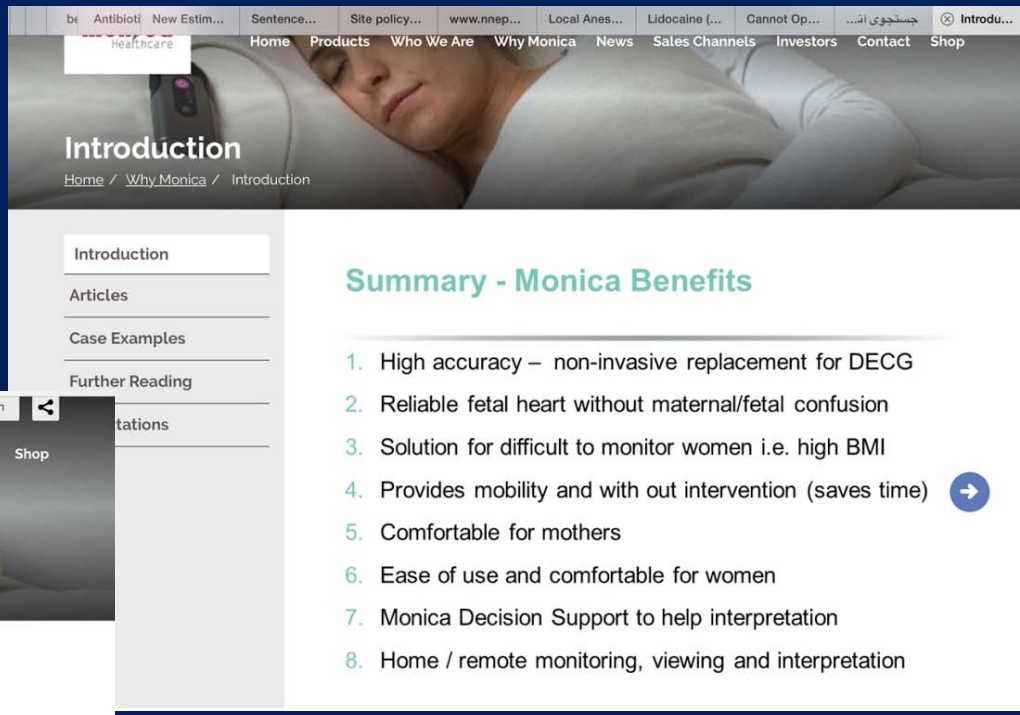
Topic outline: ▪ Fetal Physiology ▪ Fetal Assessment ▪ Fetal Surveillance ▪ CTG Normal ▪ Maternal Heart Rate	**Same topic outline as OFSEP** Additional items include: ▪ individual login with own username and password ▪ sequential compliance pathway to track individual progress ▪ short MCQ assessments after each module ▪ a score of 80% and above is required to move to next topic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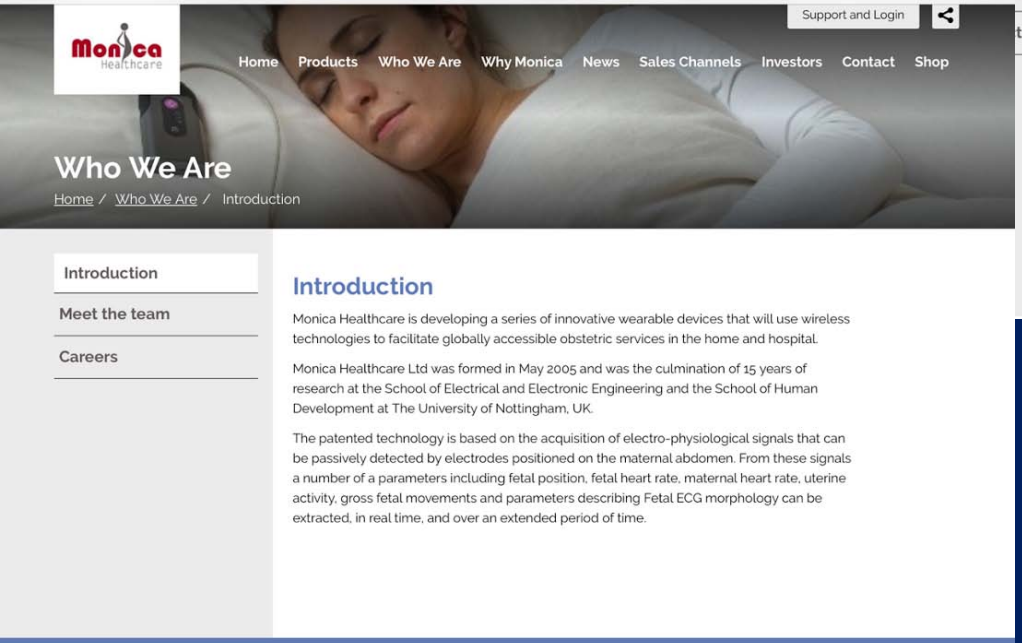
8. Home / remote monitoring, viewing and interpretation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Monica Healthcare Ltd was formed in May 2005 and was the culmination of 15 years of research at the School of Electrical and Electronic Engineering and the School of Human Development at The University of Nottingham, UK.
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