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This journal is published quarterly and available on-line with open access.

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The Sri Lanka Journal of Obstetrics and Gynaecology (SLJOG) is an editorially independent publication owned by the Sri Lanka College of Obstetricians and Gynaecologists (SLCOG). The journal publishes peer-reviewed work in all areas of Obstetrics and Gynaecology, including contraception, Urogynaecology, fertility, oncology and clinical practice.

Submission for Publication

Full instructions for submitting a manuscript can be found on the website at: <http://sljog.sljol.info/about/submissions>. *Please note that SLJOG does not accept paper submissions.*

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Editors who submit manuscripts, either as corresponding author or contributing author, take no part in any stage of the editorial processing of their manuscript.

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Subscription information

Copies of the journal are provided free of cost to life members of the SLCOG.
The electronic version of the journal is accessible free of charge at the website-
<http://sljog.sljol.info>.

Published by

The Sri Lanka College of Obstetricians & Gynaecologists,
No.112, Model Farm Road,
Colombo 08, Sri Lanka.
Tel +94 11 26 89 036 Fax +94 11 26 89 036
Email slcogoffice@gmail.com

Printed by

Ari Investments (Pvt) Ltd.
No 19, St. Joseph Road, Nugegoda, Sri Lanka.
Telephone +94 112 852410 Fax +94 112 822615
E-mail: ariyaw@gmail.com

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Malik Goonewardene

Intra-partum Fetal Monitoring – Cardiotocograph

Gunasena CGA^a, Jayasundara JMSW^b

Key words : Intra-partum fetal monitoring, Cardiotocography, Fetal scalp blood sampling, Fetal hypoxia

INTRODUCTION

The course of labour is the first challenge one ever undertakes. The uterine contractions during labour exposes the fetus to a possible risk of hypoxic brain injury due to repeated cord compression or reduction of utero-placental perfusion¹. Intrapartum fetal surveillance evolved with the principal aim of preventing adverse perinatal outcomes arising from fetal metabolic acidosis / cerebral hypoxia related to labour. However, the severity of an asphyxial injury is influenced by many factors (e.g. tissue perfusion, tissue substrate availability, fetal condition prior to the insult, duration of the insult and the severity of the insult). Therefore, the relationship between metabolic acidosis and cerebral injury is complex. Furthermore, it is clear that very often damage is actually sustained during pregnancy, prior to labour, rather than arising *de novo* during labour and delivery². In spite of this, intrapartum fetal surveillance for early detection of fetal hypoxia has become a key component of modern maternity care. Intrapartum fetal surveillance was traditionally carried out by intermittent auscultation (IA) of the fetal heart rate (FHR). This approach would be adequate to monitor a fetus at low risk of compromise, but may be inadequate for high-risk pregnancies. Therefore, the use of intrapartum electronic fetal monitoring (EFM) with cardiotocography (CTG), has steadily increased over the last three decades in an attempt to reduce the incidence of intrapartum fetal morbidity and mortality.

For low-risk women in labour, it has

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Competing interests: The authors report no conflict of interest

Table 1: Antenatal risk factors which would justify continuous intrapartum cardiotocography -adapted from RANZCOG Guidelines 2014 (2)

Abnormal antenatal CTG
Abnormal Umbilical Artery Doppler velocimetry
Suspected or confirmed fetal growth restriction
Oligohydroamnios or polyhydroamnios
Prolonged pregnancy ≥ 41 weeks
Multiple pregnancy
Breech presentation
Antepartum haemorrhage
Prolonged rupture of membranes
Known fetal abnormality requiring monitoring
Uterine scar
Hypertensive pregnancy
Hyperglycaemia in pregnancy requiring medication, or if it is poorly controlled, or if associated with macrosomia
Current or previous conditions which constitute a risk of fetal compromise e.g. cholestasis, isoimmunisation, substance abuse
Significantly reduced fetal movements preceding labour
Morbid obesity BMI ≥ 40
Maternal age ≥ 40

Table 2: Intrapartum factors which would justify continuous cardiotocography (2).

Abnormality detected on IA or CTG
Induction of labour with prostaglandin / oxytocin
Oxytocin augmentation
Regional anesthesia and paracervical block
Abnormal vaginal bleeding in labour
Maternal pyrexia ≥ 38 c
Absent liquor following amniotomy
Meconium or blood-stained liquor
Prolonged first stage of labour
Prolonged second stage of labour
Pre-term labour
Tachysystole
Uterine hypertonus
Uterine hyperstimulation

been suggested that the only clinically significant benefit from the use of routine continuous EFM in comparison to IA was in the reduction of neonatal seizures but with no statistically significant improvement in long-term outcomes such as cerebral palsy, although it increased the caesarean section and operative vaginal delivery rates³. Therefore, the use of continuous CTG for low risk pregnancies is not recommended⁴. It is also widely appreciated that there are still shortcomings in interpretation of CTG which is evident by the reviews of cases with poor perinatal outcomes⁵.

In order to improve the interpretation of CTG, it is important to understand the control of the fetal heart rate by the sinoatrial node, sympathetic and para-sympathetic autonomic nervous systems, baroreceptors, chemoreceptors, catecholamines and cardio-regulatory center. In addition to that, the physiology of fetal oxygenation and how the fetus reacts to hypoxia, the types of intra partum hypoxia (gradual, subacute, acute, chronic), the pathophysiological basis for CTG abnormalities, and the principles of EFM should also be clearly understood for decision making on intrapartum CTG^{6,7}. Because of the inherent limitations of the CTG, especially its poor specificity, newer techniques such as analysis of the ST segment of the fetal electrocardiograph (STAN), the calculation of the Fetal Physiological Score (FPS), and computer assisted interpretation of CTG are being studied⁸.

CARDIOTOCOGRAPHY

An Intrapartum CTG provides information about the fetal condition. A normal trace indicates a well-oxygenated fetus but an abnormal trace has poor specificity with upto 60% false positive rates being reported⁹. It has been suggested that FHR accelerations are a sign of a neurologically responsive fetus that does not have hypoxia/acidosis. However, the absence of accelerations in an otherwise normal CTG is of uncertain significance but it is unlikely to indicate hypoxia/acidosis¹⁰.

A mistake which could occur with intrapartum CTG is the recording of maternal heart rate, especially in the second stage of labour. A change in the pattern, sudden change in baseline rate and

accelerations coinciding with contractions may help in the differentiation from FHR.

TACHYSYSTOLE AND UTERINE HYPERTONUS

More than five uterine contractions in 10 minutes without FHR abnormalities is defined as tachysystole. Contractions lasting >2 minutes in duration or contractions occurring within 60 seconds of each other, without FHR abnormalities is referred to as uterine hypertonus². The management of these conditions includes reduction or cessation of oxytocin infusion and consideration of tocolysis.

UTERINE HYPERSTIMULATION

Tachysystole or uterine hypertonus in the presence of FHR abnormalities is defined as uterine hyperstimulation². If conservative measures such as cessation of oxytocin and tocolysis fail, urgent delivery needs to be considered. Commonly used protocols for acute tocolysis include terbutaline 250mcg iv or subcutaneous, salbutamol 100mcg iv, and glyceryl tri nitrate spray 400mcg sublingual.

REDUCED VARIABILITY

A bandwidth amplitude < 5 bpm for > 50 minutes in baseline segments, or for more than 3 minutes during decelerations indicates reduced variability. It can occur due to central nervous system hypoxia/acidosis, but it can also be seen with any condition which causes central nervous system (CNS) depression namely; deep fetal sleep, previous cerebral injury, infection, administration of CNS depressants or parasympathetic blockers, prematurity, and fetal abnormalities¹⁰.

INCREASED VARIABILITY (SALTATORY PATTERN)

A bandwidth of > 25 bpm lasting > 30 minutes indicates increased variability. Although its pathophysiology is poorly understood, it is thought to be due to a hyperactive fetal autonomic nervous system. It may be seen in fetal hypoxia associated with decelerations (10).

FETAL TACHYCARDIA

A baseline FHR > 160 bpm lasting > 10 minutes is considered as fetal tachycardia but is not associated with hypoxia in the presence of accelerations or with normal variability and absent decelerations¹⁰. Maternal fever and tachycardia, sympathomimetic medications (eg. terbutaline and salbutamol) chorioamnionitis, fetal tachyarrhythmias, a high inherent rate due to prematurity are some of the known causes of fetal tachycardia apart from fetal hypoxia.

FETAL BRADYCARDIA

A baseline value < 110 bpm lasting > 10 minutes is considered as fetal bradycardia. However a baseline FHR between 100 and 109 bpm with normal baseline variability and no variable or late decelerations should not prompt any further action¹¹. The causes of fetal bradycardia include low inherent rate, medications (e.g. local anesthetics), maternal hypothermia, maternal hypotension, fetal heart conduction defects, prolonged umbilical cord compression, and sustained hypoxia¹⁰.

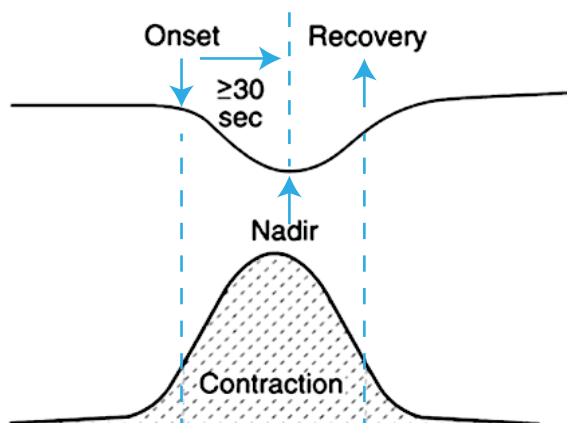


Figure 01: Early deceleration

DECELERATIONS

Decelerations are defined as decreases in the FHR of > 15 bpm below the baseline, and lasting > 15 seconds. Early decelerations are uniform in shape and they start and finish with the contraction. They probably occur due to head compression and usually the decrease is < 20 beats from baseline. They do not indicate fetal hypoxia or acidosis.

VARIABLE DECELERATIONS

Variable decelerations represent a baroreceptor-mediated response to increased arterial pressure, as occurs with umbilical cord compression. They are variable in shape, depth, duration and timing with the contractions and exhibit a good variability within the deceleration. They typically have a rapid onset (onset to nadir < 30 seconds) and a rapid recovery to baseline. Variable decelerations constitute the majority of decelerations during labour. They are seldom associated with an important degree of fetal hypoxia/acidosis. Increases in FHR immediately before and after a variable deceleration have been referred to as “shoulders.” These increases can be visually similar to accelerations, and this led to speculations that they had a similar predictive value. However, there is inadequate evidence to support the notion that “shoulders” have the same predictive value as accelerations¹².

Variable decelerations were formerly categorized as ‘typical’ if they were considered to be normal and not indicating fetal hypoxia and ‘atypical’ if they were considered to be abnormal and indicating probable fetal hypoxia. Although the terms ‘typical’ and ‘atypical’ are not currently used, the non-reassuring features in variable decelerations which require appropriate action, as they indicate the likelihood of fetal hypoxia, have been clearly described^{2,6,11}. These non-reassuring features which are also referred to as ‘complicated variable decelerations’ include a persistently large amplitude (> 60 bpm in depth) and / or long duration (> 60 seconds duration), a slow return to the baseline after the contraction, smooth (with no baseline variability) post-deceleration shoulders (“overshoots”) often associated with a rising baseline or a baseline tachycardia and a reduced baseline variability⁶.

Variable Deceleration

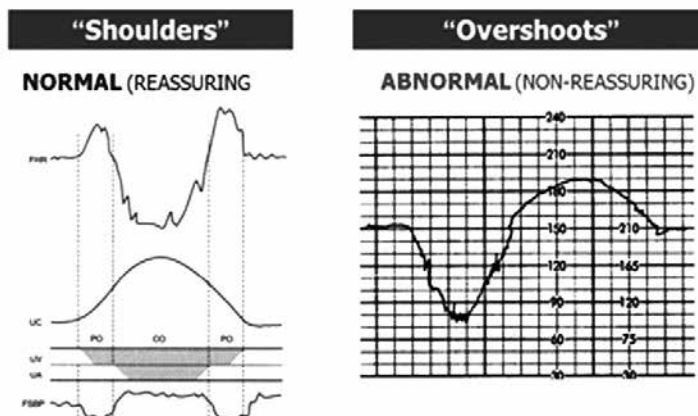


Figure 02: Shoulders and Overshoots in variable decelerations

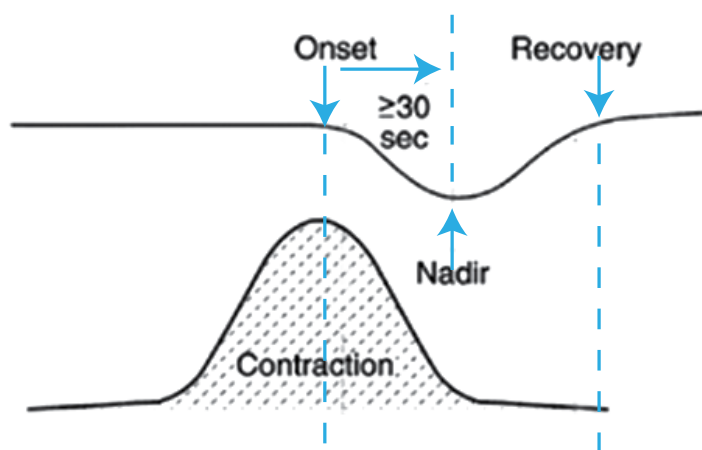


Figure 03: Late deceleration

LATE DECELERATIONS

Late decelerations are caused by contractions in the presence of fetal hypoxia. These are uniform and repetitive. Late decelerations start after the start of the contraction and the nadir of the deceleration is more than 30 seconds after the peak of the contraction. They return to the baseline after the contraction has finished. Late decelerations of any depth are significant and should be immediately attended to. In fact shallow decelerations (with decreases of < 10 bpm) with reduced baseline variability are particularly dangerous and indicate significant fetal hypoxia. These can even be detected with careful IA of FHR immediately after uterine contractions, but not in between uterine contractions when the FHR has returned to its normal baseline

PROLONGED DECELERATIONS

Decelerations lasting > 3 minutes are defined as prolonged decelerations. They may indicate chemoreceptor-mediated hypoxaemia. Decelerations exceeding 5 minutes, with FHR less than 80 bpm and reduced variability within the deceleration are frequently associated with acute fetal hypoxia/acidosis and require urgent intervention¹⁰.

SINUSOIDAL PATTERN

This is an oscillating pattern resembling a sine wave (very smooth with a regular cycle rate). It has a relatively fixed period of 3-5 cycles per minute and typically an amplitude of 5-15 beats. Its pathophysiology is poorly understood but

it may be seen with severe fetal anemia (fetal-maternal haemorrhage, twin-to-twin transfusion syndrome, anti-D alloimmunization and vasa previa) It has also been described in cases of acute fetal hypoxia, infection, cardiac malformations, hydrocephalus, and gastroschisis¹⁰.

There is agreement about baseline FHR and fetal tachycardia, with FIGO describing a time frame too. Although NICE Guidelines describe FHR of 160–180 bpm as non-reassuring and FHR > 180 bpm as pathological, FIGO Guidelines do not sub categorize fetal tachycardia in this manner. A fetal tachycardia (FHR > 160 bpm) may be secondary to fetal compensatory response to evolving hypoxia. A mere increase in

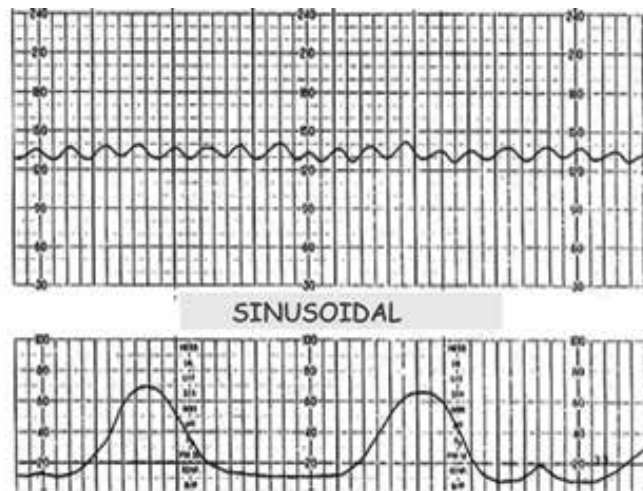


Figure 04: Sinusoidal pattern

Table 3. Comparison of guidelines on cardiotocography

Category	FIGO	NICE	RANZCOG
Baseline heart rate	110-160 bpm	110-160 bpm	110-160 bpm
Tachycardia	>160 bpm for more than 10 minutes	>160 bpm	>160 bpm
Bradycardia	<110 bpm for more than 10 minutes	<110 bpm	<110 bpm
Normal Baseline variability	5 – 25 bpm	5 bpm or more	6 – 25 bpm
Reduced variability	<5 bpm for more than 50 minutes	<5 bpm for 30 – 90 minutes (non-reassuring) <5 bpm for more than 90 minutes (abnormal)	3 – 5 bpm
Absent variability	no comment	no comment	< 3 bpm
Increased variability	>25 bpm for more than 30 minutes	no comment	>25 bpm
Accelerations	The presence of accelerations denotes a fetus that does not have hypoxia/acidosis, but their absence during labor is of uncertain significance	The presence of fetal heart rate accelerations is generally a sign that the baby is healthy. The absence of accelerations in an otherwise normal cardiotocograph does not indicate acidosis	Absence of accelerations in isolation is unlikely to be associated with fetal compromise
Prolonged decelerations	Deceleration >3 minutes	Deceleration >3 minutes	Decelerations > 90 seconds but less than 5 minutes

the baseline FHR > 160 bpm by itself is not associated with fetal compromise as it could be physiological (preterm due to the immaturity of the parasympathetic nervous system), or could occur secondary to maternal dehydration, drugs (eg. sympathomimetics). However, any increase in the fetal heart rate associated with changes in baseline variability is considered to be abnormal. In addition, if the original baseline fetal heart rate was 105 bpm, even an increase in FHR to 145 bpm should be considered abnormal for that fetus.

The FIGO Guidelines consider FHR < 100 bpm as pathological and RANZCOG Guidelines also describe prolonged bradycardia of FHR < 100 bpm for more than 5 minutes as a likely feature of fetal compromise. It has been suggested that a stable baseline fetal heart rate between 90 and 99 bpm with normal baseline variability (having confirmed that this is not the maternal heart rate) may not be pathological¹¹. However, it is essential that patients with such CTGs are evaluated properly by an experienced senior obstetrician.

There is no significant difference in the definitions of normal baseline variability and reduced variability, between the different guidelines. However, the NICE Guidelines further categorize reduced baseline variability being non-reassuring or abnormal according to the duration, which would be more useful in clinical decision making.

INTRAPARTUM FHR INTERPRETATION AND MANAGEMENT; A STEP-WISE PHYSIOLOGIC APPROACH

An Intrapartum CTG, just like all investigations / tests in clinical practice, should be interpreted in the background of the total clinical picture which should include the patient's clinical details, indication for EFM, previous CTGs, and results of all previous other investigations including sonological assessments. All the other information such as medications, oxytocin infusions, stage of labour and progress of labour must also be considered. **MOTHERS** is a mnemonic which could be useful when considering the clinical picture; **M**econium stained liquor, use of **O**xytocin, maternal **T**emperature

(infection), **H**yperstimulation / **H**aemorrhage, **E**pidural, **R**ate of progress in labour, and **S**car³.

STEP 1—THE NORMAL AND THE ABNORMAL INITIAL CTG

If the CTG is normal the fetus is very likely to be neurologically intact, normoxic, without acidaemia or acidosis, at low risk of intrapartum asphyxia, and is able to react and defend itself against intrapartum hypoxia. Surveillance may continue depending on the situation or the woman may be monitored by IA¹³.

STEP 2—RECOGNITION OF THE COMPENSATED AND THE DECOMPENSATING FETUS

An intact fetus with a previously normal CTG will exhibit predictable patterns of FHR responses if exposed to hypoxic ischaemic insults during labour. Based on the intensity and duration of hypoxic stress during labour, three types of intrapartum hypoxia had been described, namely: gradually evolving hypoxia; subacute hypoxia; and acute hypoxia. The management should be tailed according to the type of hypoxia to optimize fetal outcome⁷.

GRADUALLY EVOLVING HYPOXIA

The hypoxic stress evolves over time (hours) giving the fetus time to use its compensatory mechanisms effectively in order to prevent hypoxic damage. The first feature on CTG is the presence of decelerations with contractions. If the hypoxic insult continues, the decelerations will be followed by ABCDE.

Disappearance of Accelerations

Increase in Baseline heart rate

Compensated stress

Decompensation

End stage

The accelerations disappear in order to conserve oxygen and energy substrates in the fetus. The catecholamine release increases the FHR and cardiac output to maintain perfusion to the vital organs. Therefore, a sustained FHR tachycardia in association with uterine contractions is a sensitive marker of a compensatory

increase in fetal cardiac output. If the hypoxic insult continues, depending on the fetal reserve and the intensity and duration of hypoxia, fetal decompensation may ensue. When the perfusion to the brain is compromised, loss in the baseline variability would be observed in the CTG trace. Finally, myocardial hypoxia and acidosis may lead to a terminal bradycardia resulting in the 'step-ladder pattern to death'⁷.

The management should be tailored according to the level of hypoxia. In the presence of a stable baseline in between decelerations and normal baseline variability, labour can be continued with continuous CTG monitoring. If raised baseline and or abnormal variable or late decelerations appear on the CTG, care should be taken to improve fetal environment (intrauterine resuscitation) which may include stopping or reducing oxytocin, iv fluids and placing patient in the left lateral position. If baseline variability is reduced despite conservative measures, immediate delivery should be considered⁷.

ACUTE HYPOXIA

This is characterized by a sudden drop in the baseline heart rate, which is also known as a 'single prolonged deceleration'. This could be either suspicious (lasting for < 3 minutes and returning to the normal baseline with good variability) or abnormal (lasting for > 3 minutes)

First, it is essential to exclude three major intrapartum accidents (placental abruption, umbilical cord prolapse and uterine rupture) and two iatrogenic causes (hyper-stimulation due to oxytocin or prostaglandins and maternal hypotension usually secondary to supine hypotension or epidural analgesia). In case of intrapartum accidents, delivery should be expedited via the safest and quickest way to save the fetus.

If acute hypoxia is considered to be due to an oxytocin infusion, 'intrauterine resuscitation' should be initiated immediately. In the presence of normal variability prior to deceleration and within the first three minutes of deceleration and the three accidents mentioned above are absent, unto 90% of the prolonged decelerations have been reported to recover by 6 minutes and 95% by 9 minutes¹⁴.

In case of acute hypoxia occurring in the absence of intrapartum accidents or iatrogenic causes, the '3, 6, 9, 12 and 15-minute' rule, which includes the following, should be applied: if a normal baseline variability has been noted before the onset of deceleration and within the first 3 min of the deceleration, appropriate intrauterine resuscitation by 6 min, moving the patient to a theatre by 9 min, and if the CTG shows no signs of recovery, commencing delivery by 12 min with the aim of delivering the baby by 15 min¹⁵.

Reduced baseline variability before or within the first 3 min of the deceleration, repetitive late decelerations before the onset of the prolonged deceleration or a drop in the heart rate to >60 bpm are associated with a poor outcome. In these circumstances, the '3, 6, 9, 12 and 15-minute' rule should not be applied, and immediate delivery should be undertaken⁷.

SUBACUTE HYPOXIA

This is characterized by complicated variable decelerations, with the amplitude of the deceleration ≥ 60 bpm and lasting for ≥ 90 seconds. When the FHR returns to its baseline in subacute hypoxia, it spends less than 30 seconds at the baseline level before the onset of the next deceleration^{7,16}. Therefore, the time available at the baseline to wash off the acid and carbon dioxide and to obtain fresh oxygenated blood from the placenta becomes progressively shorter. Therefore, a rapidly cumulative build up of CO₂ takes place which results in an initial respiratory acidosis and a subsequent metabolic acidosis. The baseline FHR may remain within the normal range (110-160 bpm) as the fetus is unable to raise its baseline heart rate because of the short duration of time spent at the baseline in between two decelerations. Subacute hypoxia is associated with a rapid decline

in pH, usually at the rate of 0.01 every 2-4 minutes, in contrast to the gradually evolving hypoxia¹⁶.

Once subacute hypoxia is established, there is likely to be insufficient time to obtain, analyze and react to a fetal blood sample result without the risk of severe acidaemia. Therefore, in clinical practice, it is crucial to recognize this pattern¹⁶.

The management involves in utero resuscitation and discouraging active maternal pushing for the next few contractions to ensure oxygenation of placental venous sinuses. Immediate delivery should be considered if changes are not reversed with conservative measures⁷.

CHRONIC HYPOXIA

In this situation, the fetus has been exposed to a prolonged period of hypoxia with or without resultant neurological injury during the antenatal period before the onset of labour. This happens usually secondary to chronic utero-placental insufficiency. The fetus adapts several compensatory mechanisms for survival including reduction in growth, movements and diversion of oxygenated blood and nutrients from non-vital organs to supply the vital organs⁷.

The affected fetus may be identified by the features observed on the CTG; increase in the baseline rate with reduced variability and the presence of shallow decelerations. Even though some degree of cerebral damage may have already taken place, the presence of this CTG pattern requires immediate delivery. This is because, there will be further reduction in oxygenation of the fetus with the onset of labour due to uterine contractions, intermittent umbilical cord compression and reduction in utero-placental circulation. This will eventually lead to hypoxic ischaemic encephalopathy, terminal bradycardia and

fetal death⁷.

FETAL SCALP BLOOD SAMPLING (FBS)

This is a test to assess the acid-base status of the fetus. The units employing EFM are encouraged to have access to FBS facilities to aid in the management of labours where fetus shows equivocal CTG changes. FBS can be assessed for pH and lactate levels. The incidence of false positives from an abnormal CTG can be reduced with FBS. A reduction in the total caesarean section rate from 18% to 11% and caesarean section indicated by fetal distress from 7% to 3%, when fetal scalp blood sampling was allowed, has been reported¹⁷.

The most important question is the identification of the appropriate clinical situation for FBS. Delivery must be expedited and FBS should not be undertaken, if there is clear evidence of serious and/or sustained fetal compromise. Delivery also needs to be expedited if CTG abnormalities are of a degree requiring further assessment, but FBS is unavailable or contraindicated.

CONTRAINDICATIONS TO FBS

1. Evidence of serious, sustained fetal compromise.
2. Fetal bleeding disorders (e.g. suspected fetal thrombocytopenia, haemophilia).
3. Face or brow presentation.
4. Less than 34 weeks of gestation.

Maternal infection.

(e.g. HIV, hepatitis B, hepatitis C, herpes simplex virus and suspected intrauterine sepsis). Group B Streptococcus carrier status does not preclude FBS².

If facilities are available, for medico legal

Table 04: Normal and abnormal values of pH and Lactate on Fetal Scalp Blood Sampling (2, 11)

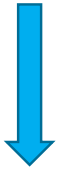
pH	Lactate	Management
≥ 7.25	≤ 4.1 mmol/l	Normal
7.24 – 7.21	4.2 – 4.8 mmol/l	Suspicious (Repeat in 30 minutes)
≤ 7.20	≥ 4.9 mmol/l	Abnormal (Recommend delivery)

Types of Intrapartum Hypoxia

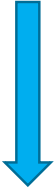
Acute Hypoxia

Prolonged deceleration < 80 bpm for > 3 minutes

Sudden baseline drop > 60 bpm following gradually evolving or subacute hypoxia



pH drops at the rate of 0.01/minute



Management

Exclude 3 intrapartum accidents (abruption, uterine rupture and cord prolapse)

Present

Immediate delivery (category 1)

Uterine relaxants if delay in delivery anticipated

Absent

Gradually Evolving Hypoxia

Starts with decelerations followed by **ABCDE**

Accelerations disappear

Baseline variability increases

Compensated stress

Decomensation

End stage



Rate of pH drop depends on duration and intensity of hypoxia and fetal reserves



Management

If stable baseline in between decelerations and good variability
Continue Labour

If raised baseline and /or complicated or late decelerations
Improve environment (stop / reduce oxytocin, IV fluids, Left lateral)

If baseline variability reduced despite conservative measures
Immediate delivery

Exclude hyperstimulation (stop oxytocin +/- uterine relaxants)

IV fluids

Left lateral position

Apply “3, 6, 9, 12, 15,” rule

Subacute Hypoxia

A fetus typically spends < 30 s at the baseline and > 90 s below the baseline.

Recurrent complicated variable or late decelerations with or without overshoots and saltatory pattern in between decelerations



pH drops at the rate of 0.01/2-3 minutes



Management

Improve environment (stop / reduce oxytocin, IV fluids, Left lateral)

Stop active pushing to improve utero-placental circulation

Immediate delivery if changes are not reversed with conservative measures

Figure 05: Types of Intrapartum Hypoxia (7)

purposes, paired (arterial and venous) umbilical cord blood gas and lactate analysis should be undertaken within one hour of delivery if any of the following are present:

- a. Apgar score < 4 at 1 minute.
- b. Apgar score < 7 at 5 minutes.
- c. Fetal scalp sampling performed in labour.
- d. Operative delivery undertaken for fetal compromise.

CONCLUSION

Intra partum care was expected to take a new turn with the introduction of EFM 30 years back. Decades later, however, the results are not too convincing. Despite questions about its efficacy and outcomes associated with its use, FHR monitoring continues to be the predominant method for intrapartum fetal surveillance. A considerable proportion of asphyxial injury is thought to occur before the start of labour and EFM may not offer additional benefit for those fetuses. The challenge of EFM is to identify early, those fetuses who are compromised and to intervene before injury occurs. The various mechanisms which control the fetal heart rate, the physiology of fetal oxygenation and how the fetus reacts to hypoxia, the types of intra partum hypoxia, the pathophysiological basis for CTG abnormalities, and the principles of EFM need to be well understood in order to improve the interpretation of CTG and facilitate intra partum decisions

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Using the internal inguinal ring as a landmark for the safe placement of secondary trocars at laparoscopy

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Abstract

Key words

Inferior epigastric vessels; laparoscopic vascular injuries; trans-illumination; internal inguinal ring

Aim

Avoidance of injury to the epigastric vessels is an important safety consideration in siting secondary ports at laparoscopy. We evaluated trans-illumination, direct visualization of vessels and using the internal inguinal ring, which has a constant relationship with the inferior epigastric vessels as an anatomical landmark to locate these vessels.

Methods

Ninety consecutive women undergoing laparoscopy were recruited for the study. The Ability to trans-illuminate the superficial and inferior epigastric vessels, to visualize inferior epigastric vessels and to locate the round ligament entering the internal inguinal ring were documented. The body mass index (BMI) was noted. The association of the body mass index (BMI) with these variables was calculated by simple regression analysis.

Results.

Ability to visualize the internal inguinal ring was significantly higher than that of locating the superficial or inferior epigastric vessels by trans-illumination or by direct visualization. $p < 0.001$.

Ability to visualize and to trans-illuminate the inferior epigastric vessels decreased with increasing BMI ($p < 0.05$). There was no significant relationship between body mass index and the ability to visualize the round ligament entering the deep inguinal ring. ($p = 0.64$)

Conclusion

Locating the deep inguinal ring and using it as an anatomical landmark and placing the lateral trocars lateral to a sagittal line originating from the internal ring is a reliable and useful method in preventing unintended injury to anterior abdominal wall vessels during secondary trocar placement

to account for one of the two fatalities reported in their series⁴. In addition, significant morbidity and litigation results from damage to abdominal wall vessels as well⁵. In a study done on entry-related laparoscopic injuries in Finland, it was found that 10% of all entry related injuries occurred due to small vessel injuries with one case needing laparotomy to achieve hemostasis⁶. Injury to epigastric vessels could also create a nuisance, with blood trickling down to the operative field⁷.

In most patients it is possible to avoid these injuries by identifying the vessels by direct laparoscopic visualization and by trans-illumination⁸. Standard teaching dictates that the inferior epigastric vessels should be identified either by trans-illumination or direct visualization⁹. Trans-illumination can often identify the superficial vessels but except in the extremely thin individuals, trans-illumination of inferior epigastric vessels are difficult⁸. Direct visualization of the inferior epigastric vessels by laparoscope is also not possible in all patients, especially in the obese⁷.

It has been suggested that other landmarks may have to be considered prior to insertion of secondary trocars for the above reasons⁸. Inferior epigastric vessels arise from the external iliac vessels just before they enter the femoral canal¹⁰. They then run on the medial margin of the internal inguinal ring to run along the under surface of the lateral third of the rectus abdominis muscle¹⁰. Its course can be surface marked on the anterior abdominal wall in thin individuals from a point midway between the pubic symphysis and anterior superior iliac spine¹⁰. As it runs superiorly its distance from the midline reduces. The distance from the midline to the inferior epigastric vessels at the level of the pubis is 7.5 cm while at the level of the umbilicus it is 4.6cm¹. Since its relationship to the medial margin of the internal ring is consistent, entry of the round ligament provides a convenient pointer to the internal inguinal ring. At laparoscopy, displacement of the uterus in the opposite direction using

INTRODUCTION

With evolution of technology and instruments, laparoscopy has achieved a preeminent role in gynaecologic surgery.

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Competing interests: The authors report no conflict of interest

Vascular injuries are one of its dreaded complications. While avoidance of injuries to major vessels such as the aorta, inferior vena cava and iliac vessels has received a great deal of attention, avoidance of injury to the vessels situated in the anterior abdominal wall has received less attention.

Abdominal wall vessels injuries occurs in 0.2-2% of all procedures^{1, 2}, almost exclusively during placement of secondary ports. Both the superficial epigastric and inferior epigastric vessels are at risk³ and injuries to these vessels could be potentially lethal. In a large multicenter observational study, Jansen et al. found injury to the inferior epigastric vessels to be the most common complication encountered and

the uterine manipulator will make this identification easier.

Therefore, if the lateral trocars are placed lateral to a vertical line originating and extending cephalad from the internal inguinal ring, injury to the inferior epigastric vessels could be avoided.

The objective of this study was to determine the effectiveness of trans-illumination, direct visualization and using the internal inguinal ring as an anatomical landmark to locate the epigastric vessels to safely place secondary ports. We also compared the value of each of these methods with variation of body mass index (BMI).

METHOD

A prospective descriptive study was conducted at the Professorial Gynaecology unit of the Teaching Hospital, Peradeniya, Sri Lanka. Ethical clearance was obtained from the Ethics Review Committee of the Faculty of Medicine of the University of Peradeniya. Informed written consent was obtained from all the participants in the study. Ninety consecutive women undergoing laparoscopy for gynaecologic indications were included in the study. The body weight in kilograms and the height in centimeters, was recorded prior to surgery. The Veress needle was inserted trans-umbilically to create the pneumo-peritoneum in all patients and the abdominal cavity insufflated with carbon dioxide gas to a filling pressure of 20 mmHg. A 10mm trocar was inserted via the umbilicus and the laparoscope introduced through this trocar.

The ability to identify superficial and inferior epigastric vessels by trans-illumination and the ability to directly visualize the inferior epigastric vessels by laparoscopy were recorded. Trans-illumination was done by switching off the theatre lights and visualizing the vessels through the skin while holding the laparoscope intra-abdominally with the laparoscope light on. And direct visualization was done using the laparoscope to visualize the inferior epigastric vessels running underneath the peritoneum in the anterior abdominal wall. The ability to locate the round ligament entering the deep ring and visualization of the origin of the inferior epigastric vessels intra-abdominally by laparoscopic inspection was also recorded.

To differentiate between superficial and deep vessels the assistant applied firm perpendicular pressure over the vessel located via trans-illumination. Laparoscopically it was determined whether this coincided with the direction of the inferior epigastric vessels⁸. If the inferior epigastric vessels were visualized directly it was also inspected for 5 to 8 cm above the pubic symphysis since this area has been shown to be ideal for placement of the trocar¹¹. This was done on both sides of the abdomen. The rest of the surgical procedure was done according to the surgery being carried out.

Proportions of cases with positive results were compared using the chi-square test. A *p* value of <0.05 was considered to be significant. The relationship of the effectiveness of trans-illumination, direct visualization and the use of the internal inguinal ring as an anatomical landmark to locate the epigastric vessels with changes in BMI was evaluated using simple regression analysis. The statistical package Minitab (Version 15) was used.

RESULTS

A total of 90 women with a mean age of 31.8 years (range 21-44 years, SD; 4.95) were enrolled. The mean BMI was 23.9 kg/m² (range 15.6-43 kg/m², SD; 4.93) with 55 having a BMI <25 kg/m² and 35 having a BMI >25 kg/m².

Internal inguinal ring was successfully visualized in 84 (94%) patients, whereas the ability to trans-illuminate superficial epigastric vessels was successful in 50% of the cases and the ability to transilluminate inferior epigastric vessels was successful only in 30% of cases. Inferior epigastric vessels were directly visualized in 56% of cases of which 38% of cases it was also visualized 5cm-8cm above the pubic symphysis which is the area commonly

used to insert the lateral trocars. (Table 1)

Ability to visualize the internal inguinal ring was significantly higher than the ability to visualize superficial or inferior epigastric vessels either by trans-illumination or directly from the laparoscope (*p* < 0.001). (Table 2). Ability to trans-illuminate the superficial epigastric and inferior epigastric as well as ability to visualize the inferior epigastric vessels directly from the laparoscope decreased with increasing BMI (*p* < 0.05). However there was no significant relationship between the BMI and the ability to visualize the round ligament entering the deep inguinal ring. (*p* = 0.64). (Table 3).

DISCUSSION

Visualization of the internal inguinal ring was easier than direct visualization or trans-illumination of the superficial and inferior epigastric vessels. The fact that visualization of internal inguinal ring was unaffected by BMI increases its value as a landmark in introducing the lateral ports.

Superficial epigastric vessels were more amenable to trans-illumination than inferior epigastric vessels and there was a strong inverse relationship between the BMI and the ability to trans-illuminate the superficial and inferior epigastric vessels. This same relationship was observed when the inferior epigastric vessels were visualized directly using the laparoscope. These findings have been reported in previous studies as well⁸.

The above findings indicate that traditional methods of avoiding lateral abdominal wall vessel injuries during placement of lateral trocars at laparoscopy like trans-illumination and direct visualization are helpful when the patient is of normal BMI. These methods become less reliable when the patient becomes overweight

Table 1 Success of Methods used for safe placement of the second port (n= 90)

	Right	Left
Visualization of round ligament entering internal inguinal ring	84	76
Transillumination of superficial epigastric vessels	38	42
Transillumination of inferior epigastric vessels	25	25
Direct visualization of inferior epigastric vessels	50	41
Direct visualization of inferior epigastric 5-8cm above pubic symphysis	33	34

Table 2 Comparison of Success of Methods used for safe placement of the second port (n= 90)

Method	Comparison	Percentage Visualized	p value
Visualization of at least one round ligament entering internal inguinal ring	Trans illumination of inferior epigastric vessel	94% vs 30%	<0.001
Transillumination of inferior epigastric vessels	Direct visualization of inferior epigastric vessel	30% vs 56%	<0.001
Direct visualization of inferior epigastric vessels	Trans-illumination of inferior epigastric vessel	55%vs 30%	<0.001

Table 3 Association of the body mass index with the success of the method used for safe placement of the second port (n= 90)

Method		Maximum likelihood ratio Chi Square	p Value
Visualization of at least one round ligament entering internal inguinal ring		0.212	0.64
	Superficial Epigastric Vessels	5.119	0.02
	Inferior Epigastric Vessels	15.273	0.001
	Inferior Epigastric Vessels at their Origin	5.231	0.02
	Inferior Epigastric Vessels at 5cm-8cm above pubic symphysis	4.166	0.04

or obese. This disadvantage was not seen when using internal inguinal ring as an anatomical landmark to locate the origin of the inferior epigastric vessels and placing the lateral trocars lateral to a sagittal line extending cephalad from the internal ring. The use of anatomical landmarks as a guide to placing lateral trocars has been described in other studies as well^{8,11}. However none of these studies have suggested the use of round ligament entering the internal ring as an anatomical landmark for this purpose. Using midline trocars are also helpful in avoiding lateral abdominal wall vessel injuries but not all gynaecologists are comfortable doing surgeries with midline secondary trocars.

Our study population consisted only of women in the District of Kandy, Sri Lanka and we were not able to assess the effect of difference in skin color on

trans - illumination of anterior abdominal wall vessels. This could be verified only by conducting studies in women with different skin colours.

In conclusion, identifying the internal inguinal ring is easier than identifying abdominal wall vessels. Placing the secondary trocars lateral to a sagittal line originating from the medial border of the internal inguinal ring and extending cephalad is a good method to safely insert the secondary trocars at laparoscopy. This method is not affected by the BMI of the woman.

ACKNOWLEDGEMENTS

Dr Sampath Tennakoon, Senior lecturer in Community Medicine, Faculty of Medicine, University of Peradeniya assisted with the statistical analysis

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Case Report: Intracranial Hypertension in Labour

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Idiopathic intracranial hypertension, Abducens nerve palsy, Induction of labour, Prostaglandins

Abstract

Neurological manifestations are seldom reported during labour with majority of cases being eclampsia. We report a case of 33-year-old lady in her second pregnancy developing features of raised intracranial pressure (bilateral Abducens nerve palsy and papilloedema) after administration of Prostaglandin E2 for induction of labour. Radiological imaging was unremarkable and the neurological symptoms spontaneously resolved within 24 hours of the delivery of the baby..

thrombophilia and hypofibrinolysis seen in high estrogen conditions such as pregnancy, obesity, and polycystic ovarian syndrome lead to thrombosis of arachnoid villi and reduced CSF absorption, increasing the intracranial pressure².

The most common symptoms are headache and visual disturbances including transient visual obscurations and diplopia. Treatment for IIH during pregnancy includes dietary control, carbonic anhydrase inhibitors

INTRODUCTION

Raised intracranial pressure (ICP) is rarely encountered in obstetric practice although it is common in neurology and neurosurgery. It can arise as a consequence of localized mass lesions (haematomas, neoplasms and abscesses), disturbance of CSF circulation (hydrocephalus), obstruction to major venous sinuses (cerebral venous thrombosis), diffuse brain oedema (encephalitis, meningitis) and idiopathic intracranial hypertension¹.

Idiopathic intracranial hypertension (IIH) is a syndrome of elevated intracranial pressure with normal CSF composition and no evidence of hydrocephalus or mass lesion. IIH is most often seen in obese women of reproductive age (19.3/100,000) and is reported only occasionally during pregnancy². It can occur in any trimester during pregnancy. It is postulated that

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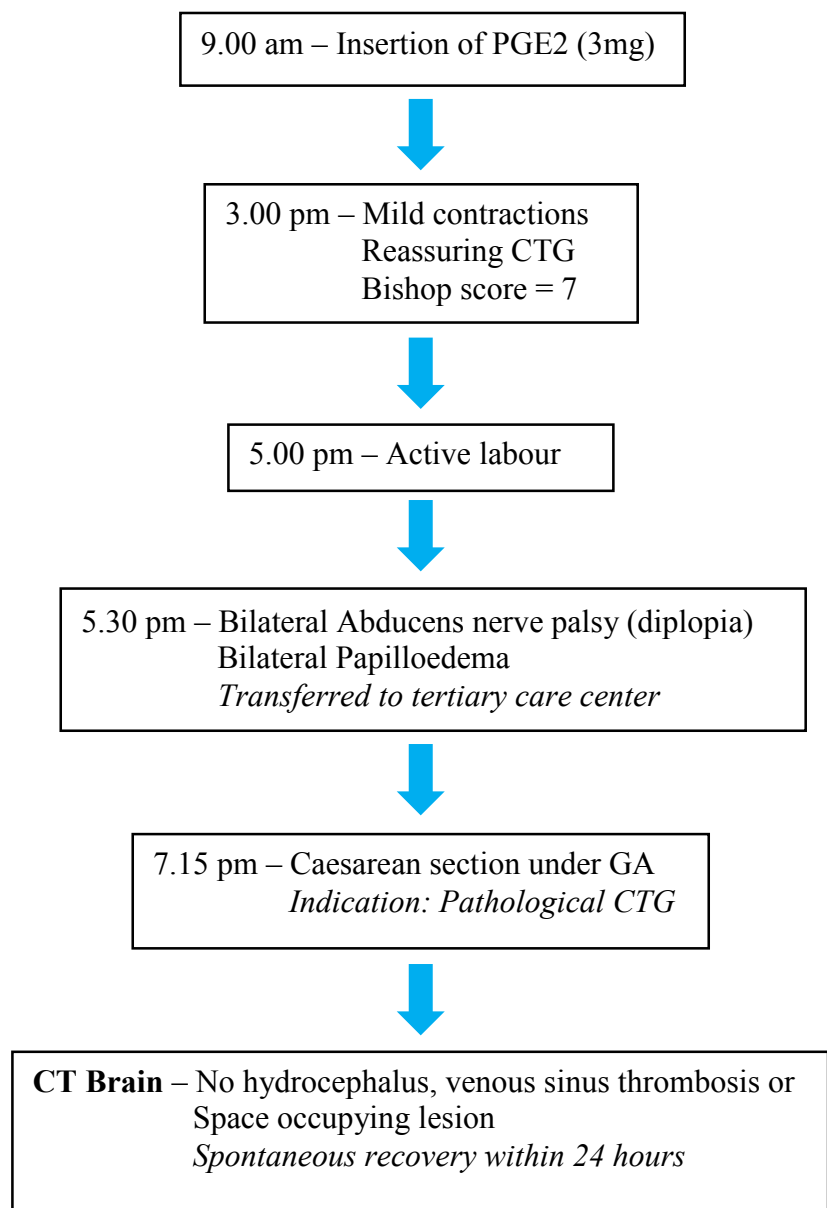
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Competing interest: The authors report no conflict of interest



Time line of the case

- acetazolamide (may be teratogenic), furosemide, corticosteroids and serial lumbar punctures^{2,3}.

CASE REPORT

A 33-year-old mother of one child was admitted to ward at 40 weeks of gestation for confinement. Her BMI was 24.5 at the booking visit. She received shared care and did not have features suggestive of raised intracranial pressure such as headache or visual disturbance. A decision was made to induce labour at 40 weeks +2 days as she complained of persistently reduced fetal movements despite a normal biophysical profile and normal cardiotocographs (CTGs). The Bishop score was 5 and vaginal Prostaglandin E2 (3mg) was administered for induction of labour around 9.00 am in the morning.

Around 4.00 pm, the uterine contractions became more intense and she was sent to the labour ward at 5.00 pm in active labour. She received Entonox for analgesia and fetus was monitored with continuous CTG. Around 5.30 pm, she complained of a headache and shortly afterwards she became acutely distressed with sudden onset blurred vision due to diplopia. Her blood pressure was 130/70 mmHg and vaginal examination revealed a 4 cm dilated cervix with intact membranes. The CTG showed uterine hypertonus with occasional variable decelerations. She was examined by the consultant physician and bilateral Abducens nerve (VI nerve) palsy and bilateral papilledema were detected. The clinical diagnosis of intracranial hypertension was made and she was transferred to the nearest tertiary care center for further management. On arrival at the tertiary care center, she was haemodynamically stable; but the CTG was pathological with uterine hyperstimulation. The delivery needed to be expedited and an emergency caesarean section was planned. Without radiological imaging to exclude a space-occupying lesion prior to surgery, the anaesthetic team decided provide general anaesthesia. She had an uncomplicated caesarean section around 7.15 p.m. and the baby was healthy at birth. After the delivery, she had a non-contrast CT scan which was unremarkable (no hydrocephalus, venous sinus thrombosis or space occupying lesion). She was admitted to the intensive care unit for observation and no special medication was needed as her neurological

manifestations spontaneously resolved within 24 hours of the delivery of the baby. All other blood and urine investigations were normal. She was discharged from medical care on day five.

DISCUSSION

In this case scenario, intracranial hypertension was clinically diagnosed by the presence of bilateral Abducens nerve palsy and papilledema. Considering the acute nature of the clinical manifestations, the possibility of a vascular lesion such as venous sinus thrombosis or intracranial haemorrhage needed to be excluded as a priority. But her radiological imaging did not reveal any intracranial pathology leaving a diagnostic dilemma.

Idiopathic intracranial hypertension (IIH) was considered a probable diagnosis in this patient. The diagnosis of IIH is by exclusion using modified Dandy criteria⁴.

Modified Dandy Criteria	
1.	Signs and/or symptoms of increased intracranial pressure.
2.	Documented elevated intracranial pressure (>250mm water).
3.	Normal CSF composition.
4.	No evidence of hydrocephalus, mass, structural, or vascular lesion on imaging.
5.	No localizing neurological signs except a unilateral/ bilateral VI nerve paresis.

Table1. Modified Dandy Criteria

Although this patient had signs of increased intracranial pressure like papilledema and bilateral VI nerve palsy, neither intracranial pressure nor CSF composition was measured as she had general anaesthesia for caesarean section instead of spinal anaesthesia. A lumbar puncture would have been a better option as it is one of the treatment modalities in IIH allowing CSF drainage and reduction in CSF pressure². However general anaesthesia had to be considered in the absence of prior radiological imaging.

With the assumption of asymptomatic IIH in this patient, an acute and a transient increase in the intracranial pressure can be expected if she had adopted Valsalva maneuver in response to uterine hypertonus secondary to PGE2 administration. But in

the absence of confirmatory evidence, IIH still remains a clinical diagnosis.

An idiosyncratic reaction to Prostaglandins was also considered a possibility. Although mild visual disturbances are reported with PGE2, there are no case reports of bilateral VI nerve palsy after PGE2 administration. Contrarily, PGE2 induces vascular relaxation and animal studies have shown that PGE2 decreases intracerebral pressure⁵.

All her neurological symptoms and signs improved with the gradual reduction of intracranial pressure after the delivery of the baby. The final diagnosis is still open for discussion.

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Accidental self-insertion of an intrauterine contraceptive device into the bladder

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Key Words: Self-Insertion, IUCD, Bladder

CASE REPORT

A 20-year old mother of one child had an intrauterine contraceptive device (IUCD) inserted by a primary care medical doctor. Few days later her husband complained of discomfort during coitus and the woman removed the IUCD by herself. Following coitus, due to the fear of another pregnancy she self inserted the IUCD. Few days later she developed dysuria and suprapubic pain. As the symptoms were persisting even after two days, she sought medical advice. She did not have haematuria or difficulty in passing urine. A transvaginal ultrasound scan could not locate the device in the uterine cavity, but showed the IUCD freely floating inside the bladder. X-ray pelvis showed the IUCD in the region of the bladder. (Fig 1)

A cystoscopy showed the floating IUCD in the bladder, with no features of perforation or migration. It was retrieved with ease by grasping forceps (Fig 2).

DISCUSSION

Foreign bodies in the bladder can be due to self-insertion, migration from adjacent organs (uterus) and by iatrogenic causes. Among commonly self inserted objects, cotton swabs, tampons, paper clips, grass leaves, and plastic beans are reported in literature and are mostly related to acts of sexual gratification.¹ Transmigration of IUCD from the uterine cavity to surrounding pelvic organs (bladder, pelvic colon) and retroperitoneal space has been



Fig 1 X ray showing the IUCD in the pelvis



Fig 2 Cystoscopy view of the IUCD inside the bladder

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Competing interests: The authors report no conflict of interest

reported and is well known.^{2,3} Accidental primary placement of IUCD is rarely reported.⁴ Accidental self-insertion of an IUCD into the bladder via the urethra is unreported. Although the short female urethra could provide easy access to the bladder for foreign body placement, the extended limbs of the IUCD makes it less likely to be accidentally inserted to the bladder via the urethra. Short duration of few days makes migration unlikely in this patient. Discomfort to the male partner during coitus due to long trailing threads of IUCD is well known and the remedy is to cut the threads shorter. Women should be advised to seek medical assistance for problems related to the IUCD and should

be discouraged to manipulate the IUCD by their own.

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A Historical Perspective

Diagnostic challenges in obstetrics and gynaecology in the pre sonology and cardiotocography era

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Sonology and cardiotocography (CTG) were introduced to obstetrics and gynaecology in Sri Lanka at the Teaching Hospital Peradeniya in 1980. However both were not available in the Teaching Hospital Mahamodara Galle (THMG) until 1986. Diagnostic challenges faced by the author in the pre sonology and CTG era are presented.

ESTABLISHING THE CAUSE OF A FETAL DEATH

A 27 year old woman who had not received any antenatal care for her 2nd pregnancy, was admitted to the academic obstetric unit of the THMG on the morning of 3rd December 1982 at 34 weeks gestation, with significant lower abdominal pain of sudden onset since the previous evening. She appeared to have a tender uterus extending almost up to the xyphisternum but with ill-defined lateral margins. A *death in utero* was diagnosed with a Pinnard fetal stethoscope. Identification of fetal parts was difficult but the fetal head was thought to be in the right upper quadrant of the abdomen. She had mild pallor but was haemodynamically stable. The cervix was not effaced, the os was closed and there was no bleeding. She had a two year old son who had been delivered normally but 10 days later she had probably undergone an evacuation of retained products following a secondary postpartum haemorrhage (no details were available). The tentative clinical diagnosis was placental abruption. A general surgeon excluded the need for any surgical intervention and agreed with conservative

management and awaiting the spontaneous onset of labour. The next morning as she had still not established labour, a plain X-ray of abdomen (antero-posterior view) was carried out which confirmed the *death in utero* with marked over riding of skull bones (Spalding's sign), and also showed that the fetus was lying transversely in the upper part of the abdomen with its head in the right hypochondrial region. As the initial diagnosis was held in doubt, an exploratory laparotomy was carried out. A macerated fetus of 34 weeks gestation was found in the peritoneal cavity, with its placenta attached to the fundus of the 'post partum' uterus which had a transverse fundal rupture of approximately eight centimeters in length. Approximately 300 ml of altered blood and meconium was found in the peritoneal cavity. The uterine rupture was repaired in two layers and she was transfused with 500 ml of blood. She had an uncomplicated recovery. The spontaneous rupture of the uterus was presumed to be as a result of a weak scar on the fundus of the uterus, probably consequent to a uterine perforation during a probable evacuation of retained products after the previous normal delivery two years earlier.

ESTABLISHING THE UNDERLYING CAUSE OF SUSPECTED HYPEREMESIS GRAVIDARUM IN A YOUNG GIRL

On 30th September 1983 a 14 year old girl who was *virgo intacta*, and denied any form of sexual contact, was admitted to the academic gynaecological unit of the THMG with episodes of vomiting following 17 weeks amenorrhoea, nine months after her menarche. She had an abdomino pelvic mass corresponding to a 16 weeks gravid uterus. Urinary human chorionic gonadotrophin (hCG) was > 700,000 iu/L. In the absence of liquor on aspiration of the mass, an attempted radiological amniogram, using a contrast

medium, showed a radio opaque mass with irregular outlines but no vesicles. A haemorrhagic, necrotic tumour about 25 cm in diameter, filling the Pouch of Douglas, the right fallopian tube overlying it and the uterus and the normal left adnexae on its left, was found on exploratory laparotomy. There was no ascites or secondary deposits. The tumour was almost completely dissected out. Histopathology confirmed a pure chorio carcinoma. Post operatively she was treated with intravenous Methotrexate and Cyclophosphamide in the academic gynaecology unit itself, and she is quite well up to date. Taking into account the clinical and histopathological features, the tumour was considered to be a primary non gestational choriocarcinoma arising from the right ovary.

Both the above cases have been reported earlier^{1,2} but are briefly presented again to enable a comparison with the current methods of diagnosis with the aid of sonography and CTG. Spalding's sign is now observed by sonology and not by radiology and the absence of the fetal heart sounds is confirmed by a hand held Doppler fetal heart detector, CTG or sonology, and not by a Pinnard fetal stethoscope. Only semi-quantitative assays of urinary hCG were available then, using stepwise increasing dilutions of urine and a 'Gravindex Test' which had a sensitivity of only 3500 iu/L. Amniograms are not carried out today to confirm trophoblastic tumors, because sonology is available.

LOCALISATION OF THE PLACENTA PRIOR TO AMNIOCENTESIS.

Prior to the sonology era, localizing the placenta prior to amniocentesis, to prevent inadvertent injury to the placenta during the insertion of the needle, was a challenge. The method adopted then was to: a) exclude a placenta praevia by the ability to palpate the fetal head directly

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Competing interests: Conflicts of interest none

through the vaginal fornices, b) identify the back of the fetus because the placenta would most likely be opposite to it, c) ensure that the bladder was empty, d) elevate the fetal head away from the pelvic inlet towards the umbilicus, in order to increase the volume of liquor in the 'forewaters' below the head, and e) to insert the amniocentesis needle in the midline immediately above the pubic symphysis³.

ESTABLISHING LUNG MATURITY OF A FETUS PRIOR TO ELECTIVE, EARLY PRETERM DELIVERY

Fetal lung maturity prior to elective preterm delivery could be predicted by using the foam stability test ('Shake or Bubble Test')⁴. This was a rapid and simpler method compared to the assessment of the Lecithin Sphingomyelin ratio, which required sophisticated and expensive biochemical techniques⁵. The amniotic fluid obtained by amniocentesis was serially diluted in normal saline,

an equal volume of 95% ethanol added, shaken for 15 seconds, allowed to stand for 15 minutes, and examined for the presence of bubbles at the level of the meniscus. Absence of bubbles indicated a negative test while the positive results were graded according to the dilution at which bubbles persisted. Although antenatal corticosteroid therapy for impending preterm birth was being practiced, because surfactant therapy was not available in the THMG then, the foam stability test was useful in high risk women who required elective, early preterm delivery (between 28 -31 weeks gestation) and were unwilling to be transferred to Colombo for delivery.

Paradigm changes have occurred in the diagnosis of diseases, including obstetric and gynaecological problems, over the last four decades. Technological advances have contributed a great deal to these changes. A brief glimpse of the past has been provided. This may perhaps amuse the younger obstetricians and make them chuckle, while bringing nostalgic memories to those who are more senior.

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