PERSONAL PRACTICE

The unreactive fetal heart rate

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The 'flat' fetal heart rate (FHR) trace both before and during labour is viewed ominously by many obstetricians and midwives. The current medicolegal climate relating especially to care in labour is such that much emphasis is placed on an unreactive FHR. There is a tendency to link an unreactive FHR with an adverse outcome. However, it is only in a minority of cases that this is true. Furthermore, there is a wide variation in both definition and interpretation. Like many aspects of pregnancy care, the unreactive FHR is an example of 'a little knowledge being a bad thing'.

Definition

There is no agreed definition of an unreactive FHR; there is more agreement about what constitutes a reactive FHR. Therefore the simplest definition of an unreactive FHR might be one that fails to meet the criteria for a reactive FHR. A reactive FHR is one which has at least two accelerations of at least 15 beats per minute (bpm) amplitude (some would use 10 bpm¹), each lasting for at least 15 seconds, during a set time period, on which there is no general agreement, but it varies from 10 to 120 minutes.^{2 3} From the results of fetal behavioural studies¹ we use a 15 bpm amplitude of acceleration on at least two occasions in association with fetal movements within 40 minutes as our definition of reactivity in the fetus of 30 weeks' gestation or more, and consider 10 bpm to be more physiological at under 30 weeks (see below).

The problem with defining an unreactive FHR as one with no accelerations is that it does not take into account baseline FHR variation. There are two types of variation: short and long term. Short term variation is the variation in successive beat-to-beat intervals of the FHR. However, this variation cannot be accurately measured using external FHR monitoring via Doppler ultrasound with autocorrelation. Even when true beat-to-beat variation is accurately recorded using a fetal scalp electrode in labour, this cannot be visually quantified by someone observing the FHR. This is not surprising since the true mean (SE) beat-to-beat variation is 2.1 (0.2) ms.⁴ In the clinical setting the only method of quantifying short term FHR variation is by using computerised FHR analysis. However, even when this is available it appears that beat-to-beat FHR variation is not as clinically useful as when beat-to-beat intervals are aver-

aged over 1/16th of a minute and the difference between consecutive 1/16th of a minute intervals are used.⁵

Therefore it is long term FHR variation which is most commonly referred to in clinical practice. Long term variation is a visual assessment of the irregular oscillations of the baseline FHR. The normal amplitude of variation of the FHR baseline is commonly accepted as being between 5 and 15 bpm,⁶ though some maintain that 25 bpm is a more realistic upper limit.⁷ The considerable inter-observer and intraobserver variation in interpretation and quantification of FHR variation has led to the introduction of computerised FHR analysis.⁸

Although theoretically computer analysis of FHR would appear to be the panacea for problems of FHR interpretation, it is only as good as the program criteria. Such criteria need to be validated over tens of thousands of pregnancies if they are to detect fetuses at high risk of intrauterine compromise. This point is well illustrated by a sinusoidal FHR with pathologically low FHR variability, being computed as having normal long term variation, which could only be diagnosed as abnormal by including a computation for short term variation.⁹ It is noteworthy that the computer criteria for long and short term variation (evolved by comparing the analysis of thousands of FHR recordings with the subsequent infant outcome) differ remarkably from those used clinically. Furthermore, although computer analysis appears to detect antepartum fetal hypoxia in small for dates fetuses, the same criteria were not found to be applicable in labour.¹⁰ Although promising, computerised analysis needs further evaluation.

In clinical practice we suggest that an unreactive FHR should be defined as an FHR which fails to demonstrate accelerations (at least two periods of 15 seconds' duration with 15 bpm or more for pregnancies of more than 30 weeks, and 10 bpm for pregnancies less than 30 weeks) in association with fetal movements during 40 minutes' continuous recording. If the FHR fails to fulfil these criteria after 40 minutes of continuous recording then it should be regarded as abnormal. This conclusion would be reinforced by the demonstration of FHR baseline variation persistently being less than 5 bpm during the 40 minutes. The presence of decelerations of the FHR (a fall of at least 15 bpm lasting for at least 10 seconds) would be further evidence of patho-

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logical FHR pattern. However, it must be remembered that decelerations of the FHR are a normal feature of the very immature FHR¹ and are therefore less likely to be pathological before 28 weeks.

It is our view that this definition of the unreactive FHR with the caveats relating to baseline variation and decelerations is equally applicable to antepartum and intrapartum recordings of the FHR.

Causes

The normal FHR recording is determined by the dynamic interplay of the autonomic nervous system, cerebral centres, and cardiovascular reflexes.¹¹ The autonomic innervation of the heart is the most important mechanism in controlling FHR variation. The parasympathetic muscarinic cardiodecelerator activity (producing a decrease in FHR but increased variation) is of primary importance in influencing beat-to-beat variation because of its rapid effect on FHR. In contrast sympathetic cardiac stimulation (producing an increase in FHR and decreased variation) leads to a much slower FHR response.¹²

The causes of unreactive FHR can be broadly divided into physiological and pathological (table 1).

PHYSIOLOGICAL

Gestation is the most important physiological influence on FHR. The FHR fails to manifest accelerations in association with movements and is thus normally unreactive before 24 weeks.¹ From 24 weeks onwards, FHR accelerations are seen for a greater proportion of the time although these are not normally of 15 bpm or more until 30 weeks onwards.¹ Over the last trimester fetal rest/activity cycles become manifest and these evolve into sophisticated fetal behavioural states from 35 weeks.¹³ During the last trimester the commonest reason for an unreactive FHR is fetal

quiescence or fetal behavioural state 1F.¹³ At term a fetus can spend 30–40% of the time in quiescence and the average (SD) continuous period spent in quiescence is 21 (8·6) minutes.¹⁴ Therefore we would recommend that an unreactive FHR should be investigated if quiescence lasts longer than 40 minutes.

Another normal feature of advancing gestation is the effect on baseline variation. Early in the second trimester variation is very slight (less than 5 bpm) but gradually increases as pregnancy progresses so that it is usually greater than 5 bpm after 26 weeks.¹ The mean baseline variation is approximately 7 bpm at 34 weeks. Thereafter the mean baseline variation depends on the state of activity of the fetus: in quiescence, the mean variation falls to reach 4 bpm at 40 weeks, whereas in activity variation continues to rise to about 10 bpm at 40 weeks.¹ Other fetal factors which are associated with an increase in FHR variation include fetal breathing and fetal mouthing movements.^{1 15}

Maternal physiological factors which influence FHR reactivity include diurnal variation (more fetal activity and hence reactivity in the evening¹⁶) and glucose ingestion (increases fetal breathing and baseline variation).

External stimulation (for example, with vibratory or acoustic stimulators) of the fetus increases reactivity of the FHR and has been claimed to affect fetal behaviour generally.¹⁷

PATHOLOGICAL

A number of different pathologies can produce an unreactive FHR by directly affecting the fetal central nervous (CNS) or cardiovascular systems (CVS). Examples of pathologies directly affecting the CNS are structural abnormalities such as anencephaly, infections such as varicella, acute hypoxia (secondary to uterine contractions in labour, or placental abruption) and a major non-fatal hypoxic or anoxic insult¹⁸ causing brain death such as placental abruption or maternal cardiorespiratory collapse.¹⁹ The pathophysiological effect of chronic placental insufficiency as seen with severe pre-eclampsia or recurrent placental bleeding is to cause hypoxia which affects FHR variation.²⁰ In such cases when decompensation occurs there will be a resultant progressive reduction in FHR variation which will nearly always be accompanied by recurrent late or variable deceleration.^{21 22} This effect has been shown by cordocentesis to be associated with fetal hypoxia.²³ If the decompensation continues, hypoxic acidosis will result which is characterised by loss of accelerations, decreased baseline FHR variation (<3 bpm) and shallow decelerations in response to Braxton Hicks' contractions.^{24 25}

The commonest CVS cause for an unreactive FHR to cardiac arrhythmia. This can be primary idiopathic (supraventricular tachycardia, atrial flutter), or secondary to a structural cardiac abnormality. Cardiac failure in the fetus (secondary to anaemia, abnormal karyotype, cardiac anomaly or arrhythmia) also results in an unreactive FHR but this is probably due to the resulting hypoxia.

Table 1 Causes of an unreactive FHR

	Physiological	Pathological
Fetal	Immaturity Rest/activity states, 1F behavioural state,* or quiescence	Central nervous system: Anomalies (for example anencephaly) Infection (for example varicella) Hypoxia: Chronic (for example placental insufficiency, pre-eclampsia) Acute (for example intrapartum asphyxia, placental abruption or maternal cardiorespiratory collapse resulting in brain death) Cardiovascular system: Arrhythmias Anomalies Cardiac failure (various causes)
Maternal	Diurnal variation External stimuli	Accidents (for example cardiorespiratory arrest) Drugs: Opiates Anaesthetics (for example epidural) Parasympatholytic Sympathomimetic Magnesium sulphate

*Four categories of behavioural states are used to describe the fetus and these are numbered 1 to 4 and the letter 'F' is added to differentiate fetal states from neonatal states. Behavioural states are determined by heart rate, fetal gross body movements, and eye movements. For example, state 1F, which is synonymous with fetal quiescence, is characterised by absent eye movements, a stable fetal heart rate with low baseline variation, which can be interrupted by isolated heart rate accelerations which are strictly associated with brief gross body movements, mostly startles.

Maternal ‘pathological’ causes of an unreactive FHR are primarily confined to the effects of drugs taken by the mother, which cross the placenta and affect the fetal CNS and CVS. Those that predominantly have their effect on the CNS²⁶ include opioids such as pethidine, hypnotics such as diazepam, and anaesthetics both general and regional (epidural, spinal). However, the effect of an epidural or spinal anaesthetic in producing an unreactive FHR may be more commonly due to an effect on uterine perfusion. Drugs with predominant effects on the fetal CVS include parasympatholytic agents, such as atropine and tricyclic antidepressants, sympathomimetic agents such as ritodrine and salbutamol. Other drugs include magnesium sulphate and even paracetamol.²⁷

A rise in maternal temperature will result in a rise in the FHR baseline with a reduction in variation, but accelerations tend to be preserved.

It should be stressed that not all these pathological causes of an unreactive FHR are confined to labour. Indeed the majority pre-date labour onset. This observation leads to the speculation that at least some of the unreactive FHR records which are identified in labour may reflect some longstanding problem which has not been identified because no FHR recording was undertaken before labour (for example undiagnosed intrauterine growth retardation).

Management of an unreactive FHR

ANTEPARTUM

The first principle of management when an unreactive FHR is reported or suspected is to ensure that the strict criteria for definition are fulfilled (see above) since the commonest cause will be fetal quiescence. The next principle of management is to try and ascertain the cause (table 2). A clinical assessment should be undertaken to exclude conditions such as pre-eclampsia, antepartum haemorrhage, drug taking, and a small for dates fetus. After this clinical evaluation, ultrasound fetal

assessment is mandatory. A biophysical profile should be performed²⁸ which if normal is reassuring (which can be especially helpful in interpreting unreactive FHR below 28 weeks) but if it is abnormal it is associated with an increased risk of fetal compromise. In addition, during the profile procedure more critical observation of the relationship between the FHR and fetal activity may prove useful in diagnosing fetal pathology. For example, in brain death there is an unreactive FHR and absent fetal movement, whereas dissociation of fetal activity with an unreactive FHR is found with anencephaly and congenital abnormalities.²⁹

A detailed anomaly scan will help identify fetuses with obvious CNS, CVS, and karyotypic abnormalities. Cardiac arrhythmias should be categorised using M mode ultrasound. Fetal size assessment is important in diagnosing intrauterine growth retardation which is a common feature of many of the pathologies associated with an unreactive FHR. Similarly, abnormal Doppler ultrasound studies of the umbilical artery (increased systolic to diastolic ratio or absent end diastolic frequencies) are found in many conditions which can result in an unreactive FHR. Indeed the combination of intrauterine growth retardation, abnormal Doppler recordings, and an unreactive FHR is associated with a poor prognosis for the fetus.³⁰

Fetal stimulation has been used as an adjunct to FHR monitoring in order to assess fetal wellbeing. The oxytocin stress test is commonly used in North America if the FHR is unreactive. However, it is not without risks such as aggravating fetal compromise and preterm labour. An alternative stimulation test is vibroacoustic stimulation which has been found useful antenatally in shortening the time to achieve a reactive FHR.³¹ However, to date whereas a positive response is reported as reassuring (associated with a non-acidotic fetus), it is less useful in identifying fetal distress.³² Furthermore, there have been suggestions that vibroacoustic stimulation may be potentially hazardous to an already compromised fetus.³³

If the cause is still not certain then invasive testing may be necessary. In the antepartum period the diagnosis of chromosomal abnormality is the commonest indication for invasive testing, especially if ultrasound stigmata of anomalies are identified. While fetal blood sampling will provide a relatively quick answer, within two to three days, allow haemoglobin and blood gas and acid-base estimation (in case of fetal hydrops), and serological screening for viral infections, it is not without significant risk to the fetus especially in the growth retarded fetus.³⁴ Chorionic villus sampling may prove to be safer and quicker and the results as reliable. The relative merits of the two approaches have not yet been tested in controlled studies. Antepartum definitive treatment will depend upon the cause. If investigations conclude that the fetus does not have a lethal irreversible cause for the unreactive FHR or one which can be

Table 2 Management of an unreactive FHR

Ensure that the criteria for diagnosis are fulfilled to exclude physiological causes	
Unreactive FHR—need to exclude a pathological cause	
History and examination	Pre-eclampsia Antepartum haemorrhage Drugs Reduced/absent fetal activity
Ultrasound assessment	Small for dates fetus Biometry (fetal size) Anatomy CNS/cardiac Cardiac rhythm (M mode) Biophysical assessment Coincidence in fetal behaviour Fetal Doppler studies Stress/stimulation tests
Invasive testing	
Antepartum	Karyotype Viral serology Haemoglobin Blood gases Fetal scalp sampling
Intrapartum	
Treatment	
Antepartum	Fetal specific cause (for example antiarrhythmic drug or blood transfusion) Delivery

safely treated in utero (for example anti-arrhythmic therapy³⁵ or fetal blood transfusion) then delivery is the usual treatment, often by caesarean section.

INTRAPARTUM

In labour while clinical assessment in order to determine the cause is still possible and should be undertaken, the full range of investigations may be neither available nor feasible. In practice the commonest investigation employed is fetal blood pH estimation obtained by fetal scalp sampling (which has an added advantage of acting as a stimulation test). However, there are limitations to this approach. Unless detailed fetal assessment has been undertaken before the onset of labour (for example as part of the admission assessment), management in labour is going to be largely on the basis of fetal acidemia so that fetal normality before labour has to be assumed. This assumption may be proved to be incorrect once the infant is delivered. Furthermore, a normal scalp pH in the presence of an unreactive FHR does not exclude pathology.³⁶

In labour, general fetal resuscitatory measures should be employed even when the scalp pH is low since the cause of the unreactive FHR and decelerations may be secondary to reversible causes such as cord compression or hypertonic uterine contractions or uterine hypoperfusion after epidural anaesthesia. The advantages are that in utero resuscitation is more efficient than neonatal resuscitation and an operative delivery may be avoided.³⁷ These measures may include stopping an oxytocin infusion and/or administering tocolytics, fluid infusion, changing the mother's position to avoid caval compression, and giving facial oxygen. If these measures are unsuccessful, vaginal delivery is not imminent and the scalp pH is <7.2, delivery by caesarean section should be performed.

Conclusion

When faced with an unreactive FHR it is important that the criteria for diagnosis are fulfilled. The majority of so called 'unreactive FHR traces' are only such because FHR monitoring has not been continued for long enough. In the antepartum period an unreactive FHR should be investigated as quickly as possible. In labour the options are more limited and testing for fetal acidemia is commonly the only investigation possible. However, it must be remembered that an intrapartum unreactive FHR may be caused by antepartum pathology.

Prompt delivery is the usual therapeutic option if the cause of the unreactive FHR is thought to be pathological, the effects on the fetus reversible and non-lethal.

At present there is no objective assessment of FHR other than by computer analysis which if used serially may enable management of an unreactive FHR in the antepartum period to be improved.

For the future, research should first concentrate on improving the understanding of the pathophysiological processes which ultimately lead to an unreactive FHR both antepartum and intrapartum. Secondly, efforts should be directed towards extending the range of investigations which are available, especially in labour, to obtain a more comprehensive picture of the fetal condition.

- 1 Pillai M, James DK. The development of fetal heart rate patterns during normal pregnancy. *Obstet Gynecol* 1990;76:812-5.
- 2 Paul RH. The evaluation of antepartum fetal well-being using the nonstress test. In: Petrie RH, ed. *Fetal monitoring. Clinics in perinatology*. London: Saunders, 1982;9:254.
- 3 Brown R, Patrick J. The nonstress test: how long is enough? *Am J Obstet Gynecol* 1981;141:646.
- 4 Lawson GW, Dawes GS, Redman CWG. A comparison of two fetal heart rate ultrasound detector systems. *Am J Obstet Gynecol* 1982;143:840-2.
- 5 Lawson GW, Dawes GS, Redman CWG. Analysis of fetal heart rate on-line at 32 weeks gestation. *Br J Obstet Gynaecol* 1984;91:542-50.
- 6 Steer PJ, Eigbe MB, Lissauer JJ, Beard RJ. Interrelationships among abnormal cardiotocograms in labour, meconium staining of the amniotic fluid, arterial cord blood pH and Apgar scores. *Obstet Gynecol* 1989;74:715-21.
- 7 FIGO News-Guidelines for the use of fetal monitoring. *Int J Gynaecol Obstet* 1987;25:159-67.
- 8 Dawes GS, Visser GHA, Goodman JDS, Redman CWG. Numerical analysis of the human fetal heart rate: the quality of ultrasound records. *Am J Obstet Gynecol* 1981;141:43-52.
- 9 Street P, Dawes GS, Moulden M, Redman CWG. Short-term variation in abnormal antenatal fetal heart rate records. *Am J Obstet Gynecol* 1991;165:515-22.
- 10 Dawes GS, Rosevear SK, Pello LC, Moulden M, Redman CWG. Computerised analysis of episodic changes in fetal heart rate in early labour. *Am J Gynecol* 1991;165:618-24.
- 11 Renou P, Newman W, Wood C. Autonomic control of fetal heart rate. *Am J Obstet Gynecol* 1969;105:949.
- 12 Warner HR, Cox A. A mathematical model of heart rate control by sympathetic and vagus efferent information. *J Appl Physiol* 1982;17:349.
- 13 Nijhuis JG, Prechtl HFR, Martin CB Jr, Bots RSGM. Are there behavioural states in the human fetus? *Early Hum Dev* 1982;6:177.
- 14 Pillai M, James DK. Behavioural states in normal mature human fetuses. *Arch Dis Child* 1990;65:39-43.
- 15 Wheeler T, Gennser G, Lindvall R, Murrills AJ. Changes in the fetal heart rate associated with fetal breathing and fetal movement. *Br J Obstet Gynecol* 1980;87:1068-79.
- 16 Patrick J, Campbell K, Carmichael L, Natale R, Richardson B. Patterns of gross fetal body movements over 24-hour observation intervals in the last 10 weeks of pregnancy. *Am J Obstet Gynecol* 1982;142:363-71.
- 17 Gagnon R, Hunse C, Carmichael L, et al. Effects of vibratory acoustic stimulation on human fetal breathing and gross fetal body movements near term. *Am J Obstet Gynecol* 1986;155:1227.
- 18 Nijhuis JG, Kruyt N, van Wijck JAM. Fetal brain death: two case reports. *Br J Obstet Gynaecol* 1988;195:198.
- 19 van der Moer PE, Gerretsen G, Visser GHA. Fixed fetal heart rate pattern after intrauterine accidental decerebration. *Obstet Gynecol* 1985;65:215-7.
- 20 Henson GL, Dawes GS, Redman CWG. Antenatal fetal heart rate variation relation to fetal acid-base status at caesarean section. *Br J Obstet Gynecol* 1983;90:516.
- 21 Devoe LD, McKenzie J, Searle NS, et al. Clinical sequelae of the extended nonstress test. *Am J Obstet Gynecol* 1985;151:1074.
- 22 Visser GHA, Bekedam DJ, Ribbert LSM. Changes in antepartum heart rate patterns with progressive deterioration of the fetal condition. *Int J Biomed Comput* 1990;25:239-46.
- 23 Visser GHA, Sadovsky G, Nicolaides KH. Antepartum heart rate patterns in small for gestational age third trimester fetuses: correlations with blood gases obtained at cordocentesis. *Am J Obstet Gynecol* 1990;162:698-703.
- 24 Visser GHA, Redman CWG, Huisjes HJ, Turnbull AC. Nonstressed antepartum heart rate monitoring: implications of decelerations after spontaneous contractions. *Am J Obstet Gynecol* 1980;138:429.
- 25 Krebs HB, Petres RE, Dunn LJ. Intrapartum fetal heart rate monitoring. *Obstet Gynecol* 1983;145:297-305.
- 26 Petrie RH, Yeh SY, Murata Y, et al. The effects of drugs on fetal heart rate variation. *Am J Obstet Gynecol* 1978;130:294-99.
- 27 Rosevear SK, Hope PL. Favourable neonatal outcome following maternal paracetamol overdose and severe fetal distress. Case report. *Br J Obstet Gynaecol* 1989;96:491-3.
- 28 Manning FA, Platt LD, Sipos L. Antepartum fetal evaluation: development of a fetal biophysical score. *Am J Obstet Gynecol* 1980;136:787-95.

- 29 Pillai M, Garrett C, James D. Bizarre fetal behaviour associated with lethal congenital anomalies: a case report. *Eur J Obstet Gynecol Reprod Biol* 1991;**39**:215-8.
- 30 Maulik D, Yarlagadda P, Downing G. Doppler velocimetry in obstetrics. In: Morrison JC, ed. *Antepartal fetal surveillance*. *Obstet Gynecol Clin North Am*. London: Saunders, 1990;**17**:181.
- 31 Smith CV, Phelan JP, Broussard P, *et al*. Fetal acoustic stimulation testing III. Predictive value of a reactive test. *J Reprod Med* 1988;**33**:217.
- 32 Shaw KJ, Paul RH. Fetal responses to external stimuli. In: Morrison JC, ed. *Antepartal fetal surveillance*. *Obstet Gynecol Clin North Am*. London: Saunders, 1990;**17**:235-46.
- 33 Visser GHA, Mulder HH, Wit HP, Mulder EJH, Prechtl HFR. Vibro-acoustic stimulation of the human fetus: effect of behavioural state organization. *Early Hum Dev* 1989;**19**:285-96.
- 34 Maxwell DJ, Johnson P, Hurley P, Neales K, Allan L, Knott P. Fetal blood sampling and pregnancy loss in relation to indication. *Br J Obstet Gynecol* 1991;**98**:892-7.
- 35 Sykes GS, Molloy PM, Johnson P, Stirrat GM, Turnbull AC. Fetal distress and the condition of the newborn infants. *BMJ* 1983;**287**:943-5.
- 36 Smolencic JS, James DK. Intermittent fetal tachycardia and hydrops fetalis. *Arch Dis Child* 1991;**66**:1160-1.
- 37 Caldeyro-Barcia R. Intrauterine fetal reanimation in acute intrapartum distress. *J Perinat Med* 1991;**19**: (2):11.