
**A 50 YEAR OLD MYTH: THE RELATIONSHIP OF INTRA-PARTUM CARDIO -
TOCOGRAPHIC MONITORING TO CEREBRAL PALSY AT CLINICAL AND
MEDICO-LEGAL LEVEL**

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Abstract: The birth of a child is normally surrounded with much joyful anticipation. The human psyche wants perfection in progeny. Anything short of this elicits a vast range of reactions, often may be confirmed and lead to crippling damages, possibly, at times in conjunction with separate medically punitive actions by the local medical council. Once a few test cases – and most Court cases fail – do result in substantial financial awards, the precedent is likely to set the ball rolling. Some members of the legal profession are posed to take up the gauntlet, be the plea, justifiable or not proportionate to the outcome. In the presence of a situation which is suspicious or suggestive of Cerebral Palsy (diagnosis is done within about 12 months from birth) benumbs and throws the unfortunate parents into an unexpected maelstrom of terrifying emotions and worries. The zenith of happiness cruelly metamorphoses into the nadir of despair. Cerebral Palsy (CP) is the commonest disorder of movement in children¹, and, generally speaking (with much local variation) it occurs in about 2.1 per 1,000 live births². It would be grossly abnormal for such parents, not to ask questions. These questions, increasingly being asked in a Court of Law where obstetric liability.

The purpose of this paper is to show a firm affirmation that the correct instruments of science be recognised, both clinically and medico-legally in cases of CP. It is an attack on those aspects of the juridical procedure, during which scientific facts fly in the face of contemporary science.

Keywords: cardio-tocographic monitoring, cerebral palsy, obstetricians, birth, babies.

INTRODUCTION

It is within these parameters that this paper examines certain aspects of Electronic Fetal Monitoring in labour the form of Intra- Partum Cardio-Tocographic (I-P CTG) and Cerebral Palsy. The relationship between the two has a well defined beginning which is scientifically challengeable. What is called here as the Great Myth deals with a misconceived I-P CTG – CP and born in the USA of the 1960's and therefrom disseminated world wide. It has caused great and irreparable damage and injustice to numerous individuals, be they patients or doctors, to national health schemes and to science especially obstetrics.

It is only right for parents, to query whether their child's CP was preventable and whether lack of the expected standard of medical practice was responsible. And it is not unexpected that, at times, they turn to legal counsel. Parents initially speak out of pain and if, later they proceed to litigation and maybe even Court action, it is often both out of a desire to punish³ presumed medical negligence as well as for financial compensation. Be that as it may, it is their sacrosanct right to do so. It is also a sacrosanct duty, for Court to adjudicate using the right contemporary instruments. And the key word here is contemporary.

THE GREAT MYTH IS BORN

In the USA of the 1960's there occurred an unplanned synchronicity of events which seemed to shed light on CP causation and prevention. These events would also produce a legally assumed unshakeable scientific artefact in the form of an electro-thermally burnt tracing (CTG strip) which could be conveniently studied and presented as hard evidence in a Court of law.

The USA of the 60's is mostly known for iconic memories such as Elvis Presley, Flower Power, the anti-Vietnam demonstrations and a myriad, other dated phenomena. Few now know that there was also an ever-increasing USA public awareness and interest in the suffering of Cerebral Palsy. This awareness had been growing since the 1940's, when two philanthropic and personally involved couples, Leonard and Isabelle Goldenson and Jack and Ethel Hausman, had striven hard to publicise the plight of children with CP. In 1949, they formed the precursor group of

the eventually powerful United Cerebral Palsy Association (UCP) which would also take strong root in Canada and Australia. Awareness of the condition flowered in the 1950's and it peaked with the publication of the 1952 best-seller book "Karen", a true story, of the author Marie Killilea's daughter's agonies. In the 1960's the USA officially recognised the rights of disabled individuals by legislation and established individual care with the 1963 Community Mental Health Act enabling community-based care in addition and as an alternative to institutionalised care. Such was the highly expectant and sympathetic public atmosphere of the 60's to the condition of CP.

Documented since the 5th century BC by Hippocrates⁴, CP was extensively revisited in the mid-19th century by William John Little(1810 – 1894)⁵. Both Little and Sigmund Freud (1856 – 1939) considered birth asphyxia as a major cause but Freud also hypothesised that abnormal fetal development might also be causative, long before medical science considered it. In the USA of the 1960's a number of circumstances would come to confirm Little's original causation theory that CP, in its majority was due to oxygen deprivation during labour⁶. What were the facts leading to the resurrection of this 19th century myth?

In 1953, Virginia Apgar, a paediatric anaesthesiologist, appalled at the poor resuscitation of neonates, designed a score⁷, both to assess, as well as to resuscitate, the new-born. Five years down the line, in 1958, James et al⁸, proposed active ventilation of infants suffering from severe metabolic acidosis. This, in itself, was excellent work, based on correct observation, interpretation and rectification of daily clinical problems. Routine oxygen administration to all new-borns followed.

However, there also sprang the belief that just as oxygen administration was helpful to the new-born, as if by corollary, oxygen deprivation at birth or in labour, led to brain damage. Animal experiments in the late 1970's seemed to confirm the long suspected causal link between perinatal asphyxia and brain damage⁹. There is, of course, hardly any doubt in the rational mind that depriving oxygen to the fetal brain may lead to brain damage. This is a far cry from stating that the majority of cases of CP are due to intra-partum or peri-partum oxygen deprivation. Yet this was the wrongly drawn conclusion drawn in the USA of the 1960's. One of the twin pillars of the Great Myth was established while the second one was being built almost *pari-passu*.

Obstetric concerns at the time had been centred on the scientific need to detect intra-partum hypoxia, a topic on which some excellent minds had been ardently engaged. Now there was the added premium, that prevent such hypoxia and acidemia would essentially eliminate CP. Many people had been researching the subject of intra-partum hypoxia and its electronic detection including Alan Bradfield, Orvan Hess and Edward Hon. It was they who pioneered the invention of electronic fetal monitoring in 1958, while the refined and clinical version of CTG was the result of Konrad Hammacher working in conjunction with the firm Hewlett Packard. Much modifications followed and follow still. Great hopes were entertained that CP incidence would be eliminated or grossly dented.

Practical exponents of medical law were also watching. Medical science was actually providing them with a CTG strip CTG as potential evidence of medical malpractice and negligence in labour. It would be unchallengeable evidence of timed action, inaction or late action by an obstetrician to intervene in labour. Virtually overnight, commencing in the USA of the 1960's, CTG was medico-legally grasped, put into action and presented at Court, almost as an intra-partum standard of care of the obstetrician¹⁰.

Unfortunately, it would not be long before results showed that incidence of CP was essentially unaffected by the use of CTG. Publications such as Scott's¹¹ in which he stated that "time and time again, it has been shown that very few cases of CP can be explained on the basis of birth asphyxia" were appearing as early as 1976. Paneth¹², Nelson and Leviton¹³ and others stressed the same facts which challenged the by now established medical and legal thinking. It was a hard pill to swallow, but the fact was and is that the positive predictive value of a non-reassuring pattern to predict Cerebral Palsy among singleton new-borns with birth weights of 2,500 g or more, is 0.14%, meaning that out of 1,000 fetuses with a non-reassuring FHR pattern, only one or two will develop cerebral palsy¹⁴. The 21st century, would witness more damning evidence, such as Macones¹⁵ stressing that the false positive rate of External Fetal Monitoring for predicting cerebral palsy > 99%.

Intra-partum CTG did not and does not, in any substantial way predict Cerebral Palsy. Intra-partum CTG monitoring does have a much-guarded role to play in detecting intra-partum hypoxia as a screening test but, since Cerebral Palsy is not, in its majority, as originally surmised in the 1960's, due to intra-partum hypoxia, it cannot be prevented by acting on an abnormal tracing by immediate delivery of the child.

Although science was demolishing the presumed veracity of the Great Myth, much harm was perpetrated by it. Some of these snowballing harmful effects are shown diagrammatically in Figure 1. The use of CTG in CP Court cases resulted in a 10% rise by 1985 compared to the law pre-1970's litigation cases, most of the increase being related to multi-million CP cases.

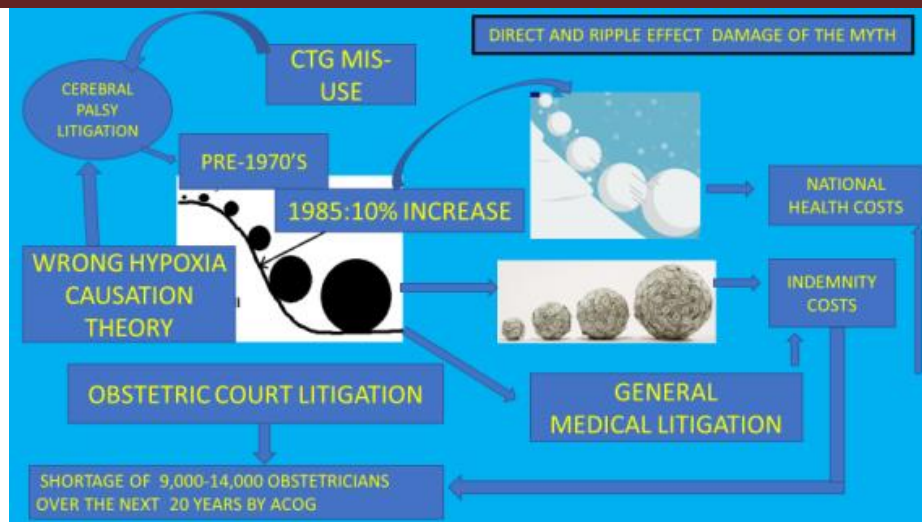


Figure1

While obstetric science was taking stock of its great delusion, Court practice, was far less interested in the emerging facts. As a result of this increase on obstetric litigation, all other disciplines followed suit and furthermore, a similar pattern of activity commenced across the Atlantic. The UK would witness the beginning and ever increasing tide of litigation with subsequent litigation costs.

Giving a safe margin, and by-passing CTG-CP centred Court cases in the 1970 -2000 period, and looking at 21st century Cerebral Palsy jurisprudence, we still find a great preponderance of intra-partum CTG tracings being used as sole and major parameters of assessment of obstetric performance. This is clear even if we refer to a few cases from the United Kingdom Courts, such as *Nation v King's Healthcare NHS Trust* (High Court, 3/11/03 – Treacy J); *The Baglio v. St. John's Queens Hosp.*, 303 A.D.2d 341 (2d Dept. 2003), Appellate Court, *Bruce v Kaye* [2004] NSWSC 277, *Gossland v East of England Strategic Health Authority* [2008] EWHC 2175, *L v West Midlands Strategic Health Authority*, [2009] All ER (D) 259, *Brodie McCoy v East Midlands Strategic Health Authority* [2011] EWHC 38 (QB), *LT Pursuer against Lothian NHS Health Board Defender* 2018 Scot (D) 28/5.

This does not necessarily imply wrong judgements, for jurisprudence is beyond the scope of this paper. However, there is a clear dichotomy of evolution of evaluation of CTG-CP evaluation, between the scientific and legal thinking. Whereas science over the last 50 years has downgraded the importance of CTG in CP causation, CTG argumentation still holds sway in many Court cases. In *L v West Midlands Strategic Health Authority*, [2009] All ER (D) 259, we find that the causation of the Cerebral Palsy seemed to hinge on a period of 6 minutes of non-action on a worrying CTG:“..... A reasonably competent obstetrician at 21.43 having reached her decision to intervene would have been outside the range of acceptable practice if in the circumstances of the instant case, she had taken the time represented by more than one contraction (more than two minutes)..... with the result that the claimant would have been delivered at 21.49 and resuscitated at 21.50; some six minutes sooner than was in fact the case.”

One must look at such evaluation with much discernment. A period of 6 minutes of non-action in the presence of a worrying CTG may constitute grounds for one type of malpractice. But what one is considering here is such presumed malpractice and the causation of CP. There may also be other aspects of malpractice if one ignores a seriously disturbed CTG for 6 minutes and they may be important in evaluating a case of CP. However, CTG management seems to hold not only centre stage but all of it.

In *Brodie McCoy v East Midlands Strategic Health Authority* [2011] EWHC 38 (QB), we find a circuitous and confusing discussion and evaluation of CTG monitoring interpretation which leaves one perturbed at ending up more confused than illuminated:... reference was made to the 1987 FIGO Guidelines for interpreting CTG traces. Mr Porter pointed out that there was an apparent internal inconsistency in the FIGO classification of decelerations in antepartum CTGs, as these state that the “absence of decelerations except for sporadic, mild decelerations of very short duration” is consistent with a normal fetal heart pattern; but “sporadic decelerations of any type unless severe” are part of the definition of “suspicious” fetal heart patterns. Thus, in cases such as this, where decelerations are difficult to identify, it is not obvious whether a CTG should be classified as normal or “suspicious”.

HOW SCIENTIFICALLY REASSURING IS COURT INTERPRETATION OF A CONTENTIOUS SUBJECT LIKE CTG, IN CEREBRAL PALSY LITIGATION?

Apart from the fact that the CTG-CP myth has been disproved as the majority of CP cases are not due to peri-partum hypoxia, CTG itself is a highly contentious parameter in the detection of intra-partum hypoxia. Having said that, it is important to re-iterate the crucial importance of intra-partum CTG monitoring as a screening test for intra-partum hypoxia in high risk cases. It is far from ideal, but it is what science has given us for daily clinical use, and one is not justified in disparage it by stating that all CTG monitors should be cast out of the window and calling it “the leading hoax of the twentieth century”¹⁶ although he is correct when he states that EFM spawned a birth injury litigation crisis centered on the myth that it predicts cerebral palsy (CP)¹⁷.

Yet, CTG interpretation does leave much to be desired. And there is much evidence that CTG centred Court litigation is often oblivious of these shortcomings. In *The Baglio v. St. John’s Queens Hosp.*, 303 A.D.2d 341 (2d Dept. 2003), Appellate Court, the discerning mind detects another by-way of worry through short-circuiting of the abnormal CTG as signifying intra-partum hypoxia.

“The fetal monitoring strips would give fairly conclusive evidence as to the presence or absence of fetal distress”. Here not only do we find spill-over from the Myth but the over-simplification of intra-partum CTG, in that CTG abnormalities are considered as “fairly conclusive evidence” of fetal distress, when a good 60% of abnormal CTG tracings are known not to be associated with intra-partum hypoxia and acidaemia.

There is clear and repeated evidence of the Court not evaluating the inherent weaknesses¹⁸, of I-P TG tracings when these are presented at Court. These include, but are not limited to, features such as Low sensitivity, High specificity, High Inter-observer error, High intra-observer error, Multiple, varied and debatable classifications of CTG abnormalities.

Furthermore, it is disconcerting, to find Court CTG-CP evaluation in Cerebral Palsy litigation employing incorrect terminology or out-dated nomenclature. In *Bruce v Kaye* [2004] NSWSC 277, we find:

“He arrived at 11.35pm. Inter alia, he found Ms Chevelle six centimetres dilated and read the FSE trace as type 2 decelerations with variable dips”. This was in 2004 – an odd 40 years after Roberto Caldeyrio-Barcia had discarded his own classification involving the use of ‘type 1’ and ‘type 2’ ‘dips’. In this particular case we find an unhealthy admixture of terminology involving the extinct ‘dips’ and the modern ‘decelerations.’ To cognoscenti of the subject this reveals a worrying lack of basic contemporary interpretative knowledge. Furthermore, to speak of type 2 decelerations with variable dips, unfortunately, does not make much sense. If anything, one may speak of variable decelerations with a late component.

THE EXISTENCE AND THE COST OF OBSTETRIC MALPRACTICE AND ITS LIABILITY

Since CP in its majority is not due to intra-partum hypoxia, most cases of CP do not involve intra- or peri-partum obstetric mismanagement. This statement must be qualified by two points: First - There may still be obstetric liability of CP not related to hypoxia-oriented mismanagement. This may even involve ante-natal care. Second - Some cases of CP are still the result of hypoxia-oriented labour mismanagement. This is estimated at about 1 in 5 cases or 20%. See fig. And, although 20% of a condition affecting about 2.1% per 1000 live births may not seem much, we must recall that compensation per case runs into big millions of dollars, pounds sterling, etc. Hence the statistics in Fig.1 where in 2016/17 United Kingdom NHS Obstetric liability claims were shown to be £2.185 billion. This was mostly Cerebral Palsy pay out related.

In fact, while demolishing the Myth, we must not sweep aside such malpractice which contributes to an estimated 20% of Cerebral Palsy cases. Such malpractice embraces a wide spectrum of obstetric management, which contrary to 1960’s thinking, should also embrace antenatal management and is certainly not limited to intra-partum mismanagement which itself encompasses much more than simply asphyxia-oriented negligence, for by modern medico-legal thinking, well post-1960’s, obstetric liability in labour has a wide spectrum and is not just limited to peri-partum hypoxia.

Hence, labour mismanagement does cause some cases of CP and the cost of such malpractice is staggering. These costs fall into perspective when we remember that we speak of a disabling and life-long condition with multiple and numerous effects on the individual, the parents, the family and their style and pattern of living. Specially modified vehicles, home adjustments and sometimes even new houses may be involved. Quantum claims may include, but be limited to: General, psychological and moral damages; Personal/ home care; Loss of earnings of the individual as projected from his estimated life span; Loss of earnings of parent/parents incurred by time lost from work, etc.; Travel expenses; Medical Care/equipment; House/car modifications; Special education; Rehabilitation.

These expenses may be worked along set systems which are updated from time to time. For example in the UK, recent increase in liability damages have been effected. A child with Cerebral Palsy currently awarded £12 million, will soon be receiving about £22,560,000.

In the USA, liability damages are hardly likely to be less than 14,500,000 (as in *Welker v. Penn Highlands* 2017 (inappropriate use of syntocinon) and *Stewart v Metro Health* 2014 (delayed timing of C-Section), but, awards up to \$130,000 are not unknown.

These crippling amounts of money, awarded by the Courts of whichever country, imply the undoubted existence of medical malpractice as causative of some cases of Cerebral palsy.

A PARADIGM SHIFT IN MEDICAL AND MEDICO-LEGAL CONCEPTS OF CP CAUSATION

The acute and practical need felt for a major shift in conceptualising the causation of Cerebral Palsy would come from the USA, the home of the original Great Myth. In 2003, an American College of Obstetricians and Gynaecologists (ACOG) Task Force in conjunction with the American Academy of Paediatrics (AAP) produced a crucially important document entitled *Neonatal encephalopathy and cerebral palsy: Defining the pathogenesis and pathophysiology*¹⁹. This was further updated in 2014 by the ACOG Executive Summary and entitled *Neonatal encephalopathy and neurologic outcome*.

Briefly, these documents, set a new road map, formally demolishing the I-P CTG Myth, without actually doing so. Basically, they stressed the fact that Hypoxic/Ischaemic Encephalopathy, which is the inevitably pathology antedating and underlying Cerebral Palsy, must be defined along certain precise criteria. If these criteria are not present, technically, no case exists as to the presence of Cerebral Palsy resulting from an acute peri-partum partum episode of hypoxia which is termed the sentinel episode or event.

For this sentinel episode of hypoxia to be accepted, there must be all four of these Essential Criteria: Metabolic acidosis (Ph<7 and Base Deficit ≥ 12 mmol/L), Early onset of severe/moderate encephalopathy in infants born at ≥34 weeks, Spastic quadriplegia/Dyskinetic Palsy, Exclusion of other identifiable causes e.g. trauma, infectious conditions or genetic disorders.

The document does not speak of liability or jurisprudence for this is the Court province. It supplies facts of science and not their legal implications. Hence, for example, it does not rule out obstetric liability of CP but defines the type of CP and the sentinel episode which could have caused it. There can be the most clearly defined situation of hypoxia induced spastic quadriplegia with a clear causation episode, due for example to Abruption Placentae and liability, still deemed not to be present. On the other hand there may be no obvious hypoxia but liability still incurred from, say, mismanaged antenatal chorio-amnionitis. Hence, care is needed in interpretation of the medico-legal implications.

One also notes that hypoxia induced Cerebral Palsy must be of the spastic quadriplegia or dyskinetic form. There have been Court cases claiming hypoxia related liability and involving hemiplegia, spastic diplegia, ataxia, intellectual disability, autism, and learning disorder in a child without spasticity, all of which are not considered linked to intrapartum hypoxia.

The list of secondary criteria are more relevant with regard to the timing of the sentinel event of peri-partum hypoxia. Notice (underlined in the figure above) that CTG is placed in this second criteria, which group is not even a requirement for diagnosis. One of the main pillars of the old Great Myth has not even made it to the first rank of required criteria for the basic pathology of hypoxia-induced CP!

One should also remember that severe/moderate neonatal Cerebral Palsy usually develops within 24 hours of birth encephalopathy in infants born at 34 or over. In such cases, early imaging e.g. by MRI is crucial as the post-hypoxic oedema following an acute insult, may appear within 6-12 hrs but will also disappear clears within 4 days. Regarding the Apgar scores, one should note that it is the 5 minute cut-off point which is required advised by the guide-lines. Furthermore, apgar scoring may be liable to an element of subjectivity.

Figure 2 is a brief schematic table comparing the effects of the 1960's thinking with that of post-2003 scientific thinking on CP jurisprudence.

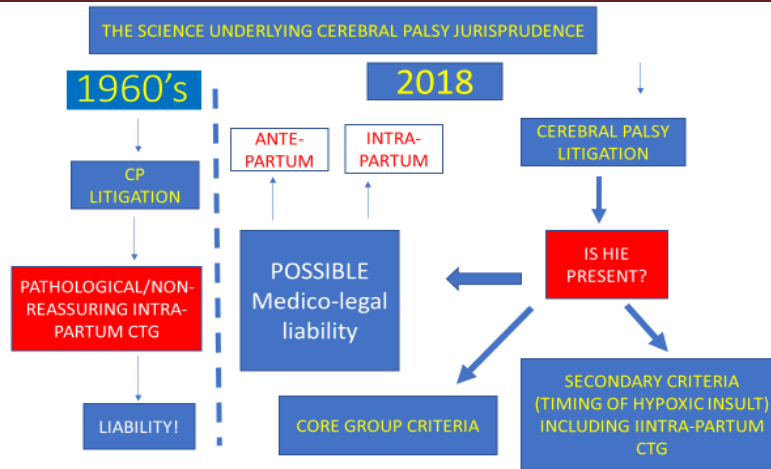


Figure 2

The document by the Executive Summary of 2014 threw light on a number of other aspects, not included in the first document. This includes the need to consider the whole of obstetric care, and not just labour management in searching for possible causation of Cerebral Palsy. It also stresses the need to search for the root causes of brain injury in new-borns and the broad evaluation of all potential contributory causes. This moves away from the simple focus of the 2003 report which focused on the potential peri-partum hypoxic ischaemic event as the cause of neonatal encephalopathy.

As more light emerges on causation of Cerebral Palsy, it becomes clear that aetiology is far more complex than surmised in the 1960's. This is crucial in prevention, management, as well as in the retrospective analysis of obstetric care and liability.

NEW IMPLICATIONS OF THE POST-MYTH CONTEMPORARY SCIENTIFIC FACTS

The elimination of the I-P CTG-CP myth in the scientific world, albeit not in medico-legal confrontation, created a vacuum which has, slowly but surely, been replenished by Hypoxic Ischaemic Encephalopathy (HIE) oriented evaluation. Figure 3 compares and differentiates between the completely different roles of CTG in medico-legal argumentation in the 1960's and post-2003. In the former and, unfortunately often still, I-P CTG retains centre stage and often acts alone. On the other hand, post-2003 and certainly in 2018, centre stage should comprise HIE with I-P CTG attaining a relatively minor role in the second rank criteria related to HIE. Should, but often is not. One is reminded here of liability sought for hypoxia-induced CP, allegedly resulting from obstetric intra-partum negligence.

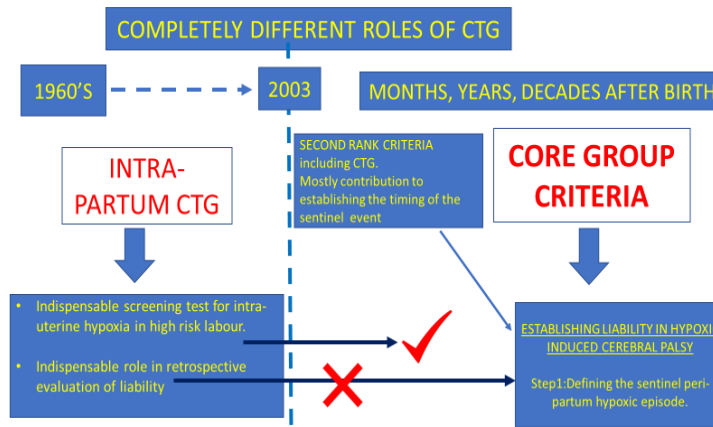


Figure 3

The new medico-legal argumentation would, as a preliminary to any Court action, need to: 1 Confirm the presence of HIE; 2 Identify and define the sentinel episode causing it, using the core group criteria, etc.; 3 Ideally time the episode using at least 3 of the second rank criteria.

It is then, that obstetric liability may be evaluated as responsible or not with regard to the sentinel episode.

Cerebral Palsy but must pass through the intervening stage of causation of Hypoxic Ischaemic Encephalopathy, which can be confirmed using the ACOG -AAP main criteria, as described while the secondary criteria, (which include I-P CTG) will also assist with establishing the timing of the event. Timing is extremely important, firstly with regard to treatment and establishing prognosis and secondarily in guiding liability and its degree.

Intra-partum CTG remains the essentially only practical investigation available to screen for intra-partum hypoxia and, if confirmed, allow us to act pro-actively to avoid hypoxic induced damage, including that leading to HIE. This is acceptable and unchallengeable. However, this limited but indispensable relevance of CTG in detecting intra-partum hypoxia does not equate, with the same relevance in the retrospective assessment of causation of hypoxia associated Cerebral Palsy.

Having established the flow-chart management of figure 4, one must also consider the presence or absence of aggravating factors spanning both the intra-partum as well as retracing management in the ante-natal period. For example the concept of the “two-hit theory” where a pre-existing hostile intra-uterine environment first existed during the pregnancy and then a second intra-partum or neonatal event is super-imposed on it resulting in the final over-all damage. This underlies the importance of ante-natal management both therapeutically and pre-emptively but also retrospectively and medico-legally. A likely scenario would be one associated with growth restriction where a sub-optimal utero-placental environment is then subjected to the periodic contraction induced placental blood flow drop of labour. Taking intervillous blood space flow cessation as the standard 30 secs in a 45 secs contraction, and 45 secs in a 60 sec contraction, if placental blood flow in such a case is 75% of normal (generously speaking) at the start of labour, it may easily drop to 50% and even 40% of normal during contractions. At such levels, repeated contractions may easily effect serious intra-partum hypoxia leading to organ damage and even death in utero.

The ACOG 2014 report also analyses the modern treatment of neonatal encephalopathy and emphasizes the necessity of medical expertise in the caring paediatricians, which subjects are, to some extent²⁹, beyond the scope of this paper. Even so, the role of adequate treatment and the expertise of the attending paediatrician, which along with the particular Unit’s ability to deliver, may determine the final outcome, which in itself, may be crucial in allotting degree of liability.

IN CP JURISPRUDENCE, ARE THE COURTS LISTENING TO SCIENCE?

We can add here the use of I-P CTG as a distracting or even misdirecting tool, used consciously or not. An example can be quoted in *Gossland v East of England Strategic Health Authority* [2008] EWHC 2175, involving a case of Cerebral Palsy, in the presence of a history of: A very traumatic Kiellands rotational forceps; 5.22kg baby at birth; Shoulder dystocia with a fractured clavicle.

In the face of these most worrying of facts, we find the I-P CTG still given surprising prominence: “.. the cardiocotograph trace was not such as to lead an obstetrician of ordinary competence to take the view that Omar had a ‘complicated tachycardia’ such as to make it imprudent to administer oxytocin or to make it mandatory to take a blood sample from Omar’s scalp. The cardiocotograph trace showed a tachycardia aptly described by Mr MacKenzie as a ‘moderate’ one which could not properly be characterised as a complicated tachycardia. In Dr Emmerson’s words it was ‘somewhat abnormal’ but nevertheless ‘a common occurrence’”

So, we have the nightmare of an obstetric stramash, but all is well with the obstetric performance because the I-P CTG showed a tachycardia which was complicated but not properly complicated though somewhat abnormal though, again, it was a common occurrence! Does one need add more?

Let just and fair litigation proceed, if and when indicated, for obstetricians are not above the law. Yet, let us keep in mind that rampant litigation begets the vicious spiral of even more rampant litigation. Compensation is an alluring treat even for those with no valid cases, especially if fanned on by ‘ambulance chasing’ lawyers. In that case the harm done is not just to the doctor, to the profession but to the national budgets but to society at large. This money – and unnecessary Court action is extremely expensive – could be going where it is truly required. One must also remember what is often referred to as the second victim in this story – and that is the obstetrician himself/herself. Truly guilty of negligence or not, the obstetrician undergoes a personal purgatory, which drags within it, his practice and his family. He undergoes stages of guilt, fear, anxiety, anger, insomnia and depression which may reach suicidal levels. Little wonder that areas particularly associated with rampant obstetric litigation tend to become obstetrician depleted. Court cases may drag for years and even if a case is won by the defendant doctor – and most

are – much damage is likely to have been inflicted on his or her persona, health, work practice and family. Indeed, a victory at Court may be a pyrrhic one.

The question, of whether the Courts are listening to science, is, thus a most valid one. The relevant scientific milestones established in the USA in the 1960's and which gave birth to the Great Myth have undergone such revision that they are beyond recognition. Few discern the fact the Great Myth may be in a museum of obstetrics but still lives and breathes in many Courts. The material from the UK, presented here is but a fraction of universal CP Court-room thinking, across both sides of the Atlantic and should be guarded against in Europe where the wheels of CP litigation are slowly grinding in motion.

The truth is that the science of causation of CP is still evolving. We do know that the simple CTG- CP oriented medico-legal thinking should have no place in modern Court. Yet, it is not easy for a litigation lawyer to let the convenient CTG strip slip out of his hands. But a deep -rooted change in mentality is now an inescapable duty. Some lawyers even view the 2003/2014 ACOG-AAP advisory documents as a way for doctors to “slip out of their obligations of CP damage”. These people are clearly missing the great opportunities for justice to reign for the proposed changes even recommend the importance of scrutinising aspects of obstetric responsibility such as antenatal care which traditional CTG-CP thinking had, in its myopic blindness, completely ignored.

Over-all it does not seem as of there is synchronicity of any extent between relevant scientific evolution and Court practice. Admittedly the wheels of law grind much slower than those of science.

Let us look at a case from the UK High Courts of 2018. Although, this exercise does not reflect that wrong conclusions were effected by the venerable Court, it is still instructive. In the case LT Pursuer against Lothian NHS Health Board Defender 65,782Scot (D) 28/5. Lady Wolffe, the official Court transcript of 65,782 words has 622 references to the word CTG. Besides the use of fetal pH and Apgar score we find no other reference Criteria parameters. The Apgar score referred to was at 1 minute post-birth and not as the ACOG – AAP recommend at 5 minutes.

Yet, in the case AW Pursuer against Greater Glasgow Health Board Defenders CSOH 99 (2015), an official Court transcript of 29,712 words contains only 40 references to the word CTG. This is relatively about 277 times less than in LT Pursuer. The Apgar score quoted was at 5 minutes along ACOG-AAP recommendations. Evidence of discussion of topics of neurological imaging, hypoxic ischaemic encephalopathy and non- focal cerebral abnormalities are clear. Moreover, the Court discussion involved 13 experts from the fields of obstetrics, midwifery, paediatrics, neonatology, paediatric neurology, paediatric neuro-radiology, and neuro-radiology.

A case such as AW Pursuer against Greater Glasgow Health Board Defenders CSOH 99 (2015), is a true light-bearer. It clearly exemplifies a working application of the principles, advocated by this paper. It is clear that this case rejects the Great Myth and embraces the latest scientific principles to help adjudicate what, in reality is an extremely complex and difficult matter and still evolving. This is the right and correct attitude to let true justice speak its voice.

CONCLUSION

This paper has quoted examples of Court use of I-P CTG interpretation employing incorrect nomenclature and incorrect classification of abnormalities. The ubiquitous use of I-P CTG as a monopoly of obstetric performance and management has been criticised as a dangerous remnant of the Great Myth.

This paper is a firm affirmation that the correct instruments of science be recognised, both clinically and medico-legally in cases of CP. It is an attack on those aspects of the juridical procedure, during which scientific facts fly in the face of contemporary science.

REFERENCES

- Apgar V. (1953). A proposal for a new method of evaluation of the newborn infant. *Curr Res Anesth Analg*. 32: 260-267.
- Buttigieg G G, Buttigieg G. (2014).Medico-Legal Litigation: The clinical contractual nature of the Obstetric Anaesthetist -Patient relationship. *Malta Medical Journal*; (26)1.
- Buttigieg GG. (2017). Lessons from the Great Medico-Legal Chapter of Cerebral Palsy. *J Neurol Disord* 5: 335.
- Buttigieg GG (2017). Lessons from the Great Medico-Legal Chapter of Cerebral Palsy. *J Neurol Disord* 5: 335.
- James LS, Weisbrot IM, Prince CE, Holaday DA, Apgar V. (1958). The acid-base status of human infants in relation to birth asphyxia and the onset of respiration. *J Pediatr*. 52: 379-394.
- Buttigieg GG. (2016). The shifting sands of medico-legal intra-partum Ctg (I-P Ctg) monitoring. *Medico-Legal Journal*. Vol. 84(1) 42–45

-
- Buttigieg G G. (2016). Obstetric/Paediatric Interaction in Brain Damage Litigation: Learning from the Courts. *Pediatr Neurol Med.* 1 (3):115.
- Macones George A. et al., (2008). National Institute of Child Health and Human Development Workshop Report on Electronic Fetal Monitoring Update on Definitions, Interpretation, and Research Guidelines
- MacLennan, A. (1999). A template for defining a causal relation between acute intrapartum events and cerebral palsy. *BMJ.* 319:1054 –105
- National Institutes of Health (2014)
- Nelson KB, Leviton A. (1991). How much of neonatal encephalopathy is due to birth asphyxia? *Am J Dis Child.* 145:1325–1331.
- Nelson KB et al. (1996). Uncertain value of electronic fetal monitoring in predicting cerebral palsy. *N Engl J Med.* 334:613–8.
- Obladen M. (2011). Lame from Birth: Early Concepts of Cerebral Palsy. *Journal of Child Neurology.* 26: 248-256.
- Paneth N. (1986). Birth and the origins of cerebral palsy. *N Engl J Med.* 315:124–126.
- Panteliadis, C; Panteliadis, P; Vassilyadi, F. (2013). Hallmarks in the history of cerebral palsy: from antiquity to mid-20th century. *Brain & Development.* 35 (4): 285–92.
- Sartwelle. P (2009). Defending a neurologic Birth Injury, Asphyxia Neonatorum Redux’ *The Journal of Legal Medicine,* 2: 181-247.
- Skoui, M; Coutinho, F; Dykeman, J; Jetté, N; Pringsheim. (2013). An update on the prevalence of cerebral palsy: a systematic review and meta-analysis, *Developmental Medicine & Child Neurology.* 55 (6): 509–19.
- Scott H. (1976). Outcome of very severe birth asphyxia. *Arch Dis Child.* 51:712–716.