



High-Level Human and Animal Experimental Evidence Validates the Importance of Classifying Fetal Heart Rate Decelerations into Early (Non-Hypoxemic) and Late (Hypoxemic) Types

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Abstract

The pathophysiology of fetal cardiovascular and metabolic response to hypoxemia induced by uterine contractions is complex and important. However, in practice, the clinicians have to depend on cardiotocography fetal heart rate patterns in the context of clinical setting. Acute fetal hypoxemia characteristically triggers chemoreflex-mediated decelerations, centre-stage in cardiotocography interpretation. A novel but flawed hypothesis that “all decelerations are due to fetal hypoxemia and timing of decelerations is a red herring” has a strange attraction. Notwithstanding, this review article for the first time presents detailed direct empirical evidence from human and animal studies that the hypoxemic decelerations are mostly late in timing and moreover not the majority. Well conducted/ judiciously interpreted laboratory experiments can provide best quality evidence because a direct ‘cause and effect’ (unlike clinical studies) can be surmised with the intervention causing prompt changes. Additionally, the dose-response relationship can be tested, while reliably controlling confounding factors. At the same time, the clinicians should guard against ‘pathological science’ and repeated misinterpretation of animal experiments and their misapplication to human labor and fetus. This article presents robust empirical evidence that the majority decelerations, which are of early timing, cannot be explained by hypoxemic chemoreflex. The chemoreflex provides time-bound and constrained protection to the human fetus. Currently, there aren’t safe/reliable biomarkers of fetal decompensation. Therefore, persistent hypoxemic (late) decelerations should be differentiated, ameliorated, additional tests undertaken or delivery expedited. Plentiful empirical evidence (high scientific proof) from animal cord occlusion studies consistently shows that the recovery of the chemoreflex decelerations commences only after release of the cord-occlusion (i.e., when hypoxemia starts recovering). The drop in PaO₂ is most rapid around the height of uterine contractions and starts recovering only during the late relaxation phase where the recovery of hypoxemic decelerations

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would commence. For categorizing decelerations, the etiological mechanisms, being presumptive, are much less important secondary considerations. The size/shape/area of decelerations disregarding the timing seem poor predictors of acidemia. The primarily timing-based early/late/variable categorization seems most scientific and is essentially pathophysiological i.e., hypoxemic (mostly late) versus non-hypoxemic (early) types. Fortunately, around 2020, the eFM (the official on-line CTG training programme of the Royal college of Obstetricians and Gynaecologists and the Department of Health, UK) has accepted the scientific definitions of decelerations based on 'timing' alone without the previous flawed reference to 'uniform' or 'rapid/gradual' shapes, making 'early' the commonest; and others should implement similar scientific reform.

Introduction

Serious intrapartum fetal hypoxemia in many cases is eminently preventable even though the birth-attendants have to rely on just the Fetal Heart Rate (FHR) and contraction patterns using Cardiotocography (CTG). Birth-attendants develop some intuition (art) and consequently obvious abnormalities get picked up with most CTG interpretation systems. However, the small number of difficult-to-detect cases would benefit from the most scientific pattern-recognition. These are the small numbers of adverse neonatal outcomes where even the reputed maternity units in UK, like the Nottingham University and East Kent University Hospitals, have come under major criticism in public forums that the CTGs have been misinterpreted and lessons have not been learnt.

The pioneers Edward Hon [1] and Caldeyro-Barcia [2] found timing-based early/late/variable (type I, II, III) categorization of decelerations to be a 'low hanging fruit' with easily observable correlation to fetal outcome. This categorization remains mainstay of all national guidelines to date [3-5]. However, the current concept of the vast majority of decelerations being due to cord-compression (variable) has been questioned as a mistaken pattern-recognition causing confusion and frustration [6]. This was a gross misinterpretation from the animal cord-occlusion experiments [6]. Similarly, there have been calls for discarding the 'early/late' distinction in favour of several other features like size/frequency/cumulative deceleration area or 'rapid vs gradual shape' categorization [7-13]. This has been argued to be unphysiological and unsafe [10]. Recently, it has been prominently argued that hypoxemia per se does not matter as all FHR decelerations are due to hypoxemia (chemoreflex) and one should wait until fetal tachycardia combined with the loss of baseline variability before intervening [8,9].

Establishing truth is the foundation of science. Well conducted laboratory experiments when interpreted and applied correctly are accepted to provide best quality evidence because a direct 'cause and effect' (unlike clinical studies) can be surmised with the intervention causing prompt changes with dose-response relationship affirmation feasible, while reliably controlling other confounding factors. Scientific inference is a matter of deciding between competing hypotheses [14]. Previous literature includes hypotheses, observations and limited clinical evidence for early/late decelerations, but analysis of substantial empirical evidence has not been systematically presented so far. This review collates and interprets this empirical evidence in the context of practical experience and knowledge. It is the obstetricians, not the physiologists, who have the prac-

tical knowledge and expertise in intrapartum fetal monitoring and they need to critically appraise this empirical evidence and counter any misinterpretation / misapplication.

Are the types of decelerations relevant?

Information is contained and processed in patterns. Cardiotocography interpretation is essentially pattern-recognition, hopefully truthful as much as possible. FHR decelerations are centre stage [6]. For good discriminatory power of CTG, it would be highly important to differentiate the pathophysiological patterns of hypoxemic from non-hypoxemic FHR decelerations (not per se causative mechanisms which remain mostly presumptive). Head-compression serves as one of the leading mechanisms (prototype) for the non-hypoxemic-reflex decelerations; hence, it is worth looking at the direct experimental evidence for these benign decelerations as well as their counterpart - the late hypoxemic decelerations. Many decelerations could be multi-factorial but a model of one dominant mechanism seems clinically useful and therefore valid [6].

Does head-compression cause benign (non-hypoxemic) decelerations?

Human fetal skull is mouldable with open fontanelles, which is different from animal skulls; hence, reliable animal experiments of fetal head-compression are very difficult. However, there is sizeable direct empirical evidence for the head-compression decelerations in human labor itself (as opposed to surrogate animal experiments). This evidence (often misunderstood or deliberately ignored) has been described in detail [15] and will be briefly summarised here. Chung and Hon showed that 18/19 applications of pressure with ring pessaries vaginally during labor to the human fetal head caused FHR decelerations [16]. The group of Caldeyro-Barcia subjected three fetuses to labor-like transabdominal and vaginal head-compressions for 30 to 45 seconds that elicited FHR decelerations similar to the 'type I or early dips' [2]. In another study of 25 laboring women, Walker et al applied manual pressure at five sites on the uterus from fundus down to suprapubic area (fetal head) [17]. With pressure on the head, 76% of foetuses showed FHR deceleration while only 24% showed tachycardia. In the words of Walker et al, the bradycardias were most frequent with pressure over the fetal head and decrease as the pressure site moves to the breech over the fetal body; whereas the tachycardias had exactly the reverse order [17]. In addition, there is so much observational evidence in human labor supporting head-compression decelerations that more (recent) experiments in humans could be considered unnecessary even if not difficult to conduct [15].

In contrast, a fairly controversial and unscientific claim seems that head-compressions do not cause benign decelerations at all but only when severe enough to cause fetal cerebral ischemia/hypoxia, somehow published in reputed journals [18]. This seems to be proposed without any specific proof but by simply producing doubt about experiments in human labor that numbers included were small or decelerations were short-lived etc. [18]. Most of these objections had already been answered previously [15]. Moreover, mere doubt is not a proof of negation. In 1950s, the tobacco company executives used 'manufacturing doubt and muddying the waters' as a deliberate strategy against the increasingly adverse medical evidence. Whether perfect or not, all head-compression human experiments consistently triggered majority decelerations, also consistent with the broader evidence and common observations by birth-attendants, a major strength [15]. Sometimes major/

entire emphasis is placed on the 'Cushing reflex' from raised Intracranial Pressure (ICP) [18]. However, bradycardia is Cushing's delayed second component, hence relevant mainly in persistent/chronic ICP-rise [19] with possibly smaller/uncertain role during labor contractions. If a persistently mildly raised fetal ICP during labor reaches a threshold, the additional ICP-increments during contractions may trigger Cushing reflex. More importantly, the dura-mater and falx, with rich vagal-innervation, are very stretch-sensitive [20] triggering 'vagal bradycardia' (visceral non-baro/non-chemo reflex). Thus, the distortion of dura/falx during contractions would cause benign rapid decelerations with 'early' timing - (**Figure 1**). This distortion of dura/falx cannot be reliably tested in animal experiments. The world's most widely read textbook-'Williams Obstetrics' (<https://doctorlib.info/gynecology/williams-obstetrics/24.html>) quotes, "head-compression is the likely cause of many mistaken variable decelerations attributed to cord-compression and the distortion of dura seems the causative mechanism."

Figure 1 shows a teaching example of 'early decelerations' in the three editions of an acknowledged British textbook [21]. These short-lasting FHR decelerations with nadir roughly corresponding to the peak of contractions were reported to be the commonest in labor by the British obstetricians and midwives [6]. In contrast, during any hypoxemic decelerations, the PaO₂ will continue to drop well beyond the peak of contraction until the late relaxation phase, where the FHR recovery would commence based on robust empirical evidence (**Figure 2-7**).

Head-compression decelerations - cerebral hypoperfusion and anoxia?

It has been much argued that the head-compression decelerations, when occur, are because of cerebral hypoperfusion/anoxia; and hence pathological [13]. Most grassroots obstetricians and midwives managing labor disagree. The suggestion of cerebral hypoperfusion / anoxia comes from a couple of animal studies. For example, in one animal study the heads of fetal lambs were exteriorised from pregnant uteri, carotid vessels catheterised and fetal lamb heads were compressed with both hands [22]. Are such animal studies comparable to what happens in human labor? Importantly, a systematic review of the effect of head-compression on intracranial pressure, oxygenation, blood flow and cerebral function in humans showed it not causative of fetal brain hypoxia or injury [23].

Importance of fetal peripheral chemoreflex in labor

The principles and importance of fetal chemoreflex in response to hypoxemia are well known to obstetricians and midwives since 1960s. These have been reemphasised in recent reviews but with misinterpretation of some very crucial aspects [9,13,24]. Furthermore, in practice, the birth-attendants are constrained by the inability to perform highly invasive monitoring or tests on the fetus. Even if advanced technologies (like functional MRI) become available, applying them to large number of patients could be impractical. Thus, sequential measurement of fetal tissue oxygenation, biochemistry or haemodynamic parameters like blood pressure during labor do not seem feasible in foreseeable future (apart from limited fetal scalp blood sampling - FSBS). Hence, the clinicians cannot safely wait for the fetal decompensation and then intervene. A safe approach seems to make an assessment as to which decelerations are hypoxemic and are likely to lead to an unsafe level of acidemia; and then either ameliorate these decelerations with resuscitative measures or proceed to some additional test of

fetal well-being (In UK, FSBS is practiced) or expedite delivery.

Are all decelerations in labor due to hypoxemia?

Recently, it has been unscientifically asserted that all FHR decelerations in are due to the chemoreflex triggered by hypoxemia induced by uterine contractions [9,13]. Furthermore, it is purported that the time relationship of decelerations to the uterine contractions is a red herring; and hence the decelerations should not be classified into early/late/variable types [9,13]. This needs wide debate and critical analysis because all international CTG guidelines do currently advocate early/late/variable categorization. Science is almost always a choice between two or more competing hypotheses.

Pitfalls in science generally occur because of human errors, which involve flawed abductive reasoning or mistaken consilience or both [14]. These are further exacerbated by confirmation bias (motivated reasoning), vested interests and belief persistence. Studies during labor using fetal near-infrared spectroscopy and pulse oximetry showed a wide scatter of PaO₂ levels [25,26]. These were not clinically useful (personal communications with authors [25]), but probably consistent with most uterine contractions causing very mild transient fetal hypoxemia with quick recovery in early relaxation phase [25,26]. Moreover, fetuses don't need to mount an adaptation response (chemoreflex) to these minor or insignificant degrees of hypoxemia because the vast majority of contractions in most women are not associated with any FHR decelerations. It would be tempting to assume that whenever decelerations do occur, they all (or majority) must be due to worsening of the subtle/minor degrees of fetal hypoxemia during most contractions [9,13]. However, such a jump / argument is not an empirical proof at all. On the other hand, it a fundamental 'formal' fallacy of logic called 'affirming the consequent'. In simple terms, if 'X' leads to 'Y'; then it does not necessarily follow that all 'Ys' arise from 'X'. There could be several (even more common) causes of 'Y'. Logic is a prerequisite to science. Apart from the above flawed abduction and the major logical error there seems no other proof provided for the proposition that all decelerations are due to hypoxemia [9,13].

Robust empirical evidence that hypoxemic chemoreflex causes "late" (not early) decelerations

It is of course difficult to conduct invasive experiments or detailed / continuous measurements of the fetal cardiovascular and metabolic changes in human labor. Thus, the animal fetus experiments by physiologists were the obvious and useful solution to this problem. Even in the animal experiments, it is far more difficult to reliably control the degree and patterns of hypoxemia by artificially inducing labor-like uterine contractions. Hence, the majority of animal studies involved experimental occlusion of umbilical cord to mimic the hypoxemia caused by uterine contractions. Cord-occlusion had the added advantage that the degree, duration, frequency, total time of insult could all be controlled and varied to examine the effect of endlessly different degrees of hypoxemia on fetal physiology. Since cord-compression in animal experiments caused "rapid" FHR decelerations, unfortunately all rapid decelerations were deemed to be caused by it and hence "defined" "variable" (Again a logical fallacy described above as 'affirming the consequent') [6]. The cord-compression experiments indeed generated so much excitement that for the last couple of decades a misconception (preconditioning) has taken root, asserted in a major journal as, "The most common etiology of fetal hypoxia during labor is umbilical cord occlusion, evidenced as 'variable' FHR decel-

Figure 3: FHR decelerations in response to umbilical cord occlusion (Reproduced with thanks from Westgate et al. [29]). Many sheep fetuses were subjected to repeated cord occlusions of 1 minute. The study reported decelerations lasting for 1 minute and a rapid recovery (with or without overshoot) immediately after the release of occlusion. This is a very substantial empirical evidence that the decelerations due to chemoreflex (hypoxemia) commence recovery only when hypoxemia starts recovering (not at the height of contraction like the common decelerations in Figure 1).

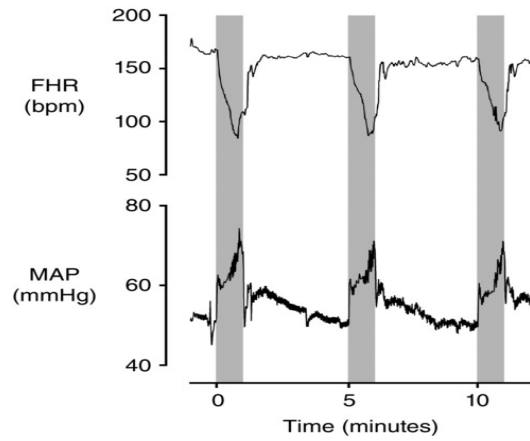


Figure 4: FHR decelerations during 1-minute umbilical cord occlusions in sheep fetus (Reproduced with thanks from Lear et al [13]).

It is noteworthy that the FHR continues to fall almost right until the end of hypoxemic period and a rapid recovery is seen only when occlusion is released corresponding to the recovery from hypoxemia (unlike the common decelerations in Figure 1).

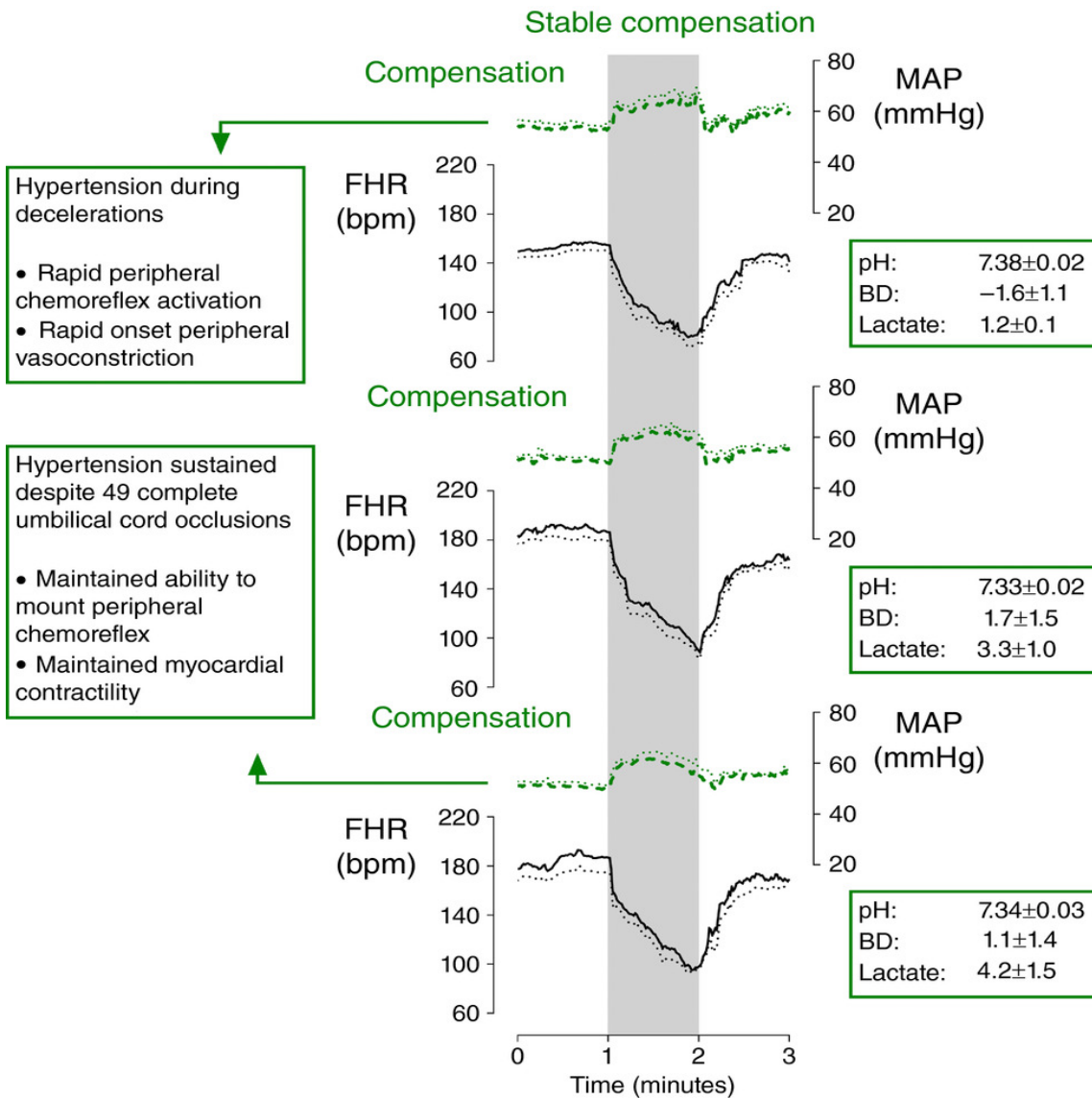


Figure 5: Schematic illustration of hypoxemic FHR decelerations (reproduced with thanks from Lear et al [9]). During 49 complete cord occlusions of 1 minute each in sheep fetuses. Note the FHR continues to fall until the end of hypoxemic period and recovery occurs after release of cord occlusion i.e. during recovery from hypoxaemia (unlike common decelerations in Figure 1).

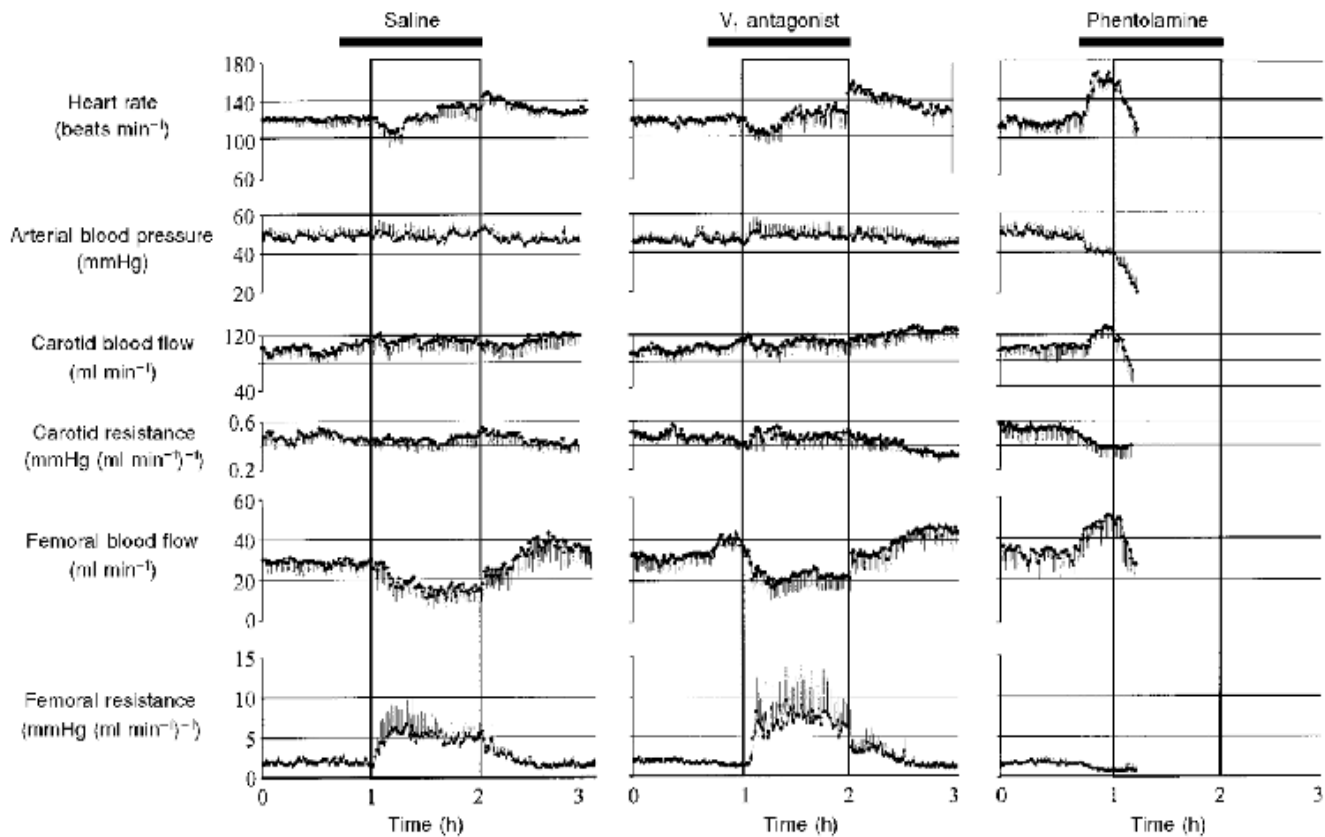


Figure 6: Fetal cardiovascular response to acute hypoxia (Reproduced with thanks from Giussani et al [32]).

Six pregnant llamas were subjected to respiratory hypoxia lasting for 1 hour. The FHR continued to drop for 15 - 25 minutes before any acclimatisation/recovery (top row, first two columns). This empirical evidence shows that the common decelerations in labour (Figure 1) where rapid recovery consistently starts around the peak of contraction cannot be explained by hypoxemia but by non-hypoxemic reflex mechanisms, which reverse at the peak of contraction [6].

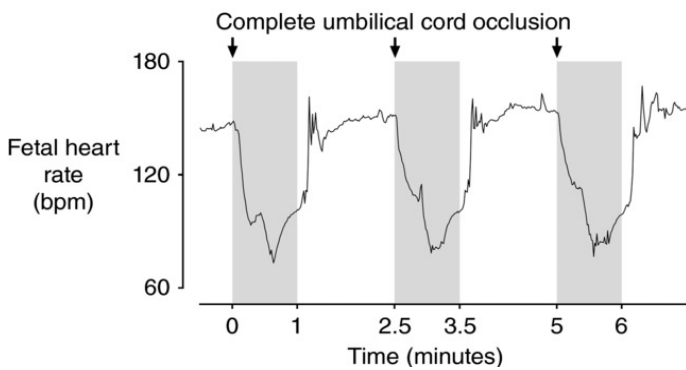


Figure 7: A somewhat atypical example of decelerations during umbilical cord occlusions (Reproduced with thanks from Lear et al [30]).

During complete umbilical cord occlusion for 1 minute, fetal heart rate falls to a nadir and then partially (25%) recovers after about 40 seconds, but most of the recovery (75%) finally starts after relief of cord occlusion. This example has been forwarded to assert that majority of decelerations in labour are due to hypoxaemia (chemoreflex) and can recover at the height of contraction [30]. However, these decelerations do not at all look like the common decelerations in labour shown in Figure 1 where complete and rapid recovery starts at the peak of contraction. Moreover, this imperfect exception does not disprove the rule revealed by almost all other animal cord occlusion experiments (Figures 3 - 6).

Perils of placing all decelerations in one basket (abandoning early /late /variable categorization)

As discussed above, placing almost all FHR decelerations into the single ‘hypoxemic’ (chemoreflex) or ‘variable’ (cord-compression) category is unscientific and untruthful. Moreover, this leads to the loss of meaning in obstetric practice because of an untruthful pattern-recognition as discussed next. Science has been aptly defined as unrelenting pursuit of truth. Cahill et al [33] in a prominent paper commented, “The summary nature of the current American nomenclature of FHR decelerations may lead to loss of information.” They were quite right because in their study [33] they found none early, very few late and almost all variable decelerations (personal communications with the authors in 2013). As a result, Cahill et al found no correlation between the fetal condition and the mistaken deceleration types [11,33]. The current American system [3] mistakenly categorizes most decelerations as ‘variable’. Not surprisingly, their further discrimination has remained unsuccessful including the ‘size’ criteria prominently proposed by a major group of American experts [34]. The National Institute of Health and Care Excellence (NICE) in UK switched over to similar ‘size-criteria’ in 2014 [5]. But the experience with these ‘size-criteria’ in the NHS hospitals (including Oxford) was a major disappointment likened to a train crash (personal communications with the RCOG). Hence, in 2017, the NICE made a ‘U’ turn back to the ‘typical/ atypical waveform criteria’ (also contested, some disproven), abandoning the size criteria [5,6]. A recent good quality American study showed that CTG interpretation based on prevalent or ‘size’ criteria detected only about 30% of academic babies under practice conditions and only 46% even on retrospective expert review [35].

Recently, the longstanding consensus that many FHR decelerations during labor are triggered by non-hypoxic mechanisms [1-6] has been unscientifically challenged [9]. Additionally, a concern has been expressed that this belief in non-hypoxic decelerations may have deemphasised the importance of repeated fetal hypoxemia in labor [9]. In contrast, the reverse seems to be true. Information is contained in a truthful pattern-recognition. If some/many FHR decelerations were to be of non-hypoxic origin but misclassified as due to hypoxemia (chemoreflex), then the birth-attendants wouldn't be able to separate wheat from chaff and a false alarm fatigue would set in. Furthermore, chemoreflex has been claimed to be an indefatigable 'guardian' of fetal adaptation to hypoxemia [9] despite it providing a time bound and constrained protection to the fetus. Such unrealistic and untruthful rhetoric may lead to 'procrastinating/normalising' the hypoxic decelerations. Although human fetuses do compensate for some degree of hypoxemia for some time, many other factors (like fetal reserve, maternal health, strength and frequency of contractions etc.) tilt the balance to hypoxic morbidity. These factors are not reliably/precisely measurable. It has been proposed that identification of hypoxemia per se is unhelpful due to the 'indefatigable' chemoreflex and the actual injury occurs in a narrow window between the intact survival and death [9]. Unfortunately, this is not a clear / safely defined window to the birth-attendants in human labor. Delivering the fetus cleverly just before this narrow window may be playing with fire unless one doesn't mind risking being born with a sheep brain! Finally, it has been proposed that we should look for or focus on some biomarkers of impaired myocardial contractility to time the delivery because that is associated with progressive fetal hypotension/cerebral-hypoperfusion [9]. But, should we wait until the myocardial contractility becomes impaired which would be the last to fail? Most studies agree that the babies be delivered with $\text{pH} < 7.10$; long before fetal hypotension / myocardial impairment [11,12,33,34]. Another ideology of rapid versus gradual decelerations disregarding early/late timing has been shown to be posing a significant risk to the babies [10]. Cardiotocography interpretation invariably involves some learned intuition which is subjective, experiential and qualitative. A false pattern-recognition would be bad science and wrong preconditioning undermining the development of such cognition and intuition.

Another impetus to abandon early/late decelerations comes from a well-intended claim that the total Deceleration Area (DA), rather than the deceleration types, shows the best correlation with the fetal acidemia [9,11,12]. It has been claimed that the use of total DA would necessitate only five Caesareans to prevent one case of acidemia [9,11]. However, this seems an oversight/reporting error because the Positive Predictive Value (PPV) of the DA to predict acidemia ($\text{pH} < 7.10$) has been reported to be 4% [11]. That translates to 25 caesareans (or expedited deliveries) being performed to prevent one case of acidemia [11]. Moreover, the best practical cut-off for the 'normal DA' would miss detection of 45% of the academic babies and 57% of the babies with composite hypoxic morbidity [11]. Even with computerized CTG, the ROC curve for 'DA' revealed that if one aims to detect 95% (desirable goal) or 80% or 50% of the academic fetuses, almost 90%, 45% and 25% of the controls (non-academic fetuses) respectively shared the same DA [12], hence not useful in clinical practice. The 'DA' may have a complementary role in future. Importantly, the early/late categorization of decelerations was the 'low hanging fruit' (often associated with stronger prediction) picked up by the pioneers [1,2].

Vital clinical empirical evidence for using early/late/variable deceleration categorization

Clinical studies and randomised controlled trials on CTG have been largely inconclusive because of multiple intractable methodological problems like low signal, numerous confounding factors and intervention changing the outcomes. Despite these odds, a prominent British study of 19,460 babies analysed perinatal outcomes in 1998, introduced team-based CTG training in 2000 and reanalysed outcomes in 2003 [36]. Infants born with 5-minute Apgar scores of < 6 decreased from 8.7 to 4.5 per 1,000 births ($P < 0.001$) and those with all grades of Hypoxic Ischemic Encephalopathy (HIE) decreased from 2.7 to 1.3 per 1,000 births ($P = 0.032$) [36]. This is almost a unique example of a large, good quality study showing excellent clinical outcomes with CTG as well as sustained improvement with training. Importantly, these training courses taught the traditional British practice (1970 - 2007) with primarily timing-based categorization of decelerations (early decelerations were majority and variable minority) [6]. The UK adopted a contradictory pattern-recognition consisting of majority variable decelerations in 2007 [5,6]. Since then, despite a decade of escalating training, resources and practice-improvements, the incidence of severe brain injury has remained 1.3/1000 births [37]. HIE of all grades would be expected more than three times that, a many times worse outcome compared to 1998 - 2003 [36]. Furthermore, there was a good degree of satisfaction with the traditional CTG interpretation (1970 -2007) in UK in contrast to current frustration and weariness on both sides of the Atlantic [6]. The late decelerations do have a low specificity for fetal acidemia. Future research should focus on further discrimination of late decelerations to improve their specificity to predict fetal acidemia and morbidity. This may include depth/duration of late decelerations or discriminating their shape (V or U) being cautious of the effect of depth of decelerations and scale of CTG paper on these shapes and taking into consideration the background risk factors. Confirmatory tests like FSBS, fetal stimulation or fetal ECG seem necessary and need more evaluation.

Reformed scientific definitions of FHR decelerations have been proposed previously [38] supported by practical experience and fairly robust empirical and clinical evidence, also presented in this article. Fortunately, there seems some progress recently in that the eFM (the official on-line CTG training programme of the Royal college of Obstetricians and Gynaecologists and the Department of Health, UK) has in the last couple of years accepted these definitions which are based on 'timing' alone without the previous flawed reference to 'uniform' or 'rapid/gradual' shapes [38,39]. This would make 'early' decelerations the commonest consistent with the scientific evidence.

Pathological science

The concept of "pathological science" [14] constitutes false science articles in reputed leading journals which attract readers' attention and hold a distinct potential of substantial publicity which can cause long lasting damage, best exemplified by Andrew Wakefield's 'vaccine theory of autism'. Most often there is no dishonesty (fabrication, falsification or plagiarism) involved; instead, experts are found to have misled themselves by insufficient scepticism about evanescent, irreproducible or ambiguous patterns, confirmation bias and belief persistence [14]. Unfortunately, a continuing repetition of 'pathological science' in reputed journals [9,18,40] has a drip-drip corrosive effect [41]. Chemoreflex is anything but indefatigable and there is no poetic licence in science. Recommending birth-attendants

to simply observe hypoxemic decelerations almost indefinitely [40] is dangerous. Birth-attendants will have to pay the cost of such unsafe recommendation.

Conclusions

This review shows very good observational as well as plenty of robust empirical evidence (human and animal studies) that the most common rapid short-lasting decelerations with recovery corresponding to the peak of contractions (**Figures 1 and 2b**) can only be explained by non-hypoxemic vagal reflex, the prime possible mechanism being head-compression (and placental compression?) [6]. Similarly, the plenty of analytical, observational and robust empirical evidence presented also shows that the FHR decelerations induced by hypoxemia from drop in uteroplacental perfusion or cord-compression would have a delayed trough and recovery starting late in the relaxation phase of the contractions (**Figures 2a and 4-7**). There are serious limitations of Deceleration Area (DA) and Capacity (DC) making them unsuitable alternatives to categorization of FHR decelerations [6]. Cardiotocography has had a difficult period because of the false and unscientific pattern-recognition. This has provided a fertile ground where new attractive but unscientific ideas can be seen to quickly become popular memes. Conforming with the robust empirical evidence presented in this article, it would be of paramount importance to adopt the primarily timing-based categorization of non-hypoxemic (early) and hypoxemic (late) decelerations, also crucial for the future computerized algorithms.

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