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Neonatal Hypoxic Ischaemic Encephalopathy: Demolishing the Cerebral Palsy Myth and Enlightening Court Litigation

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Abstract

The article sheds medico-legal light on jurisprudence based on the two original tenets of the great Cerebral Palsy myth, namely

1. The misconception that intra-partum hypoxia underlies most cases of neonatal Cerebral Palsy.

2. The role played by misapplied intra-partum cardio-tocography (I-P CTG) in "diagnosing" intra-uterine hypoxia.

Both concepts, originated by medicine, were eagerly embraced by the legal system seeking liability damages in Cerebral Palsy litigation. Although, these misconceptions have been largely rectified in medical practice, they still form the conscious or sub-conscious basis of much Court litigation, across both sides of the Atlantic. UK Court case law is referred to, in a number of instances, to illustrate various points referred to.

In the light of Hypoxic Ischaemic Encephalopathy (HIE) being the true pathological hallmark of hypoxia – induced Cerebral Palsy, the article highlights the medico-legal need, existing pre-2003, to formally accentuate the connection. In this context, reference is made to the ACOG's initiative in setting up the relevant workshops, the reports of which led to the 2003 – 2014 ACOG-AAP Criteria for diagnosing HIE. The milestone relevance of these criteria in illuminating Cerebral palsy litigation is evaluated, again with references to relevant UK Court cases.

Keywords: Court; Medico-legal; Cerebral palsy; Myth; Intra-partum hypoxia; I-P CTG; Apgar scores; ACOG core criteria

Mistaken Scientific Beginnings and Misled Litigation

Modern Cerebral Palsy science and Cerebral Palsy litigation, however, distinct, shared a common basis of origin. And the trail embarked upon, was blazed open by medical science. In its fervour to fight this cruel condition of the new-born, Medicine embarked on "scientific facts" which were later proved to be neither scientific nor facts.

However, by the time that these "facts" were seriously challenged, much medico-legal harm had been done, and the "myth" had been established. When, the light of truth slowly brought understanding to the medical world, the legal counterpart was loathe to part with the old ways. Let us review the first half of the medico-legal story of Cerebral Palsy – that part where Medicine and Law, seemed joined at the hip, even though the relationship was parasitic rather than commensal. This part of the story commences in the 1960's, the exciting decade which witnessed the first synthesis of biologically active DNA in test tube, the first laser photocoagulation of the detached retina and the role of insulin resistance in type II diabetes.

The basis of the initial 'together trip' rested on two pillars. The first pillar of the myth, that cerebral palsy was, *in its majority*, due to intra-partum hypoxia, had its nucleus formation in 1953, with the work of Virginia Apgar. No that Apgar was responsible. Appalled at the poor resuscitation of neonates, the good anaesthetist proposed a score (later the Apgar score) [1], both to assess, as well as to resuscitate, the newborn. Five years down the line, in 1958, James et al [2], proposed active ventilation of infants suffering from severe metabolic and respiratory acidosis. This, in itself, was excellent work, based on correct observation, interpretation and rectifcation of daily clinical problems.

Laudably enough, these works led to oxygen administration as part of the routine labour ward set-up. As if by corollary, there also sprang the belief that oxygen deprivation led to brain damage [3]. This naturally is a fact, but what is not a fact, is the extra-polation that cerebral palsy is, in its majority, the result of intra-partum hypoxia. Here, we have a situation, not unknown in life, where non- truths, ride triumphantly on related facts which are truths. New-borns may suffer from hypoxia. Hypoxia may cause brain damage, such as Cerebral Palsy. Cerebral Plasy may thus be due to intra-partum or neonatal hypoxia. All true. But, to state that Cerebral Palsy is due to intra-uterine or neonatal hypoxia, without further qualification is untrue in the great majority of cases. A dog is an animal. But, an animal is not necessarily a dog.

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But the belief that Cerebral Palsy's chief cause is hypoxia, had taken strong root by the early 1960's. The implication at medico-legal level was obvious. The only unsolved crux to the problem lay in proving the existence of birth hypoxia. Luckily for the legal system, obstetrics was, at that time, in the final throes of clinical application of a machine designed to detect intra-uterine hypoxia. This was the basis of the second pillar of the myth.

Electronic fetal monitoring in the form of cardio-tocographic (CTG) monitoring was essentially invented by Alan Bradfield, Orvan Hess and Edward Hon. The clinically usable machine was later developed by Konrad Hammacher, working in association with the firm Hewlett-Packard. By the 1960's, CTG monitoring was in use in most major obstetric units, not only in the USA, but also in the UK and other developed countries. CTG was considered the final trophy in the search.

The first myth was, by now, so well established, that Intra-Partum CTG monitoring (I-PCTG) was hailed as the great gift to mankind to diminish or eliminate Cerebral Palsy. Unfortunately facts would show that CTG monitoring is of no value at all in predicting Cerebral Palsy. This is not synonymous with stating that I-P CTG does not have a crucial role to play, especially in high risk labour. CTG is still in regular use and furthermore, has no viable substitute in the foreseeable future. Condemning it, would be a mistake. This paper purely condemns those aspects of the original expectations of CTG monitoring, as the "holy grail" vis-à-vis prediction and prevention of Cerebral Palsy. It was this aspect of I-P CTG monitoring which constituted the second pillar of the cerebral palsy myth.

This latter aspect of CTG monitoring was pounced upon by birth injury lawyers as the missing link to their Court argumentation pleading obstetric liability in cases of brain damage. The machine promised detection of intra-uterine hypoxia and hence through immediate action and delivery, the prevention of the aftermath of all hypoxic complications. By the then current reasoning, this included Cerebral Palsy. Medico-legally, reverse the psychology, and you have a yardstick, by which to measure an individual obstetrician's management when it comes to assessing liability in Cerebral palsy. Furthermore the monitor electronically generated a permanent strip, which could be reproduced in Court, as evidence no 1. As simple as that. Or so it seemed.

Up until this stage, Medicine and Law were, essentially moving along the same scientific lines, albeit, the information being churned out was being used for different end scopes. The clinical aim of the obstetrician was replaced by the proving of liability by the lawyer. Unfortunately, the outcome proved to be bitter fruit medically, although medico-legally the story was different. The medically expected dramatic decrease of Cerebral Palsy incidence never materialized with the use of I-P CTG monitoring, in spite of a marked increase in unwarranted labour interventions, resulting from CTG mis-interpretation and wrong weighting. Medico-legally, the statistics also changed. From 1985, obstetric litigation in the USA, mostly involving neurological sequelae, jumped from negligible to 10% and Sartwelle et al [4], contribute this to the Cerebral Palsy myth.

Ignored Red Lights

Medical science did have a number of red lights flashing well before the fallacy of birth hypoxia being the prime cause of Cerebral Palsy, finally hit home. By that time, untold damage had been done, both at individual level as well as to national obstetric practice and resultant national medical health budgets In her 1955 studies, Apgar herself had already showed "*no significant correlation between I.Q. [at 4 years] and oxygen content or saturation at any time during the first 3 hours of life* [5]. An association between a low Apgar score combined with neonatal encephalopathy and subsequent death, or a major neurological handicap, is well documented [6]. However, a low Apgar score, is, by itself, no evidence of intrapartum asphyxia, though it may be related to prematurity, congenital malformations, perinatal infections, or maternal sedation or anesthesia asphyxia [7].

Today we know that a severely depressed Apgar score, unaccompanied by seizures or feeding difficulties, is not associated with an increased risk of later neurological impairment. A massive amount of controlled scientific studies, have now displaced the myth of the simple statement, that intra-partum hypoxia leads to cerebral palsy And a similar amount of studies have shaken CTG monitoring, off its original unchallenged predictor status. In fact, a number of obstetric authorities like Quilligan [8], were voicing concerns about the significance of CTG monitoring, as early as 1975. In 1979, Haverkamp et al [9]., using a controlled trial involving 690 high-risk obstetric patients, reported no improvement in neonatal outcome with the use of CTG monitoring, but, on the other hand, an increased risk of caesarean section. In 1985, the Dublin randomized controlled trial of intrapartum fetal heart rate monitoring [10], comparing continuous electronic I-P CTG monitoring with intermittent auscultation, gave CTG a further knocking. In 1990, Shy-KK et al [11], confirmed that the use of continuous EFM, was not associated with a reduction in the incidence of cerebral palsy.

To some extent, a certain degree of confusion, is partly excusable. No one was saying that CTG monitoring was worthless. It was not and is not. The United Kingdom's Confidential Enquiries into Stillbirths and Deaths in Infancy (CESDI) in 1997, 1998 and 2000, stated categorically, that poor interpretation of the CTG was a major contributor to intrapartum stillbirths. The fine point to be discerned here, is that, although CTG monitoring does have an important intra-partum role, particularly in high risk labour, this role excludes Cerebral Palsy prediction. And extra-polating the detection of intra-uterine hypoxia into the Cerebral Palsy camp is not scientifically viable. Some, genuinely miss the difference, others obfuscate it, for ulterior motives.

The retention of the originally scientifically wrong Cerebral Palsy premises, is a phenomenon which still holds, even within a few medical quarters, including a few uninformed obstetricians. For these, it is still not registering that, CTG monitoring does not predict cerebral palsy [12]. And that no more than 10% of cerebral palsy in term, or near-term infants, suffering from cerebral palsy, have their causation *in intra-partum hypoxia or asphyxia* [13]. So much so, that, in 2016, WHO does not recommend continuous CTG monitoring in under-resourced settings [14].

Hence the persisting legal use of the Cerebral Palsy myth in seeking obstetric liability is hardly surprising. Some birth injury lawyers, do their utmost to propagate this great untruth, and for evidence one can easily go through an internet search of birth injury lawyer web-

sites. It is clear that many of these lawyers disseminate the concept that Cerberal Palsy equates with obstetric liability, until disproved otherwise. 'Otherwise' is often a long, painful and expensive Court trial. Incidentally, and for the record, one may also find, some fine and exemplary web-sites, which are meticulous in their correct and scientifically backed statements.

Obstetric Liability

The scope of this article is far from mitigating the search for genuine liability through the lowering of expected obstetric standards. There is absolutely no doubt, that Cerebral Palsy may result from legally liable, negligent, sub-standard obstetric management, where severe intra-uterine hypoxia may lead to cerebral damage. Incidentally, confirmed intra-uterine hypoxia may also carry no liability, as in a correctly managed case of intra-partum abruptio placentae. One such example is found in *Baynham v Royal Wolver hampton Hospitals* NHS Trust [15].

Obstetric liability in labour may involve other factors than mismanagement of hypoxia. It may, involve negligence in direct causation rather than ignoring or mismanagement of nature inflicting hypoxia on the unborn. It may for example, involve trauma at birth. One example comes from Fotedar v St George's *Healthcare NHS Trust* [16] where the obstetrician was found guilty of negligence in the performance of a ventouse delivery in the absence of full cervical dilatation and in the presence of cephalo-pelvic disproportion, with resultant traumatic brain haemorrhage and severe brain damage. At times, an admixture of elements, such as trauma and hypoxia are present. Thus, in *Parry v North West Surrey Health Authority* [17] we find a negligent forceps delivery, associated with both trauma and resultant hypoxia, the combination of which led to severe brain damage. The Court ruled on negligence, among other things, when the defendant attempted to:

Deliver foetus by forceps when it was too high in mother's pelvis. Negligence materially contributed to, if it did not solely cause, episode of bradycardia which in turn caused acute hypoxy-ischaemia. Midwife was negligent in omitting abdominal palpations and relying on her vaginal examinations of mother in labour.

It is crucial also to stress that in a case of Cerebral Palsy, obstetric liability may be incurred under a number of aspects of management and not even, necessarily limited to labour per se. Sub-standard obstetric management of the antenatal period involving an undiagnosed or mismanaged ante-natal chorio-amnionitis, and eventual cerebral palsy, may carry no less liability in Cerebral Palsy litigation than any labour mismanagement [18]. Thus, infective encephalopathy from an antenatal ascending infection, as discussed above, was one of the factors alleged as responsible for Cerebral Palsy of the new-born in Ingram (*a protected party by his mother and litigation friend Anita Jones*) *v Williams* [19].

In *Quinn v Midwestern Health Board* [20], Cerebral palsy liability was partly laid at mismanagement of antenatal growth restriction:

….it is submitted on behalf of the Plaintiff that her foetus was being progressively deprived of adequate fluid supplying, inter alia, blood and oxygen from some point possibly as early as the 20th week of gestation, and that this progressive starvation was the cause of damage to the foetus, itself progressive, which culminated in irreversible brain damage sometime during or after the 35th week of gestation.

Genuine Hypoxic Ischaemic Encephalopathy (HIE), the true, underlying pathology hallmark, of hypoxic induced Cerebral Palsy, may also be the result of recent peri-partum hypoxia, of an even longer standing origin [21]. The latter may open new windows of reflection on malpractice liability involving antenatal care. This, in fact, would be an example, of the advantage of Court, shifting its myopic obsessive attention to the birth process to a broader spectrum of enquiry comprising the antenatal management [22], and away from sole scrutiny of labour itself, particularly the CTG tracing.

Potential I-P CTG Misdirection in Cerberal Palsy Litigation

CTG monitoring may do a disservice to cerebral palsy litigation through a number of ways, in addition to that done by the discussed original misconceptions. Firstly, this may happen through retrospective misinterpretation and the use of wrong nomenclature, not uncommonly encountered in I-P CTG medico-legal confrontations [23]. Then, there are I-P CTG's well known intrinsic propensities for a high intra- and inter-observer error [24], lack of objectivity [25], high specificity and low sensitivity [26], which factors, may, alone, or in unision, make a mockery of Truth. Especially if CTG abnormalities are not backed up by confirmation through Fetal Blood Sampling [27], or other, emerging and more accurate methods, such as STAN. Where confirmation of hypoxia is omitted, one should remember, that even in the worst case scenario of CTG abnormalities, true fetal hypoxaemia and acidosis, can be confirmed in only 50–60% of cases [28]. And, although, this list is anything but exhaustive, one must also remember the "displacement" factor, where CTG argumentation attains primadonna status in Court, at the cost of much more valid and ignored arguments, considerations and implications [29].

Yet, many are the Court cases, where plaintiff argumentation essentially rests solely, on CTG analysis. Borrowing from the UK Court, for example, in *Coyle v Lanarkshire Health Board* [30], essentially most of the argumentation is centred around various aspects of CTG monitoring, and though the Court ruled for the plaintiff, it did so, not on the plea of the defedant's failure to perform Fetal Blood Sampling, but, rather, on midwifery breach in not seeking medical assistance. In *Tippett v Guy's & St Thomas' Hospital NHS Foundation Trust* [31], a case which was thrown out, once again, the plaintiff's argumentation mostly centres around various aspects of CTG monitoring. The point being made here, is not, whether CTG argumentation led to a ruling, for or against the defendant, but, the fact, that CTG monitoring was what furnished the core argumentation. The medico-legal world is still fixated on the old chestnut, forgetting, that in so doing it may be missing richer and juicier pickings.

Many champions have made the case against CTG-based miscarriage of justice in Cerebral Palsy Court cases. Sartwelle et al [32], are among the leading stalwarts, voicing much justifiable concerns. Although, abandoning CTG monitoring, as advised by Sartwelle et al., is not in this paper's recommendations, one must stress the depth and breadth of CTG misuse in Cerebral Palsy litigation ,over the last five or six decades.

Lawyer versus Doctor

The negative impact of CTG -oriented, Court cases, has also greatly contributed to the archetypal "doctor versus lawyer" attitude in Court confrontation. It is just and right for a doctor to answer to allegations for liability with a justified basis. But, to go through the calvary of a ruinous trial, using a defective yardstick, is morally bankrupt. The situation was excusable through ignorance, when the bent yardstick had the general blessings of limited science. It is no longer so, when, for decades, science has outrightly proved otherwise. Limiting the socio-economic damage of the plague of medico-legal confrontation should combine the efforts of both Medicine and Law by combating:

1. True medical malpractice.

2. A medico-legal system, which is not self-assessing and self-renewing with the progress of time and science.

3. Any secondary system, which is opportunistically hi-jacking, riding piggy-back and financially milking the situation [33].

Unfortunately, III is no small force to be reckoned with. It includes 'birth damage' lawyers and doctors, who are out to make a dime or two, from misrepresentation of facts. It does not include bona fide lawyers and doctors, who submit plea, or aid such pleas, seeking just recompense for those unfortunates, whose lives are ruined by genuine medical malpractice.

In a case alleging liability through mismanaged hypoxia, proving the presence of HIE should be the first burden of proof. In its absence, oxygen deprived brain damage is ruled out. The discovery of HIE as the specific encephalopathy in such cases provided a tremendous instrument for ascertaining truth in Court. Whether it is thus employed or not, is another matter. A formal and official declaration by a recognised body with clout was necessary.

College Beacon of Light

It is clear that a formal obstetric College, is ideally placed for such as task. It has the necessary respect, gravitas and authority. The American College of Obstetricians and Gynecologists (ACOG) took up the gauntlet, along with the American Academy of Pediatrics, in the form of a taskforce report entitled Neonatal Encephalopathy and Cerebral palsy: Defining the Pathogenesis [34], (2003) and which was updated in 2014 by a second report -*Neonatal Encephalopathy and Neurologic Outcome* [35]. No doubt, the future, after the assimilation of ever increasing studies, will see further updating. These reports are not in any way, medico-legally biased. They are scientific and clinical. Yet, truth is truth and fact is fact. However, the genie let out in the 1960's by other scientific "truths" and "facts" will not quietly and easily return to the bottle. Yet, it is the beginning.

Of the many critical points emanating from these reports, is the establishment of the ACOG and AAP (American Academy of Pediatrics) Criteria for establishing the presence of HIE. These criteria are divisible into two groups, the

Core group of four parameters which must be present for the establishment of the diagnosis of HIE, and a second group, comprising other parameters, and, which, though by themselves are not sufficiently specific to establish HIE, are still of great value, for example, in establishing the timing of the established cerebral damage.

It is interesting to note, that CTG monitoring falls within the second and not the core group. The end scope of CTG monitoring is the detection of hypoxia/acidosis. The core group requires established proof of hypoxia and acidosis, and not CTG argumentation of what may constitute hypoxia.

Although, detailed evaluation is beyond the scope of this article, it is useful to quote the parameters constituting the core group:

1. Apgar Score of Less than 5 at 5 Minutes and 10 Minutes

2. Fetal umbilical artery pH less than 7.0, or base deficit greater than or equal to 12mmol/L, or both

3. Neuroimaging Evidence of Acute Brain Injury Seen on Brain Magnetic Resonance Imaging or Magnetic Resonance Spectroscopy Consistent With Hypoxia–Ischemia

4. Presence of Multisystem Organ Failure Consistent With Hypoxic–Ischemic Encephalopathy

CTG is not thrown out of the window, but amply considered in the second group. It is also essential to stress that the ACOG -AAP Criteria for establishment of HIE cannot be extrapolated to the clinical management of labor. CTG monitoring's "second class group age" for HIE criteria establishment, does not put CTG in a second class category for the clinical monitoring of high risk labor. CTG still does have an indispensable role. At this point, it is crucial to keep in mind the UK's NICE [36] guidelines section on - Intrapartum Care [37], which, in section 1.10.3, advises continuous CTG monitoring in high risk situations. In Table 10 of the same guidelines, we also find two priceless gems of advice, which are of value both clinically and medico-legally:

- Do not make any decision about a woman's care in labour on the basis of cardiotocography (CTG) findings alone.

- Take into account any antenatal and intrapartum risk factors, the current wellbeing of the woman and unborn baby, and the progress of labour when interpreting the CTG trace.

The ACOG and AAP core criteria for establishing the presence of HIE, the underlying pathology of hypoxia induced Cerebral Palsy, should be contrasted with CTG oriented Cerebral Palsy Court argumentation as found in *Tippett v Guy's & St Thomas' Hospital NHS Foundation Trust and Coyle v Lanarkshire Health Board* (and a myriad other cases). On the other hand, one may quote recent examples where the latest reasoning as advised by ACOG -AAP Criteria, makes a healthy appearance. In *AW Pursuer against Greater Glasgow Health Board Defenders* [38], one finds the criteria being used admirably, to cut to the bone of all argumentation. This is the supremely ideal way to ensure fairness of presentation of Cerebral Palsy cases at medico-legal evaluation. However, maybe, one should not over rejoice at this stage. If *AW Pursuer against Greater Glasgow Health Board Defenders*, being a 2015 case, gave cause for hope, *MacLeod and another v Highland Health Board,149 a* 2016 case, sadly lets us down, for once again I-P CTG monitoring, once again makes its indomitable appearance.

One does not expect, the path of truth to run smooth and true,

with no challenges on the horizon. Here, argumentation is limited to one example, where a plaintiff's defence finds itself deprived of most of the elements necessary to fulfill the ACOG -AAP criteria, except for an abnormal I-P CTG tracing and little else. Apgar scoring is unlikely to be missing, but fetal umbilical artery pH, base deficit and neuro-imaging data may well not be available for a child born, say fifteen years ago. Should the plaintiff's lawyer refuse the case, even if it seems genuinely due to medical negligence? To these and other questions, there are no easy answers, but two points need stressing. Firstly, a case in Court is heard by a venerable judge, and the final decision is his after analysis of all that is presented before him. No expert and no scientific criteria exponent replaces that prerogative, which often knows its way on meeting the limitations of science. This is nicely expressed by the Court of Appeal Re R (a minor) (No 2) [39], as *the aetiology of the postnatal fits, the trial, had trespassed on the frontiers of medical knowledge and understanding.*

Secondly, truth, should be treasured, nurtured and disseminated. Especially so in Medicine and its cousin, medico-legal litigation. Completely dispelling the myth will not be painless, but must be done. Ask the victims over the last five or six decades.

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