



Study of interpretation of cardiocography educational illustrations by experts discarding early/late/variable deceleration categorization – Physiology or unscientific ideology, myths and road to perdition?

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Abstract

Objective: To evaluate the safety/risks of the doctrine of abandonment of categorization of fetal heart rate decelerations into early/late/variable by the expert group from St George's University Hospital, London; by analyzing the reproducibility and results of interpretation of their cardiocography teaching illustrations.

Design: Prospective observational study.

Setting: Five National Health Service Hospitals, UK.

Participants: 12 obstetric consultants, 10 registrars, 30 midwives.

Methods: Anonymised questionnaire. Statistics - Fisher's Exact test.

Results: The pure baroreceptor 'benign' cord-compression decelerations in the exemplar illustration 1 by the expert group were interpreted as pathological (hypoxemic) by 88% of the study participants ($P < 0.0001$). Illustration 4 presented as "suspicious" by expert group was interpreted as "pathological" requiring urgent fetal blood sampling/delivery by 88% participants ($P < 0.0001$). The illustration 5 was interpreted by 92% participants as requiring urgent fetal blood sampling/delivery. Mild or moderate to severe hypoxemic ischemic encephalopathy was considered likely by 23% and 42% of participants respectively. This was because of the grossly pathological decelerations termed as 'late' or 'late atypical variables' by 69% and 23% participants respectively. The expert group did not classify the decelerations because of their ideology and advised conservative measures failing to recognise the great danger to fetus, despite privy to knowledge of terminal bradycardia 10 minutes later.

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Keywords: Fetal heart rate decelerations; Intrapartum fetal monitoring; Electronic fetal monitoring; Physiology of fetal heart rate decelerations; Birth asphyxia; Hypoxemic ischemic encephalopathy

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Conclusions: The ideology (presented as physiology) by the expert group that ‘rapid’ decelerations are due to baroreceptor mechanism and that the chemoreceptor (hypoxemia) induced decelerations are ‘gradual’ has been disproven by many studies. The expert group’s doctrinal refusal of early/late/variable decelerations leads to their highly dangerous recommendation that the fetus is decompensated only when deepening decelerations are combined with maximal rise in baseline heart rate and absent baseline variability. The obstetricians, midwives and professional regulatory bodies should disallow such a perilous ideology. A few myths at the core of current CTG interpretation are also discussed.

Introduction

Cardiotocography (CTG) remains the most widely practiced technique of intrapartum fetal monitoring in the developed world. But because of its massive complexity it has had more than its fair share of controversies. The “Each Baby Counts” 2018 progress report by the Royal College of Obstetricians and Gynaecologists (RCOG, London) states that out of 700,000 babies born in 2016, there were 124 stillbirths, 145 babies died early and 854 babies sustained severe hypoxic brain injuries during labor at term [1]. In addition, there would be many times more cases of mild to moderate hypoxic neonatal morbidity. Apart from the medicolegal and economic costs, every case of hypoxic brain damage is a major human tragedy. CTG interpretation involves recognition of intricate patterns of Fetal Heart Rate (FHR) parameters in the context of complex clinical settings. The FHR decelerations are center-stage (most important) as well as most complex and controversial. Meaningful pattern recognition will only be possible if scientific framework of patterns is practiced [2]. In the UK, there has been a crisis in the form of a plethora of different recommendations / theories since the concept of rapid vs gradual FHR decelerations crept into practice from 2007 [3]. It has become fashionable to misinterpret, devalue and even abandon the simple but time-tested categorization of FHR decelerations into early, late and variable based on the pioneering work of Edward Hon [4]. This ideology

has been tested in this paper by critically evaluating the interpretation by practicing clinicians of a few key teaching illustrations of CTG. Thus the objective is radically different from just demonstrating inter-observer variation in CTG interpretation.

Materials and methods

A prospective observational multicentre study was conducted in February and March 2019 to compare the interpretation by Consultant Obstetricians, Registrars and midwives of five educational CTG traces published by a British opinion leader expert group of Edwin Chandraharan of the St George’s University Hospital, London [5].

The participants in the study were recruited on a non-selective voluntary basis face to face or by postal/email correspondence, using an anonymised questionnaire. This was an extension of study for which an approval of the Research and Development (R&D) committee of the author’s institute was obtained. Although the selection could not have been completely random, this was not necessary or relevant in common with other reported studies [6,7,8]. Participants were not informed of the source of illustrations. Ethical approval was not required by the R&D committee because no known patient data or clinical intervention was involved. Total of 52 participants (12 obstetric consultants, 10 obstetric specialist registrars and 30 midwives) from five NHS hospitals in different regions of UK agreed to participate on anonymous basis. They were asked to interpret five original CTG teaching illustrations by British experts. These original illustrations can be seen in the relatively well-known book [5]. Because of copyright issues those original illustrations cannot be reproduced in this paper but figures 1-5 show different but very similar illustrations highly representative of the original ones. The images of the original CTG illustrations were resized (enlarged) proportionately where required to match the actual size used in clinical practice. All obstetricians and midwives had undergone regular mandatory CTG training by approved trainers [3] at intervals of about every 12 months on many occasions. All participants were certified to be competent in CTG interpretation. Cochrane recommended Fisher’s Exact test was used to check statistical significance.

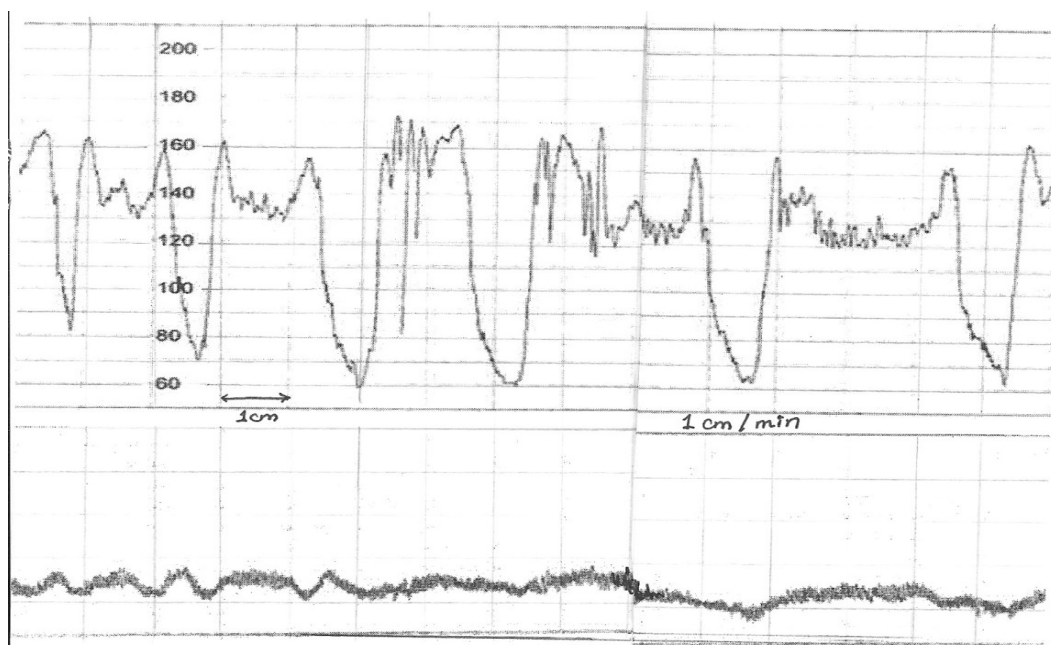


Figure 1: CTG illustration 1: Highly representative of the original illustration by the expert group (Figure 5.2, page 35) [5].



Figure 2: CTG illustration 2: Highly representative of the original illustration by the expert group (Figure 5.1, page 34) [5].

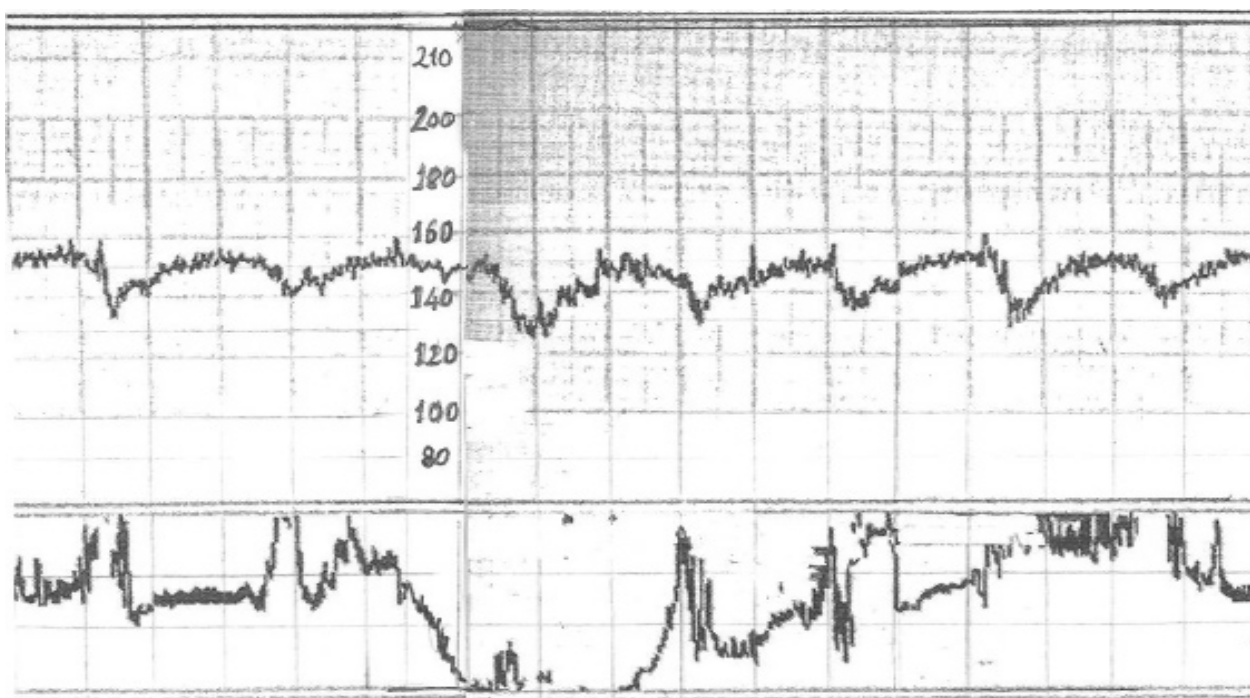


Figure 3: CTG illustration 3: Highly representative of the original illustration by the expert group (Fig 5.3, page 201) [5].

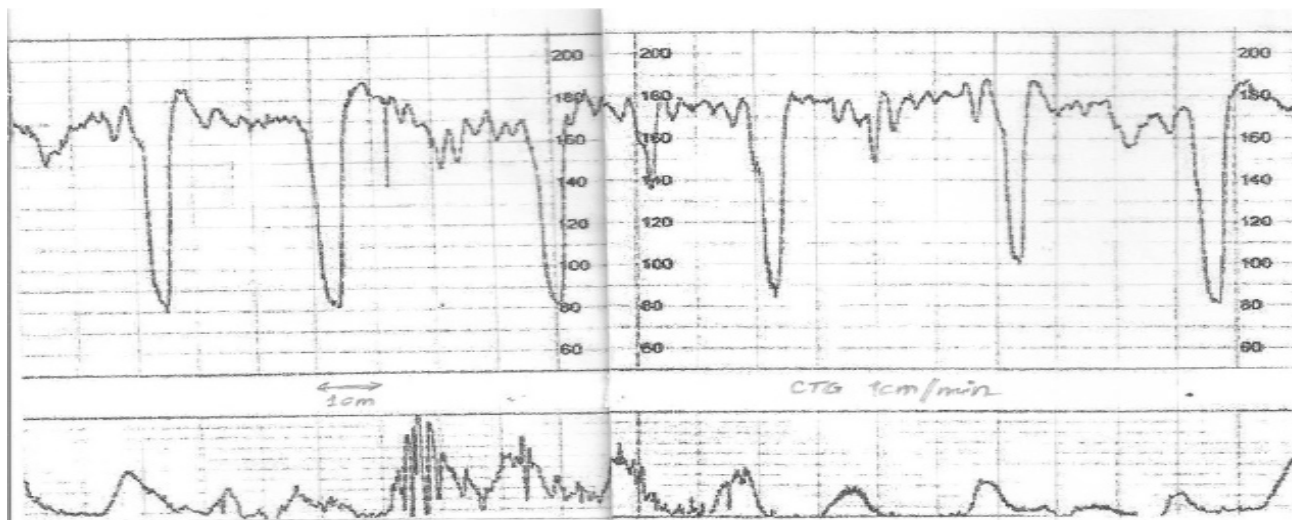


Figure 4: CTG illustration 4: Highly representative of the original illustration by the expert group (fig 5.8, page 201) [5].

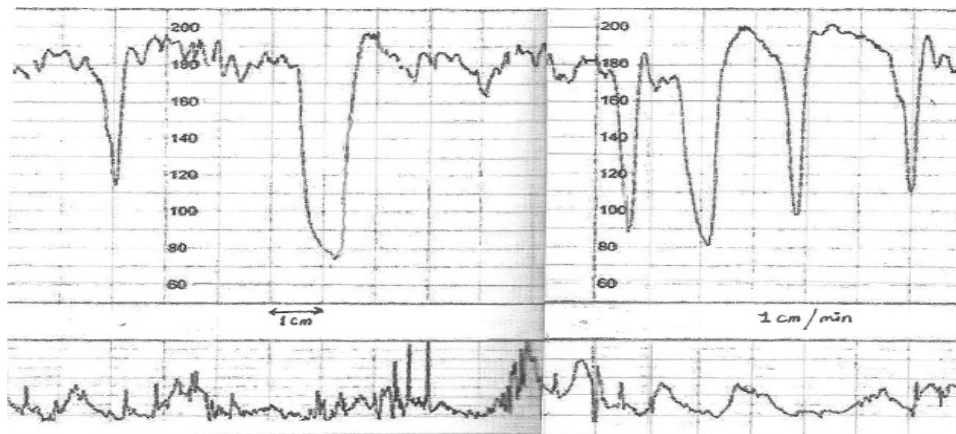


Figure 5: CTG illustration 4: Highly representative of the original illustration by the expert group (fig 5.9, page 202) [5]

Table 1: The percentage of different opinions on CTG in illustration 1 by the expert group [5] and study participants

	Benign FHR decelerations	Atypical Variable or Late FHR decelerations	Pathological CTG	Fetal scalp blood sampling (FSBS) or Deliver
St Georges Hospital Expert opinion	100 %	(Do not believe in categorization)	0 %	0 %
Study Participants' Opinion (Actual numbers in parentheses) Total No. 52	7 % # (4) (95 % C I; 2 % – 18.5 %) P < 0.0001	88 % (46) (95 % C I; 77 % – 96 %)	85 % # (44) (95 % C I; 72 % – 93 %) P < 0.0001	73% # (38) (95 % C I; 59 % – 84 %) P < 0.0001

P < 0.0001 (Fisher's exact test) CTG: Cardiotocography; FHR: Fetal Heart Rate

Table 2: The percentage of different opinions on CTG in illustration 2 by the expert group [5] and study participants.

	St Georges Hospital Expert opinion	Study Participants' Opinion (Actual Numbers in parentheses) Total No. 52
Fetal hypoxia likely	100 %	4 % # (2) (95 % C I; 0.5 % – 13 %) P<0.00001
Fetal hypoxia unlikely	0 %	96 % # (50) (95 % C I; 87 % – 99 %) P<0.00001
No FHR decelerations	0 %	12 % (6) (95 % C I; 4 % – 23 %)
Early FHR decelerations	No categorization	15 % (8) (95 % C I; 7 % – 28 %)
Typical variable FHR decelerations	No categorization	35 % (18) (95 % C I; 22 % – 49 %)
Atypical variable FHR decelerations	No categorization	15 % (8) (95 % C I; 7 % – 28 %)
Late FHR decelerations	No categorization	4 % (2) (95 % C I; 0.5% – 13%)
Normal CTG	No comments	81 % # (42) (95 % C I; 67 % – 90 %) P<0.00001
Suspicious CTG	No comments	19 % (10) (95 % C I; 10 % – 32 %)
Pathological CTG	No comments	0 % (0) (95 % C I; 0 % – 6 %)
Action: continue to observe	100%	88 % (46) (95 % C I; 77 % – 96 %)

P<0.00001 by Fisher's Exact Test

Table 3: The percentage of different opinions on CTG in illustration 3 by the expert group [5] and study participants.

	St Georges Hospital Expert opinion	Study Participants' Opinion (Actual Numbers in parentheses) Total No. 52
Fetal hypoxia likely	100%	85 % (44)
Fetal hypoxia unlikely	0 %	15% (8)
Early FHR decelerations	No categorization	7 % (4)
Typical variable FHR decelerations	No categorization	23 % (12)
Atypical variable FHR decelerations	No categorization	31 % (16)
Late FHR decelerations	No categorization	38 % (20)
Normal CTG	No comments	0 %
Suspicious CTG	No comments	40 % (21)
Pathological CTG	No comments	60 % (31)

Table 4: The percentage of different opinions on CTG in illustration 4 by the expert group [5] and study participants.

	Atypical Variable FHR decelerations	Late FHR Decelerations	Suspicious CTG	Pathological CTG	Mild HIE	Moderate/severe HIE	FSBS or Deliver
St Georges Expert opinion	No belief in categorization of decelerations	No belief in categorization of decelerations	100 %	0 %	0 %	0 %	0 %
Study Participants' Opinion (Actual Numbers in parentheses) Total No. 52	35 % (18) (95 % CI; 22 % – 49 %) P value: Not applicable	62 % (32) (95 % CI; 47 % – 74 %) P value: Not applicable	15 % # (8) (95 % CI; 7 % – 28 %) P<0.00001	85 % # (44) (95 % CI; 71 % – 93 %) P<0.00001	38 % # (20) (95 % CI; 25 % – 53 %) P<0.00001	15 % ¥ (8) (95 % CI; 7 % – 28 %) P<0.05	88% # (46)(95 % CI; 77 % – 96 %) P<0.00001

P<0.00001, by Fisher’s Exact Test ¥ P<0.05, by Fisher’s Exact Test
 CTG: Cardiotocography; FSBS: Fetal Scalp Blood Sampling; HIE: Hypoxic Ischemic Encephalopathy; FHR: Fetal Heart Rate

Table 5: The percentage of different opinions on CTG in illustration 5 by the expert group [5] and study participants.

	Atypical Variable FHR decelerations	Late FHR Decelerations	Suspicious CTG	Pathological CTG	Mild HIE	Moderate/severe HIE	FSBS or Deliver
St Georges Hospital Expert opinion	(No belief in categorization of FHR decelerations)	(No belief in categorization of FHR decelerations)	0 %	100 %	0 %	0 %	0 %
Study Participants' Opinion (Actual Numbers in parentheses) Total No. 52	23 % (12) (95 % CI; 12 % – 37 %) P value: Not applicable	69 % (36) (95 % CI; 55 % – 81 %) P value: Not applicable	0 % (95 % CI; 0 % – 6 %)	100 % (52) (95 % CI; 93 % – 100 %)	23 % # (12) (95 % CI; 12 % – 37 %) P<0.00001	42 % # (22) (95 % CI; 29 % – 57 %) P<0.00001	92 % (48) (95 % CI; 81 % – 98 %) P<0.00001

P<0.00001, by Fisher’s Exact Test, C I: Confidence Interval, CTG: Cardiotocography; FSBS: Fetal scalp blood sampling; FHR: Fetal Heart Rate; HIE: Hypoxic Ischemic Encephalopathy.

Results

The results are summarized in tables 1-5. There was no significant difference in the interpretation of consultants, registrars and midwives, hence their opinions were pooled together. The more important illustrations and tables are 1, 4 and 5.

CTG illustration 1 (Figure 1)

This CTG illustration was given by the expert group [5] as a defining or characteristic example of purely baroreceptor mediated cord-compression FHR decelerations, which means there is no element of fetal hypoxemia invoking chemoreflex. Hence, these FHR decelerations could be similar to category of “typical benign variable decelerations” of British national guidelines [3], although these particular experts’ teaching has been not to attribute a category to decelerations. Many clinicians commented that the contraction trace (tocograph) is extremely poor for a teaching illustration. Where a few contractions could be made out, these very deep (50 -70 bpm depth) decelerations can be seen to be late in timing. Hence, 88 % participants called these decelerations ‘late or atypical variable with late timing’. Remarkably, although the experts gave this example of a “normal” (non-hypoxemic) CTG; 85 % of the study participants called the illustration 1 to be a ‘pathological CTG’ (p<0.00001).

CTG illustration 2 (Figure 2)

This teaching example was given by the British expert as demonstrating ‘onset of hypoxic stress’ [5]. The FHR decelerations (only about 15 -20 bpm in depth) have been described as due to fetal hypoxia [5]. In contrast 96% of the study partici-

pants thought hypoxemia to be unlikely, 81% thought decelerations to be absent, difficult to make out or non-hypoxic.

CTG illustration 3 (Figure 3)

There was good correlation between the expert opinion and the interpretation by the study participants. The CTG was classed as pathological or suspicious by all the participants and decelerations as pathological by 69%.

CTG illustration 4 (Figure 4)

The expert group in their textbook [5] commented on this CTG as follows, “Note a further increase in FHR to above 170bpm which indicates a degree of catecholamine surge. The fetus is still attempting to maintain a stable baseline FHR and a reassuring variability, but rapid decompensation may ensue if oxytocin infusion is further increased. The clinicians considered the on-going FHR decelerations as ‘typical variable’. The clinicians failed to notice the increase in the FHR baseline and therefore wrongly classified CTG trace as ‘normal’ rather than ‘suspicious’, and increased the oxytocin further.”

The baby seems to have perished in labor about 1 hour 20 minutes after this CTG illustration 4 which the expert group would be aware [5] but not the study participants. It is worth noting that the expert group did not categorize the decelerations in this case and did not mention these to be pathological. In marked contrast, 97% of the study participants identified these decelerations as hypoxemic (late or atypical variable with late components). Most participants (88%) advised complete stoppage of oxytocin infusion, resuscitative measures (some advised

fetal scalp blood sampling) and delivery. Many participants indicated that there would be significant fetal hypoxemia / acidemia in the presence of this CTG trace and some even commented that there would be a possibility of mild (38%) or moderate (15%) hypoxic-ischemic encephalopathy (HIE) at this stage. In striking contrast, the expert group made no recommendation of any intervention (apart from conservative measures) and recommended oxytocin to be continued at the same rate [5].

CTG illustration 5 (Figure 5)

The expert group in their textbook [5] made following comments, "This CTG trace clearly shows a final attempt by the fetus to maintain oxygenation by increasing the baseline FHR to 200 bpm (Figure 5). The onset of cerebral hypoxia and acidosis will result in a reduction in baseline variability (Figure 5). The onset of reduction in baseline variability required urgent action to improve uteroplacental circulation. These include immediate stopping of oxytocin infusion, rapid infusion of intravenous fluids to dilute oxytocin concentration in maternal circulation (*another misconception*), and changes in maternal position to relieve umbilical cord compression (*Really?*). Tocolytics should be used if variability does not improve with initial measures [5]."

The baby had a terminal bradycardia about 10 minutes after this CTG which the expert group knew but not the study participants. The expert group did not categorize the FHR decelerations at all and made no comment about urgency of delivery of this baby [5]. In contrast, 100 % of study participants categorized the FHR decelerations as 'pathological' and 92% recommended urgent delivery together with resuscitative measures. Many commented that this CTG would already be associated with a chance of mild (23%) or moderate to severe (42%) HIE, and thereby already a chance of cerebral palsy.

Comments by midwives: When the midwives were approached with a request to participate in this study and give their opinion, they volunteered comments which they would not otherwise offer to obstetricians, experts or their teachers. These reflected dissatisfaction and frustration with the current system. The dysfunction is also abundantly evident from a voluntary comment by a young midwife with six years of experience (many others agreed) that while interpreting CTG currently, midwives quite often first decide what action feels necessary in that particular patient, then classify the CTG as normal / suspicious/ pathological tailored to justify that action. Then lastly, they fill in the relevant boxes for the FHR deceleration interpretation on the "CTG sticker (matrix)" in the patient notes to achieve that predetermined classification (personal communications). This amounts to CTG interpretation in a "reverse gear". In marked contrast, the previous British scientific physiological categorization of decelerations before 2007 was indeed very useful in clinical practice [9].

Discussion

This study demonstrates that there were major and statistically significant discrepancies in the CTG interpretation by the British opinion leader group [5] and the 52 study participants (obstetric consultants, midwives and specialist registrars). The information contained in a CTG pattern especially FHR decelerations depends on how coarsely or finely grained, scientific or flawed our view of the pathophysiology of decelerations is. The St George's expert group is almost unique in that they have been for many years strongly advocating/teaching abandonment of the categorization of FHR decelerations into early/

late / variable types as opposed all national professional bodies from the developed countries [5]. These experts state, "Instead of morphological classification of decelerations into early/variable/late, the clinicians should classify decelerations according to the main underlying mechanisms. Baroreceptor decelerations (occlusion of umbilical artery) are characterized by rapid fall and recovery. Chemoreceptor decelerations due to fetal hypoxia are characterized by gradual and slow recovery (page 34) [5]." The dictionary meaning of the word morphology is 'a particular form, shape, or structure'. Thus, without understanding, the St George's expert group are themselves promoting a 'morphological' categorization based on shape of the waveform 'rapid vs gradual'. In fact, this 'rapid vs gradual' ideology will be shown to be completely flawed and dangerous later on in this paper. In contrast, Edward Hon's pioneering categorization (and the traditional British categorization before 2007) was primarily 'timing based' and not morphological at all [2,4,9]. All national guidelines even today claim to derive their categorization of FHR decelerations from the description by Edward Hon [4]. Some opinion leaders seem to have a "doctrinal" practice and teaching not to recognise the early/late/ variable deceleration types e.g. as seen in the description of illustrations 1, 4, and 5. Most important question may be whether this is a safe or dangerous practice and teaching.

Illustration 1: The expert group seems to be wrongly teaching that the very deep pathological decelerations in illustration 1 (Fig 1) are 'benign' cord-compression decelerations because they have rapid descent and recovery. This baroreceptor hypothesis has been shown to be wholly unscientific (Fig 6,7). Moreover, the expert group does not seem to realise that for decelerations of depth 60-70 bpm to look "gradual on the British CTG (speed 1cm/min), the duration of the decelerations would have to a lot more than 6-7 minutes which is not plausible. As a result, deep (hence rapid) hypoxic decelerations will never be recognised as pathological which clearly is very unsafe practice and teaching. Almost all of the FHR decelerations in the illustration 1 by the expert group have the time of descent well above 30 seconds, hence will be classed as 'late' (hypoxic) even by the North American guidelines [10]. These decelerations do look eerily and alarmingly similar to the hypoxic FHR decelerations seen in cord prolapse generally requiring crash caesarean (Figure 8) and consistent with diagrammatic representation of deep hypoxic decelerations from cord-compression or rapid reduction in uteroplacental oxygenation (Figure 7). No wonder, even with a quick glance at illustration 1, many midwives, in complete contrast with the experts, commented, "That looks bad, I would be very worried with this CTG!"

Lastly, there is overwhelming evidence from animal studies for decades that the chemoreceptor stimulation (hypoxemia) is the main mechanism underlying the 'rapid' cord-compression decelerations [2,11-17]. Indeed during the well conducted studies of experimental cord-occlusion in sheep, the rapid drop in FHR long preceded the small rise in fetal mean arterial pressure [12]. Hence, the claim by the expert that there is a pure baroreceptor cord-compression deceleration [5] is unphysiological and misunderstood. There is probably no such thing as 'pure' baroreceptor mediated cord-compression decelerations. Misunderstanding of physiology aside, but not recognising the pathological late decelerations of about 70 bpm depth in illustration 1 would be very dangerous for the fetus. Fortunately, most of the study participants did recognise the seriousness precisely by categorizing these decelerations as late in contrast to the flawed ideology or doctrine of the expert group.

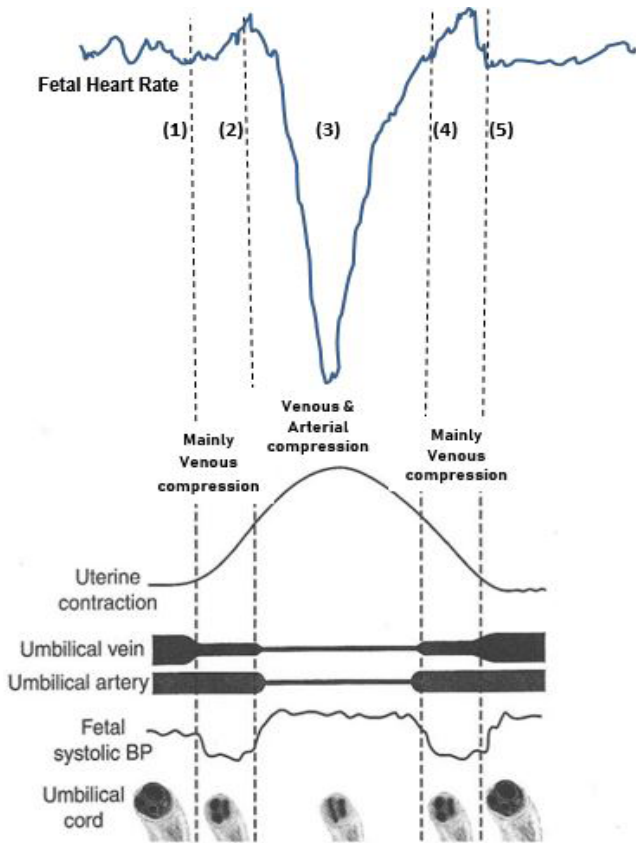


Figure 6: Flawed hypothesis of cord-compression - Schematic drawing for CTG speed 1cm/min. (1) UV occlusion is proposed to cause the fetus to lose blood volume leading to a small acceleration. (2) UA occlusion is proposed to cause rise in BP leading to rapid drop in FHR by baroreceptor stimulation. But, fetal blood volume stabilising at a lower level than before the contraction would not lead to significant rise in BP at all. (3) Relief of UA occlusion is proposed to lead to loss of blood volume again causing hypotension and rise in FHR with a small overshoot. But UA opens at point (4), where the recovery of deceleration should start rather than at point (3). Thus, the baroreceptor mechanism does not explain the drop of FHR nor the commonly seen recovery of FHR close to the peak of contraction. In fact, it is the chemoreflex from hypoxemia is accepted to be the main mechanism in cord-compression which will cause FHR deceleration as shown in figure1B. The deceleration depicted in figure 1A seems consistent with 'direct' or 'pure' vagal reflex (head compression rather than cord occlusion) [2]. FHR: Fetal Heart Rate; UV: Umbilical Venous; UA: Umbilical Arterial; BP: Blood Pressure

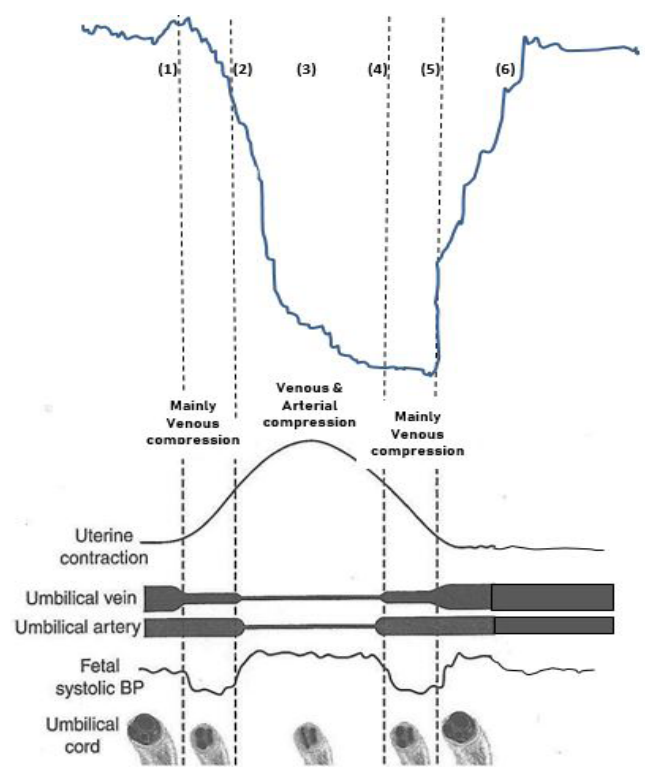


Figure 7: Truthful (pathophysiological) cord-compression deceleration: Schematic drawing for CTG speed 1cm/min. UV compression occurring at point (1) will produce progressive fetal hypoxemia with a slight delay and a chemoreflex mediated FHR deceleration. Immediately after the peak of contraction (point 3), the FHR drop will slow down but continue until UV compression is released at point (5), where the recovery of FHR will start. This deceleration will recover late after the contraction. There may be small accelerations before and after the deceleration simply as a reflection of longer but smaller sympathetic response. Thus, cord-compression, tends to cause deep late FHR decelerations as seen in practice during cord-prolapse (Fig 6), and hence it is implausible to be the cause of the common benign decelerations in labor. If similar degree of hypoxemia results from drop in uteroplacental oxygenation during contractions, then that would also result in rapid deep late decelerations on British CTG. Thus, gradual late decelerations will become rapid and deep as hypoxemia becomes severe. It has become a common pitfall in British practice to underestimate the seriousness of these rapid late deceleration [5]. FHR: Fetal Heart Rate; UV: Umbilical Venous; UA: Umbilical Arterial; BP: Blood Pressure

Cardiotocogram recording of FHR and contractions from a term human fetus who had a cord prolapse in early labour

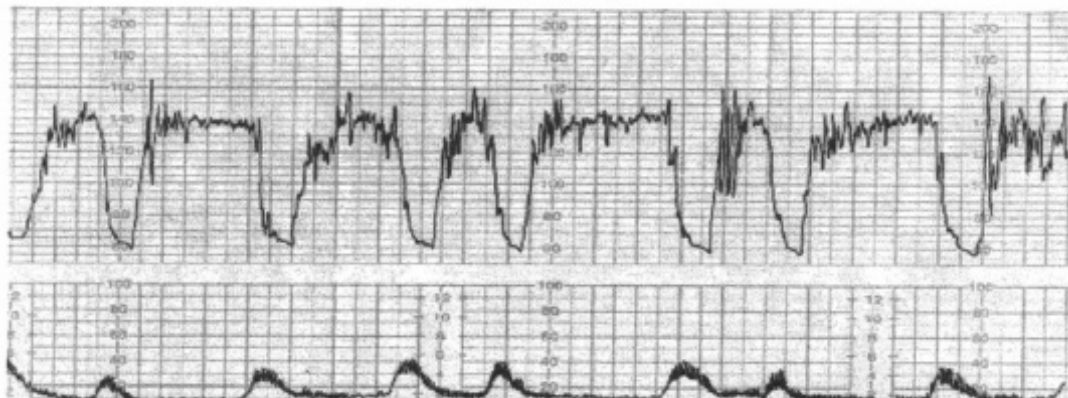


Figure 8: CTG in a case of cord prolapse showing late decelerations (reproduced with thanks from Westgate et al, AJOG, 2007 [11]). Because of severity and depth, these decelerations 'look' rapid (paper speed 1cm/min), but the 'descent-time' is well over 30 seconds (hence late by FIGO and American definitions). Most importantly, they are consistently "late" in timing.

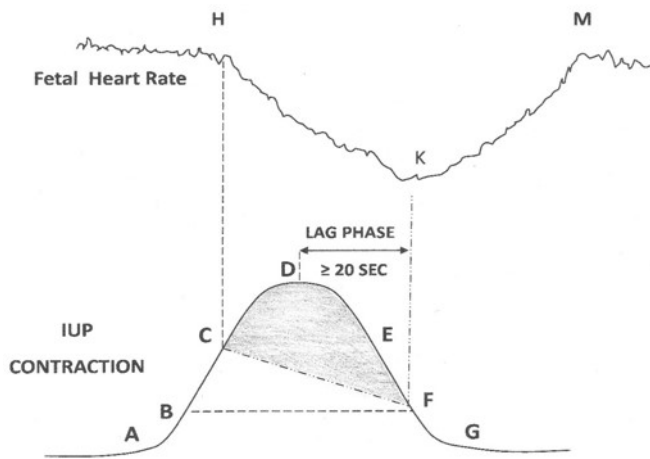


Figure 9A: Schematic drawing of FHR deceleration resulting from peripheral chemoreflex due to hypoxemia. Hypoxemic trigger is very likely produce a classical “late deceleration” [2].

A: Contraction commences, B: IUP enough to commence Fetal hypoxemia, C: Worsening fetal hypoxemia enough to start FHR deceleration, D: Peak of contraction where speed of worsening of hypoxemia will slow down but hypoxemia will continue to worsen (PaO₂ continues to drop), E: Hypoxemia will continue to worsen, F: Hypoxia will start recovering because IUP equivalent to point B. Chemoreflex induced FHR deceleration will start recovering at point F and recovery will extend beyond the end of contraction. Shaded area: Level of IUP where fetal PaO₂ will continue to drop during deceleration.

FHR: Fetal Heart Rate; IUP: Intrauterine Pressure; PaO₂: Fetal Partial Pressure of Oxygen

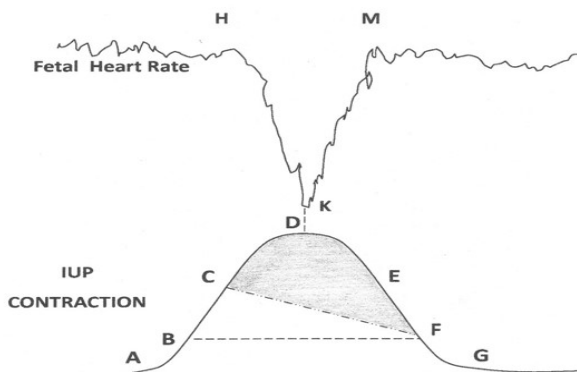


Figure 9B: Schematic drawing showing that the common rapid short-lasting FHR decelerations with early timing are unlikely to be due to fetal hypoxemia.

Some authors hypothesize mild fetal hypoxemia during most contractions [12]. Even then the FHR deceleration above can be best explained by benign non-hypoxemic reflex mechanism. This is because the recovery of decelerations starts at the peak of contraction, while the degree of hypoxemia will continue to worsen until point F.

Shaded area: Level of IUP where mild fetal hypoxemia may be hypothesized to develop, but not enough to lead to a hypoxemic deceleration.

Illustration 3: As an exception there was a good agreement between the expert opinion and interpretation of the study participants. The expert group states, “The depth of chemoreceptor deceleration is not marked as a baroreceptor deceleration” [5]. This seems a false ideology because it has been shown above that it is the chemoreceptor stimulation that causes the deep rapid cord-compression deceleration. The danger is that the very small 10 -15 bpm (hence gradual) decelerations will become deeper but not equally wider as hypoxemia worsens. These deep increasingly hypoxemic late decelerations will be then attributed to baroreceptor mechanism or called the common ‘variable’ decelerations, leading to procrastination by these experts and to severe fetal asphyxia, as indeed seen in illustrations 4 and 5.

The concept (phenomenon) of “Shift to the right” of FHR decelerations: Strong uterine contractions even in early labor can cause rapid FHR decelerations concordant with contractions, particularly in tumultuous, rapidly progressive or oxytocin stimulated labor. When recovery starts immediately after the peak of contraction, hypoxemic trigger seems extremely unlikely (Figures 9A and 9B) and a benign reflex mechanism like head compression or increased resistance in fetal placental vasculature (placental compression rather than cord compression) seem possible explanations [2]. If fetal hypoxemia / academia set in, these decelerations can be observed to “shift to the right” i.e. the trough of deceleration is more than 20 seconds later (lag phase) than the peak of contraction (Figures 4, 5). As with any numerical cut-offs, this “20 seconds” cut-off is not absolute. Rapid recovery of decelerations only seems to indicate quick cessation of the causation e.g. excessively strong contractions but without pre-existing uteroplacental compromise. Thus, the decelerations because of hypoxemia of uteroplacental origin need not always be gradual, particularly so during oxytocin augmented labor. When the trigger causes rapid hypoxemia, the ‘late’ decelerations would be rapid (Figures 4, 5). Moreover, as soon as the contraction is about to end, the FHR deceleration due to severe hypoxemia (most commonly from drop in uteroplacental oxygenation during contractions rather than cord compression) would also recover rapidly to re-perfuse the vital organs. Hence, the “late” timing seems crucial than the shape of decelerations. Such hypoxemic decelerations with late timing when allowed to continue unrecognised will progress to serious fetal acidemia and demise (Figure 4, 5).

Road to ‘Perdition’ and the Journey’s End

Where does the flawed doctrine or ideology (certainly not physiology) of refusal to categorize patterns of FHR decelerations lead to? This destination is very well surmised in the following description in the book by the expert group, “In gradually developing hypoxia, delivery should be undertaken when there is maximal rise in baseline rate with increasing depth and duration of decelerations with reduction of inter-deceleration intervals and marked reduction in baseline variability for a period of 1 hour, unless fetal scalp pH (clearly after 1 hour) shows that further observation is safe” [18]. Most obstetricians would consider this an extremely dangerous doctrine, a road to ruin for the unborn babies and the science of CTG. This doctrine can be seen to have been put into practice during the interpretation of illustrations 4 and 5 by the expert group [5] leading to major risk to the fetus as discussed later. Gradually developing hypoxia is the commonest cause of birth asphyxia and seems the most preventable as well. Very importantly, the above dangerous recommendation is in direct contradiction of a recent very high-

quality study [19] (editor's choice) of 5388 patients that only 8.8% of 57 significantly acidemic babies requiring delivery (pH < 7.10) had reduced baseline variability and only 12.3% had baseline tachycardia. A cut-off of umbilical artery of pH 7.10 was selected as acidemia serious enough to deliver but not leading to hypoxic morbidity [19]. Even bigger high-quality study of 8580 deliveries with 149 acidemic babies (pH<7.10) showed that only 29% of these acidemic babies had reduced baseline variability [20]. More importantly only 3 out of 149 acidemic (pH< 7.10) babies had a combination of recurrent FHR decelerations and markedly reduced baseline variability (Category III), not to speak of 'maximal' baseline tachycardia in addition [20]. In a smaller study of 488 fetuses, 31% had an umbilical artery pH \leq 7.0 (dangerously and unacceptably low) when the FHR variability was minimal or absent in the last hour [21]. Another small study showed that 10 out of 11 fetuses with absent variability, usually accompanied by late or variable decelerations, had dangerously severe acidemia (base deficit>16mEq/L), which must be avoided [22]. Late decelerations have been shown to have a significant correlation with fetal hypoxemia [3]. The dangerous doctrine above [5, 18] seems to be based on the ideology of non-recognition of late decelerations and a very unsafe misconception that babies need to be delivered when a significant risk of dangerous acidemia of pH < 7.00 is reached (which in the presence of deep hypoxemic deceleration would represent real and present danger of severe hypoxic morbidity and cerebral palsy). Almost all obstetricians would agree that babies should be delivered when umbilical artery pH falls to around 7.10 but well above 7.0. This seemingly can only be done by differentiating different types of FHR decelerations to judge which are hypoxemic ones and then add another test like fetal blood sampling where appropriate [23]. Not classifying FHR decelerations at all [5] is not a physiological approach but seems a hazardous practice and teaching placing unborn babies at unnecessary severe risk. Fortunately, it seems likely that where fetal ECG ST-analyser (STAN) technology is practiced, the CTG guidelines by STAN would have to be followed which do recognise early, late and variable decelerations.

Illustrations 4 and 5: These teaching illustrations given by the expert group as an exemplar of their CTG interpretation seem to offer a reliable proof of failure to recognise seriously pathological FHR decelerations directly as a result of the doctrinal practice and teaching (see results above). Most study participants wanted stoppage of oxytocin infusion and urgent intervention to deliver the baby because of seriously pathological CTG in illustration 4. The experts (even in the knowledge that the baby had terminal bradycardia about 1 hour 20 minutes later) recommend continuing oxytocin infusion at the same rate and mention nothing about delivering the baby. With CTG illustration 5, a large numbers of study participants (42%) recognised that there was likelihood of moderate to severe HIE (and cerebral palsy), hence it was already a bit too late; nevertheless, urgent resuscitation and delivery was required. Even this number (42%) seems disappointingly low in author's opinion and is reflective of the current state of confusion in CTG interpretation. The expert group on the other hand commented that the fetus has just got decompensated at this stage (figure 5: maximal tachycardia, increasingly deeper deceleration and marked reduction in baseline variability) and conservative management would somehow bring the fetus back from the brink. Their knowledge (as opposed to the study participants) that the fetus had terminal bradycardia about 10 minutes after the CTG in illustration 5 [5] merits introspection. To the author's

understanding, if a midwife in UK steadfastly refuses to recognise 'late' decelerations, he/she is very likely to face disciplinary measures and retraining.

Scientific Physiology of FHR decelerations

The 'rapid vs gradual shape' of FHR decelerations is not discriminatory of presence / degree of hypoxemia, underlying mechanism or the causative factors (presumptive etiology) [2]. Moreover, this flawed ideology is riddled with many contradictions [23]. In contrast, the primarily timing-based categorization into early / late / variable by Edward Hon [4] has a time-tested scientific physiological basis as shown in Figures 9 A and 9B.

Myths at the core of current CTG interpretation

FHR decelerations are center-stage [2,11,12,23]. Figures 6, 7, 8, 9A and 9B lead to the conclusion that there are myths or post-truth concepts at the heart of CTG interpretation as follows [23]. Myth 1: Benign early decelerations (commonly due to head-compression) should be 'gradual' and are very rare. Myth 2: Majority of 'rapid' decelerations should be called 'variable' because they indicate cord-compression. Myth 3: The small accelerations (shoulders) signify cord-compression decelerations and their absence is clinically significant. These are not benign myths. The resultant unscientific 'pattern-recognition' enforced on the grass-root clinicians leads to odds stacked against them and increased potential of harm to the babies. They land up paying the price for these myths [9,23].

Strengths and limitations of this study

This study tests important fundamental principles rather than just demonstrating interobserver differences in CTG interpretation, unlike most other studies. The number of participants in this study is not large but big enough for statistically significant conclusions could be drawn, hence this does not seem to be a limitation. In fact, the differences are so stark that the conventional statistical analysis is an added extra. Most clinicians follow national guidelines [3] in the UK and the participants came from five different National Health Service Hospitals, hence the results would be generalizable. Moreover, the main strength of the study comes from the clinical studies quoted and the critical analysis of the interpretation of the CTG illustrations by expert group and study participants in a case with chronological CTG changes and eventual very poor outcome [5].

Conclusions

This study proves that abandonment of FHR categorization into early/late/ variable seems a dangerous doctrine and a flawed ideology not supported by pathophysiology. Theory-induced blindness can be a weakness of intelligent mind. This flawed doctrine may have arisen from frustration and futility of unscientific focus on rapid vs gradual shape of decelerations but that is no excuse. It is very important to continue categorizing FHR decelerations into different meaningful patterns based on timing rather than shape (Figures 9A and 9B) [23]. Categorizing decelerations based on 'rapid vs gradual shape' is unphysiological and amounts to going down the wrong road eventually leading to a dangerous destination as reached by the expert group [5].

Declaration of interest

The author has no conflict of interest or funding to declare. The views expressed are author's opinion only and do not necessarily reflect practice or teaching.

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