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# The Medico-Legal Implications of Hypoxic Ischaemic Encephalopathy in Contemporary Cerebral Palsy Jurisprudence

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## Abstract

There are specific moments in medicine where a newly opened door leads to findings, the full import of which can only be discerned but not fully initially apprehended. The evolution of cerebral palsy litigation has seen a number of doors open since the 1950's and many of them led to scientific and legal dead ends and fatal drops. The initial mistaken scientific assumptions of the early fifties have had clinical and subsequent medico-legal disastrous implications of the first order. These pivotal facts are reviewed here as is hypoxic Ischaemic encephalopathy (HIE) the modern epicentre of the science linking cerebral palsy causology and the jurisprudence of alleged obstetric negligence in cases of infantile cerebral palsy. This paper provides reflections on both the unfortunate past and, learning from experience, it promulgates the scientific approach which Court can adopt to effect a just retrospective analysis of labour management and its potential link to hypoxic-induced peripartum cerebral palsy. Reflecting on the last 50–70 years of cerebral palsy causology and litigation are indispensable in planning the future. As matters stand, it is HIE which is the torch bearer in displacing those scientifically proven wrong aspects which still haunt some Courts and furthermore in providing a firm, uniform and just platform for future litigation and jurisprudence.

**Keywords:** Cerebral Palsy; Peripartum Hypoxia; Intrapartum Cardiotocography; Litigation; Jurisprudence; Myth; Science; Hypoxic Ischaemic Encephalopathy; Pre-Trial Hearing; Shifting The Medico-Legal Paradigm; Future

**Abbreviations:** HIE: Hypoxic Ischaemic Encephalopathy; CTG: Cardiotocography; IPCTG: Intrapartum CTG; PTH: Pre-Trial Hearing; ACOG: American College of Obstetricians and Gynecologists; AAP: American Academy of Pediatrics.

## Introduction

In the USA of the 1950's, a number of erroneously based scientific observations, accidentally "born" together were mistakenly linked in wrong arguments which concluded that the majority of infantile cerebral palsy resulted from fetal hypoxia during maternal labour [1]. Similarly misdirected thinking seemed to provide the tools to retrospectively reveal when such intrapartum hypoxia had occurred in a labour which was thought most likely negligent!. This led to an estimated increase of about 10% in obstetric lawsuits between 1970-1985 [2]. The concept and resultant litigation soon spread to Europe and beyond. Now, a good seventy years later, when post-mortems of the involved science have confirmed and re-confirmed the wrongful logic of the time, cerebral palsy litigation including jurisprudence is still not entirely rid of the harm of the 'great cerebral palsy myth'.

It is clearly not quite universally appreciated, that the

relatively recent established concept of hypoxic ischaemic encephalopathy (HIE) has offered one firm grip to the scientific world in repairing the resultant damage from originally wrongful science. Furthermore, one firm solution would be for a great medico-legal paradigm shift in the handling of infantile cerebral palsy litigation to be *universally* applied with HIE leading the cataclysmic charge.

Let us first review how science has misled both medicine as well as jurisprudence, commencing to the USA of the 1950's.

## Where did Science Go Wrong?

While retracing the "logic" which flowered in the USA into the 1950's serious misconceptions, it is difficult to comprehend the enormity of the mistaken science which misled both obstetrics and law. The erroneous conclusions were immediately pounced on by litigation lawyers who have held on to the subject to this very day, when "birth litigation lawyers" advertisements litter the related sections of the internet.

Without delving into the details of the great cerebral palsy myth hitting the scientific world, the following were some of the major tenets of the (then) new bible:

- That the great majority of infantile cerebral palsy cases was due to peri-partum hypoxia. This myth became further firmly entrenched by the early1960's [3].
- That, therefore, this was preventable by careful and orthodox management of labour avoiding intrapartum hypoxia.
- That the newly clinically introduced CTG in labour (Intrapartum CTG [IPCTG]) would invariably detect fetal hypoxia in labour.
- That, as logically understood by combining the above reasoning, the introduction of IPCTG would diminish the incidence of cerebral palsy.
- That evidence emanating from a disturbed IPCTG tracing and evaluated along the resultant management of the obstetrician in labour will provide unchallengeable and proof positive of obstetric negligence.

The unique aspect of the resultant evil of the medical and jurisprudential based fallacies of cerebral palsy has never been clearly and universally aired as a rich lesson in remembering the clay feet of the god of science. The repercussions may be too painful and potentially too expensive. In fact, the situation is even more disturbing, for there are still Courts at this very moment where obstetric negligence or otherwise is still being judged along the above mumbo-jumbo in part or *in toto*.

## Present Knowledge of Facts Pertaining to Cerebral Palsy Aetiology

This topic requires extensive evaluation but one may crystallise some of the more quintessentially important points especially in contrast to the above.

- That the great majority of infantile cerebral palsy is totally unrelated to and independent of peri-partum hypoxia. Only 14.5% of cerebral palsy cases are known to be due to intra-partum hypoxia [4].
- That, therefore, it is only this great minority which is preventable by careful and orthodox management of labour.

  Apgar et al. had already made reference to this as early as 1955, when they reported that they found.

…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].

These observations have been repeatedly confirmed such as by Nelson, et al. [6] in 1991.

- That in truly hypoxic induced cerebral palsy cases, IPCTG *may* show disturbances which *if confirmed* may indicate the presence of fetal hypoxia in the second stage of labour and if this is ignored, brain damage or death may result:

Failure to recognise and act on an abnormal CTG is one of the most common causes of intrapartum stillbirths and can lead to complex medicolegal issues [7].

The gross misdirection to cerebral palsy jurisprudence by the science of last five decades of the 20th century has been based on an assumption that IPCTG abnormalities are *necessarily indicative* of fetal distress in a case of cerebral palsy. This is now known to be false.

- That the use of CTG does not diminish the incidence of cerebral palsy.
- That CTG during labour is associated with reduced rates of neonatal seizures, but with no clear differences in cerebral palsy, infant mortality or other standard measures of neonatal wellbeing [8].
- That, cases alleging obstetric negligence as causative of eventual infantile cerebral palsy require jurisprudence which is completely different than that historically recognised in the 1950's when Court thinking was wrongly guided by science [9].

These are but superficial points which can be greatly amplified. For example, the relationship of IPCTG to the presence of intrapartum fetal hypoxia requires, as a minimum, the evaluation of those intrinsic scientific drawbacks which are now known to plague IPCTG in such circumstances. I refer

here to aspects such as the high specificity, low sensitivity, the high inter and intra-observer error of CTG. These carry significant clinical and subsequently medico-legal implications. Thus, as one example, IPCTG's sensitivity is so low that the false positive rate of External Fetal Monitoring for predicting cerebral palsy > 99% [10].

## Misuse of Cardiotocography as a Major Obstruction to Cerebral Palsy Jurisprudence

If one were to choose, the greatest offender in the search for truth in a Court case of cerebral palsy, it is, unquestionably the misuse of IPCTG. And here, one observes another interesting phenomenon, namely, the recalcitrant historical attitude to early scientifically sound warnings. Even as far back as the 1970's red lights were flashing. In 1976, Scott [11] had warned that time and time again, it had been shown that very few cases of cerebral palsy can be explained on the basis of birth asphyxia.

Not only were no scientific or legal alarm bells set ringing but at this time but the Court use of the wrong science was increasing and spreading like wildfire. In spite of the mounting scientific evidence advising prudence, the jurisprudential application of the original mistakes was becoming ever more entrenched both in the USA as well as the UK, especially fanned by birth litigation lawyers. Even the turn of the century would not witness complete annihilation of the wrong use of IPCTG in cerebral palsy litigation. Yet by the 1990's science was clearly stating 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 palsy [12].

There was another aspect by which bind faith in CTG contributed much damage to cerebral palsy jurisprudence. This was an increasing tendency for Courts to conclude on obstetric negligence essentially solely on the IPCTG tracing, which is often, open to much difference of opinion. Suffice it to state that even as far back as 1985, the American College of Obstetricians and Gynecologists published a study in which four obstetricians examined 50 IPCTG tracings with an impressively low 22% agreement rate [13]. Since then abysmal inter and even intra-observer rates have been repeatedly confirmed.

We found considerable variation in the classification of CTG patterns. Observers agreed poorly with each other and fair to good with themselves on CTG classification and clinical management [14].

And yet, numerous examples exist in both sides of the Atlantic, and indeed from many other countries, where such jurisprudence may have been solely limited to interpretation of an IPCTG tracing.

Not only is this now known to be completely misleading, but furthermore many are the published Court transcripts where it is clear that the situation was even further scientifically prejudiced by the fact that the numerous inherent scientific CTG deficiencies were compounded by mistakes pertaining to classification and nomenclature - the "Shifting Sands Phenomenon" [15]. One may quote some of the numerous available instances. In the 1994 case, *Robertson (an infant) v Nottingham Health Authority* [16] we read:

10.30 pm-trace again shows dips after contractions like loss of contact and again were not confirmed by Pinnards.

In the transcript of this case we find 8 important references to 'dips' a term dropped from CTG nomenclature altered in 1967 when the terms Type I and Type II dips had been replaced by early, late and variable decelerations. Again, in *Whiston v London Strategic Health Authority* [17] we read:

It is said that if the CTG had still been available the court would be able to tell when it was discontinued and whether there were Type II dips and, if so, for how long (i.e. whether they were continuous) [17].

This is a 2009 case and the transcript clearly reveals major case argumentation on outdated nomenclature and criteria. In *L v West Midlands Strategic Health Authority* [18], another 2009 case we find:

(viii) 21.25 the notes record "FH [foetal heart] ? 70 bpm x 1 then 75 bpm x 1. Type 1 dips. PT [patient] turned on to side. 02 via face mask given". The Partogram records these two decelerations, but not the subsequent ones, for which there is a note referring to the CTG.

Here one notes the utter confusion where first, the term 'Type I dips' is used and then 'decelerations' added. Unfortunately, one can hardly refrain from stating that the CTG here has been turned into one aspect of what is classically referred to as junk science.

## Proposing a Scientifically Based Approach to Cerebral Palsy Jurisprudence

### The Three Phases of the Evolution of Cerebral Palsy Jurisprudence

One may speak of three phases of the progressive evolution of the science underlying cerebral palsy jurisprudence.

**Phase 1** comprises the scientific application of known science, *defective as it was*, in medico-legal evolution of the causology of infantile cerebral palsy. In reality this was the dawn both of science's enunciations on the subject (albeit mistaken) and the unusually quick application of these conclusions in litigation battles.

**Phase 2** is the stage at which we are at the moment. It can be divided into:

A. The crucially important critical reflection of how and where science went wrong and its subsequent misleading of cerebral palsy litigation.
B. The acceptance of the new, scientifically repeatedly confirmed findings regarding the causology of cerebral palsy and the recognition that this knowledge is itself in its infancy.

One crucial element of Phase 2 B is the realisation of the importance of HIE . Each and every case of hypoxic induced cerebral palsy case must be preceded by HIE. In fact, one may speak as HIE as theoretically replacing the originally assumed central importance of IPCTG in the Courts of the 1950's. Not that any one feature, phenomenon or action can *by itself* be used to declare the presence or absence of intrapartum negligence!

**Phase 3** can also be considered as *commencing* contemporaneously and includes the practical application of the modern knowledge of the evolution of the causology of cerebral palsy, across the board, in all Courts and in all countries. This seemingly banally easy task should not be underestimated for even now there are Courts both in the UK and the USA where HIE does not even figure in the final jurisprudence of alleged obstetric negligence allegedly causing infantile cerebral palsy. Indeed, there are still cases involving litigation and Court action where reports of the extent of the child's cerebral palsy and an intrapartum strip tracing of the parturient mother are the only two pieces of "evidence" presented in Court. Although Court is sovereign and is not to be dictated to, it is time to exert its authority with regard to the artefacts to be presented at Court in cases of cerebral palsy jurisprudence. It is difficult in the year 2021, to envisage a just Court ruling on alleged obstetric negligence in a case of cerebral palsy without evaluation and consideration of the specific encephalopathy which *must* precede the development of the condition. And yet even now, cases *can* be found where the term encephalopathy, never mind the hypoxic ischaemic variety, is not even referred to once in the whole Court transcript!

## Pre-Trial Hearing

In Court cases centred on cerebral palsy, a Pre-Trial Hearing (PTH) offers many advantages. Among other basic facts, it may determine if evidence exists pointing to intrapartum hypoxia as the cause of presenting complaint or whether the required criteria are not even fulfilled. Here, one must be careful, for excluding hypoxia as a cause of the presenting complaint, does not, *per se,* exclude medical negligence of any other kind. Thus, birth trauma may still have occurred as the *primary* insult o the fetal brain in the absence of significant primary hypoxia.

At a PTH hearing, the Court may be satisfied that all the criteria required to establish HIE are present or not. In 2004 [19], the American College of Obstetricians and Gynecologists (ACOG) in conjunction with the American Academy of Pediatrics (AAP) published firm criteria for diagnosing HIE. A second report [20] in 2014, enriched the first with further views on the subject. Bearing in mind, the difficulties in establishing the relevant diagnosis in prematurity of 34 weeks or under, the following must be made available to the Court for an infant born at a maturity of at least 34 weeks. The main pillars of diagnosis of HIE are

- The establishment of metabolic acidosis (pH below 7 mmol/L and base deficit of ≥12 mmol/L).
- The establishment of quadriplegia/dsykinetic cerebral palsy.
- The exclusion of identifiable causes of the clinical picture.
- However, other parameters are crucial in establishing the *chronological* sequence.

This means that the following must be made available

- Apgar scores.
- PH and base deficit of the child's venous blood at birth.
- IPCTG tracing (notice that this is placed in the secondary criteria group with relevance essentially in establishing the chronologicity of events. Contrast this to its use, sometimes on its own in the Court mentality dating to post 1950's thinking).
- The exclusion of identifiable causes such as coagulopathy, infections, etc.
- The detection of any multi-system hypoxic involvement of the new-born.
- Pediatric neuro-imaging results showing acute, non-focal cerebral lesions.
- Repeated expert paediatric neurological assessments.

## As Matters Stand

It is by no means rare for a Court claim to be submitted for a birth dating back years and even decades with medical records which are never found or at best provide a partogram and if one is lucky, an IPCTG strip tracing. Court cannot pack up and go home when this happens. It must use its wisdom and discernment with what is available. However, in 2021, it should be made incumbent for all birthing units to supply all necessary information to establish the diagnosis of HIE in all cases of birth of infants with cerebral problems. It bespeaks

much poor judgement for a *contemporary* birth case to be accompanied by evidence limited to an Apgar score, an IPCTG and *maybe* ph and base deficit measurements at birth. Even in birthing units lacking neuro-imagery equipment, regional referral is almost always possible to organise once an infant is stabilised.

Phase 3, as discussed above, should involve among its numerous possible future steps, medico-legal hospital fora, where hospital corporate responsibility is evaluated in establishing investigative and management protocols in cases of worrying birth outcomes. This will eliminate *individual* obstetric/pediatric lines of investigations and management in cases involving post-birth encephalopathy. Furthermore, it should be made clear, that, sooner or later, as Phase 3 automatically evolves, the modern knowledge of cerebral palsy causology will make such investigations inevitably incumbent in cerebral palsy litigation. This must be universally understood and established to the point that non-availability of the established criteria in Court should generate a default negative standing on par with the phenomenon of spoliation of evidence. Science can only be used in the search for legal truth in Court, if it is given a serious opportunity to be evaluated by genuine scientific reasoning limited not by careless lack of available data for a particular case but by the published research of the time.

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