

Lessons from the Great Medico-Legal Chapter of Cerebral Palsy

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Abstract

It is not pleasant to reflect on the truth that science can be wrong. Even more so, when such wrong scientific conclusions have found expression in Courts of Law, and fashioned the destinies of men and women. Yet, facts are facts and truth is truth, whether we face it squarely or turn our heads to avert its gaze.

The misdirection in the aetiology of Cerebral Palsy generated in the USA in the 1960's has the onus of responsibility of misdirecting not only medicine, but, also jurisprudence, as far as the scientific advisory goes. This article has the unhappy responsibility of recalling those facts, in order for history not to repeat its sad past. It is neither uncommon nor unusual for science to correct its original statements. However when medical science has erred and its mistaken conclusions wrought mistaken clinical conclusions, it is serious business. It is doubly serious when those conclusions have been applied in a Court of Law, which needs medical advice in the form of an expert.

This article commences from the original two pillars of misconception and retraces the subsequent mistaken steps after the 1960's. One cannot point fingers at individuals, but one is certainly obliged to lance the collective buboes, and once the pus is freed, encourage regeneration of growth. And since, the essential core of the scope of the reasoning is medico-legal, it is essential that at no point does the article oppose legal retribution of sub-standard obstetric practice leading to the disastrous condition of Cerebral palsy of a new-born child. Such legal redress is right and just. However the mechanism of assessing the parameters in use in bringing forward such a claim must be fairly based on correct science. Only then, can justice prevail, both for the plaintiff as well as the defendant.

Keywords: Cerebral palsy; 1960's; CTG monitoring; Intra-uterine hypoxia; False conclusions; Misdirected court action; ACOG -AAP guidelines; Future action

Introduction

Background: A painful outcome

Birth should be one of life's greatest gifts. The birth of a child with Cerebral Palsy still brings the gift of life, but also much pain to heart-broken parents. One will also fully appreciate that such a birth occurring in a modern obstetric unit may also generate questions as to causation and preventability and eventually, often, to a sense of outrage combined with a wish both to punish and claim recompense. If labour was fully monitored, how could such a thing happen in this day and age? It is understandable that parents would blame those whom they trusted with this care. The location of the care and birth also carries an onus of responsibility and are deemed responsible for the outcome unless proved otherwise. And such proving may require a Court action dragging over many years.

No one doubts the humanity of the reasoning. Furthermore no one would question the right of such parents to seek legal redress. It would be right and justifiable, on simple human reasoning and especially in a situation which is going to radically alter the parents' lives at all levels, not forgetting the financially challenging aftermath.

But how is the issue resolved in Court? What is the logic of the medico-legal discussions? In essence, the basic pattern of proving sub-standard obstetric and/or neonatological practice must prove that intra-uterine or less likely peri-natal asphyxia was present as a result of medical omission or commission and that this hypoxia produced the underlying brain pathology leading to the clinical manifestation of Cerebral Palsy. Most cases deal with presumed intra-uterine hypoxia. Hence we are looking at the situation of labour. Labour is the time of greatest challenge to fetal wellbeing because with every contraction the cervix dilates further and the presenting part (often the fetal head) descends further down but every contraction temporarily diminished blood to the feto-placental unit. In most cases, this works out fine and

a balance is struck, by nature, until the child is born. Occasionally, for a number of possible reasons, it does not, and the child develops hypoxia and subsequently acidosis and if this is prolonged either brain (or other organ) damage ensues or the child dies in utero or at birth. Hence, does not logic dictate that if Cerebral Palsy is present, the first door to knock is the obstetrician's? To answer that million dollars question we must look at what happened in the USA in the 1960's.

A significant issue

Cerebral palsy litigation is no small matter. 73.6% of US obstetricians have faced litigation at some time - most often for alleged causation of fetal neurological impairment [1]. 60% of all obstetric malpractice insurance premiums cover birth management-related CP allegation [2]. In some states, the unbelievably high premiums, at times, up to \$200 000 per year [3], are leading to the phenomenon of obstetric practitioner depletion, by deterring the choice of the specialty or else by encouraging others to leave, either completely or else limiting themselves to gynecology [4]. The American College of Obstetricians and Gynecologists (ACOG) has predicted a shortage of 9,000-14,000 obstetricians in the USA over the next 20 years [5].

Hardly surprising given that obstetric Court settlements are among the highest verdicts, some exceeding \$200,000,000 [6]. Yet, it is interesting to note that This makes great socio-economic sense since less than 1 in 10 of the plaintiffs is awarded compensation in

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current Cerebral Palsy Court cases [2]. This fact does not diminish the psychological trauma and often unjust negative publicity of the doctors who face Court, at times, for years, until the Court rules. At this juncture, one aptly recalls the Mohr's [7], publishing the words of one physician who complained that so many lawsuits were being filed without reason or grounds that a spirit of persecution permeated medical practice. The situation bespeaks the milking of a cow and not genuine resort to Law to compensate damage from genuine malpractice. In fact the genuine case, the purpose of which is beyond any challenge, has obviously been hi-jacked by legal forces the roots of which are too deeply entrenched to be shorn out.

It is hardly surprising that such massive level of settlements is reached, when one considers what is at stake in a case of Cerebral Palsy, not just for the individual's family but also collectively for the nation. If we look at the estimate in the USA for the year 2003, for children born with Cerebral Palsy, mental retardation, hearing and visual impairment, we find costings of \$11.5 billion, \$51.2 billion, \$1.9 billion and \$2.6 million respectively [8]. 182 One person with cerebral palsy may have all the other three impairments. Furthermore, according to the USA United Cerebral Palsy Foundation [9], 764,000 children or adults manifest Cerebral palsy symptoms, 1,200 - 1,500 preschool age children are recognised to have cerebral palsy each year and about 10,000 babies born will develop cerebral palsy.

The Fateful 1960's

It is out with the remit of this paper to delve in detail in the causation of Cerebral Palsy. However certain relevant points need to be visited.

Cerebral Palsy results from brain damage or malformation that is most often innate and genetic. Such damage may result from a disturbance of the brain cell migration which occurs in utero as a result of genetic and environmental factors. Or the nerve insulation (myelination) is defective again due to genetic or growth dysfunction. Perinatal brain cell death may occur along with ruptured blood vessels when the brain is starved of oxygen. And finally the cause may be after birth by factors which effect the synapses between the brain cells and such factors.

Approximately 10-20 percent of cerebral palsy cases are now known to be caused by injuries at birth. The most common cause of these injuries is asphyxiation although birth trauma is also a potential cause. These constitute circumstances which are potentially preventable. But the rest, the massive (conservative) 80% involve pre-natal factors including infections (such as rubella, cytomegalo-viral infections, etc.), severe untreated jaundice, brain haemorrhage, and rarely, genetic causes. This is not an exhaustive list but it speaks volumes, among which is the role (in the great majority) of the pre-natal period.

The 1960's witnessed two facts, embraced by science, which shifted the search for the cause of Cerebral Palsy (and its inevitable legal liability) almost completely on the second stage of labour – the time of delivery. One should also note pre-1970, obstetrical care generated few medical malpractice claims [9], whereas by 1985, they comprised 10% of all medical malpractice lawsuits [10]. During and following the 1960's the world was led to believe that the majority of Cerebral Palsy cases are the result of oxygen deprivation during labour. Now we know that this contributes to a minority of cases. The following internet statement by the NHS (UK) sums it up nicely: In the past, doctors believed cerebral palsy was usually caused by brain damage sustained during birth as the direct result of being temporarily deprived of oxygen (asphyxiation). Asphyxiation can sometimes occur during a difficult or complicated birth. However, a major research project carried out in the

1980s showed that in babies born at term, asphyxiation was responsible for less than 10% of cerebral palsy cases. Most were due to problems with the brain that developed before the child was born [11].

Space does not allow the interesting evolution of this mistaken concept, evaluated elsewhere especially with its subsequent implications [12]. The reasoning had much clinical and medico-legal relevance, and it would be combined by the mis-use of another 1960's scientific discovery: cardio-tocographic monitoring (CTG). One must stress that it was the misuse of CTG monitoring not the invention itself, which was the second pillar of established wrong medico-legal litigation.

CTG monitoring is an electronic form of monitoring of a number of parameters of the pregnant woman and had a special role at the time of labour. The paper strip produced will contain information about the rate of maternal contractions and fetal heart information while the child is in utero. It came into clinical use in the 1960's and was hailed as the greatest discovery in the detection of intra-uterine hypoxia in labour, the time, of greatest changer. Labour is the time of greatest challenge to the fetus for with every contraction which opens the cervix, there is a concomitant temporary diminution of blood to the feto-placental unit. Most foetuses can withstand this (otherwise world population would not be what it is). But, in those situations, where, for example, a patient is starting labour with low placental reserve, the challenge of labour may tip the balance.

The strip tracing produced by the CTG monitor was a goldmine in the hands of birth injury lawyers. Science had embraced the notion that lack of oxygen in labour was most likely responsible for Cerebral Palsy and now, the actual proof, could be physically determined as produced in Court as exhibit number 1. So firmly did the medical world believe that Cerebral Palsy was, in its preponderance, due to intra-uterine hypoxia in labour, that the introduction of CTG was expected to lower the incidence significantly. It did not, and in due time it became clear that no more than a small minority of Cerebral Palsy could be prevented by dealing with intrapartum fetal hypoxia [13]. However, for the world of the birth injury lawyer (a) Cerebral palsy was due to intra-uterine hypoxia developed in labour (b) CTG strip recording of labour with any abnormalities furnished hard physical proof of such hypoxia.

From the USA and its two 'discoveries' in the 1960's, let us temporarily fly to the UK to the year 2011. We can state that intrapartum CTG monitoring is the commonest obstetric procedure in the developed world, while also being the most medically contested obstetric procedure in labour. In the same year, 2011, 'birth asphyxia' comprised 50% of the UK National Health Service (NHS) litigation costs, and in the 2000–2010 decade, the same NHS had paid out £3.1 billion for maternity medico-legal claims (the highest of any speciality), mostly involving cerebral palsy and CTG misinterpretation.

Examples from the Courts

Let us look at one UK case occurring in 2005 with the final Court decision being delivered in 2014. In *Joshua Tippet -v- Guy's and St Thomas' Hospital NHS Foundation Trust* [14]. This is a case occurring in the 21st century, when fetal brain neuro-imaging and fetal oxygenation and acid base status could have been carried out on the neonate being easily effected on the neonate. However the plaintiff's case revolved round the fact that that CTG monitoring was not continued in the face of some abnormalities. And the child was eventually born suffering from Cerebral Palsy. The Court concluded that the evidence presented was not determinative of liability. A different approach by the plaintiff could have offered findings of low Apgar score, evidence of hypoxia at birth as well as acidosis and with neuro-imaging evidence of hypoxic

ischaemic encephalopathy. Instead we have typical 1960's logic trying to link some unconfirmed CTG disturbance in a situation where CTG was discontinued. Had plaintiff showed evidence that Hypoxic Ischaemic Encephalopathy (HIE) was the underlying brain lesion, the outcome may have been different.

In *Brodie McCoy v East Midlands Strategic Health Authority* [15], a 2011 UK case, not only was the claim for medical liability purely based on a CTG tracing but this CTG tracing was an antenatal one and no mention at all is made of any monitoring during subsequent labour which led to the birth of an infant with Cerebral Palsy. In this most odd of claims, the antenatal CTG tracing showed contentious findings. The patient was discharged. Had a still-birth ensued after this discharge, one might have understood such a claim. However the claimant returned four days later in the evening, having started in labour and in the morning, delivered a child suffering from Cerebral Palsy. No information is given about any monitoring in labour but the contentiously suspect antenatal tracing of four days previously was used as grounds for claiming liability. One could call such strange argumentation as venial at best. Yet this stresses the persistent role played by CTG monitoring in the minds of both the laity as well as some of the legal counsellors who in 2011 ought to know better. Apart from the non-sequitur of the argumentation, the still dreamt of magic link of CTG abnormalities and Cerebral Palsy, still haunts the medico-legal world of the 21st Century.

Of complexities of science and popular delusions

It is most surprising that the complexities and their legal implications of the CTG- Hypoxia -Cerebral Palsy, has not generated more extensive and intensive publications, heated dialogue and condemnation. Fierce argumentation has been forward by erudite authors like Thomas P. Sartwelle (a lawyer) and Hames C Johnston, a medical doctor but on the whole the medical world has been strangely silent. It has been stated that the second pillar of the great Cerebral Palsy medico-legal myth was the wrong use of CTG monitoring and not CTG monitoring per se. This monitoring is still in use and will be in use in the foreseeable future and used correctly has a great service to offer. However, over the decades, much of its drawbacks have surfaced and are now incorporated in instructions of its correct use. In the 1960's, it was medico-legally grasped and put into action and presented at Court, almost as a standard of care of the obstetrician. Courts, across both sides of the Atlantic, are littered with Cerebral Palsy transcripts, where a CTG tracing, seems to be the alpha and omega of judging obstetric performance. This is wrong, even if CTG monitoring did not have serious drawbacks which are briefly highlighted here.

Firstly, CTG monitoring, by itself, cannot make a diagnose for fetal hypoxia and acidosis. Even in a bad case scenario the CTG tracing showing a fetal baseline tachycardia, with reduced variability, no accelerations and late decelerations, the incidence of fetal hypoxaemia and acidosis can be confirmed in only 50–60% of cases [16]. An abnormal CTG tracing is an indication for confirming or negating intra-uterine hypoxia such as by Fetal Blood Sampling (FBS) [17], with or without one of the more modern methods such STAN. Yet due to defensive stance taken by the man on the spot, aware of the potential horror of medico-legal action, countless are the unnecessary Caesarean Sections performed. Alfirevic et al. estimate an additional C-Section for every 58 women monitored and one additional C-Section for every 12 high-risk women monitored in labour [18]. How often is corroboration of fetal hypoxia and acidosis put forward in Court when CTG abnormalities are put forward as evidence of causation of Cerebral Palsy?

Furthermore, there are many weaknesses with CTG monitoring

which need to be firmly kept in mind, especially, when such monitoring has been elevated to the seeming sole standard of care in medical jurisprudence. These elements which have been elsewhere referred to collectively as the 'shifting sands phenomenon' include [19] among others high specificity and low sensitivity, high inter- and intra-observer errors. While a normal intra-partum CTG truly reflects a healthy unborn child, an abnormal tracing does not necessarily equate with foetal hypoxia. Sensitivity may be as low as 99.8%, with only 0.19% of their abnormal CTG tracings being associated with moderate or severe cerebral palsy [20].

The high inter- and intra - observer errors refer to disagreement of CTG interpretation by different observers, while the latter refers to different interpretations given by the same observer at different times. In one study with five obstetricians interpreting 150 CTG tracings, agreement was obtained only in 29% of the cases [21]. Without going in greater detail, it is only fair to stress this element of subjectivity in CTG interpretation, both clinically and medico-legally. "The subjectivity of CTG interpretation and inconsistencies in interpretation should also be considered in intra-partum management, clinical audit and in medico-legal settings [22]. To this one may add the ever-changing classification of CTG abnormalities. Which recalls the most appropriate comment by the Royal College of Physicians of Ireland: Communication between staff should convey the clinical context and use consistent terminology to describe the features of the CTG, the level of concern and the urgency of the situation [23]. One notes here that pleas for consistent terminology are being made by a major obstetric college as late as 2014.

The situation is more complex still, by the Court use, as evidenced by official case transcripts of outdated classification which is a separate issue from that of ever classification of CTG abnormalities. To quote but one example out of many: 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) [24]. The Term II dips were abolished in the 1960's by the very man who invented the early TypeI/TypeII [25] classification, namely Caldeyro Barcia.

In view of this brief look at I-P CTG pitfalls, one cannot but wholeheartedly agree with Sholapurkar that CTG can be expected to remain contentious for some time to come and NICE draft guidance may have missed significant fundamental improvements. This article repeats its strongly felt call, made elsewhere [19], that in view of the medico-legal implication of CTG evolution there is an onus for the major Obstetric Colleges across both sides of the Atlantic to issue formal advice of great prudence to all parties concerned in medico-legal interpretation of IP CTG and its application to retrospective assessment of specific obstetric intervention.

Some fall-out of the Court CTG-Cerebral palsy saga

Backlash has been rather limited but among one of the fiercest has come from the already quoted Sartwelle. Alone or with other co-authors he has repeatedly attacked the medico-legal evil of CTG monitoring. While respecting this fiery nature of the justifiable attack, one cannot agree with throwing out CTG monitor out of the window. As a practising Obstetrician, I would wholeheartedly condemn the abuse of CTG monitoring both clinically and medico-legally. CTG intra-partum monitoring, with all its faults, weaknesses and pit-falls remains the only means of monitoring labour the high-risk patient. The question is how to counter the role played by misapplied intra-partum cardio-tocography (I-P CTG) in "diagnosing" intra-uterine hypoxia in cases of Cerebral Palsy litigation [12]. The evil commenced in the 1960's is far from having been exorcised. There is no excuse for wrong

interpretation of CTG tracings and many a Court case has exposed these. Such cases often show ignorance of many aspects of obstetric management, which is one reason, why the Court should never keep its searchlight purely on a CTG tracing. Genuinely worrying CTG tracings exist and these should be a senior's opinion. But the 1960's harm by CTG monitoring was not the CTG itself but its immediate medico-legal hi-jack, at a time when the obstetric world itself was still in its learning curve of the subject. Many a birth injury lawyer has never even embarked on that same learning curve, and unfortunately, their falsely based arguments still purposefully confuse issues.

The Dawn of an Enlightened Era

Since there is no practical alternative to I-P CTG, the CTG monitor should and will remain part of high risk intra-partum care. A well interpreted Intra-Partum CTG (I-P CTG) tracing, has much to offer, in spite of its potential lack of objectivity and furthermore can also help pin-point the approximate commencement of the hypoxic challenge to the fetus. So, how best to circumnavigate the problem?

The answer lies in the underlying brain pathology of the case of Cerebral Palsy caused by oxygen deprivation. Such a case will have the features of Hypoxic Ischaemic Encephalopathy (HIE). If HIE is absent, technically speaking, no obstetric liability exists, from the point of view of mismanagement of intra-uterine hypoxia. We cannot be cavalier and state that obstetric mismanagement may be ruled out, for other factors, not involving hypoxia and HIE may be operative. For example Cerebral palsy may not be due to labour hypoxia but due to antenatal mismanagement of leaking amniotic membranes with resultant ascending intra-uterine infection. But the fact that HIE is the scientifically accepted lesion of hypoxic induced Cerebral Palsy. All cases seeking to prove medical mismanagement and subsequent liability, must as a solid first step, prove the presence of HIE. This is the first step in undoing the medico-legal harm commencing in the USA in the 1960's.

One Step in the Right Direction

The problem was not addressed directly, on medico-legal grounds, and rightly so, for the crux of the situation is primarily a clinical one. The question one should ask, as a first step is: How does one correlate clinical, neurological, biochemical, neuroimaging evidence of cerebral hypoxic ischaemia as well as similar evidence of damage in other organs to establish a proper diagnosis? The challenge was taken up squarely by the American College of Obstetrics and Gynaecology (ACOG) which in a ACOG Task Force Report issued in 2003 [26], and further amended in 2014 [27], not only stipulated the criteria of establishment of HIE but also furnished much extremely important related information. Here, time and space limit us to the HIE criteria (known as the ACOG – AAP criteria) [26]. These criteria are divided into a core group which is indispensable to the diagnosis and to a second group, the members of which are not by them specific for the diagnosis but have value in yielding further information e.g. CTG abnormalities may help establish the timing commencement of timing of the fetal cerebral damage.

The core group consists of

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 12 mmol/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.

Among the second group we find the (in) famous CTG tracing. Why in the second division, so as to speak? Because the end scope of CTG monitoring is the detection of fetal hypoxia and resultant acidosis and this is specifically called for in the Core group. However, the CTG monitor is not thrown out of the Sartwellian window, but, neither is the Sartwellian concern.

As has been stressed elsewhere [12], the relegation of CTG monitoring to the secondary group of the ACOG – AAP HIE diagnostic criteria has no clinical bearing in the management of high risk pregnancy, where CTG monitoring is still firmly recommended [28]. This is clearly indicated in the UK's National Institute for Health and Care Excellence (NICE) along with two pieces of advice which are of value not only clinically but also medico-legally, namely:

1. Do not make any decision about a woman's care in labour on the basis of cardiotocography (CTG) findings alone.
2. 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.

Keeping these two points mind, with special reference to the first, it would be salubrious to re-read the section entitled 'Examples from the Courts' in this article, bearing in mind these are but a minimal representation.

Is Anyone Listening?

It is perturbing that the medico-legal negative aspects of the progressive march of science regarding Cerebral Palsy from the 1960's to the present day, do not seem to have ruffled too many feathers. The proof that the lessons have been learnt can only be judged by Court hearings, by the plaintiff arguments presented and finally in jurisprudence itself. That is not to say that there are not some fine and exemplary Court proceedings of Cerebral palsy litigation. However, if one searches carefully, there are still numerous cases exemplifying the 1960's Cerebral Palsy myth, on both sides of the Atlantic.

Discussion and Conclusion

However it would be encouraging to conclude with one case, out of several, where the scientific aspect of case presentation in Cerebral Palsy elicits admiration. One such is AW Pursuer against Greater Glasgow Health Board Defenders [29], a UK case where the basis of the plaintiff's arguments are based on an argumentation augmentation of the ACOG – AAP criteria even if this is not referred to as such. A total of thirteen experts were involved between plaintiff and defendant, covering issues of obstetrics, neuro-radiology, neurology, neonatology and midwifery. This is the correct way to truly evaluate medical liability in Cerebral Palsy litigation. It is a far cry from two experts warring over whether a CTG artefact is significant or not. Once CTG oriented evidence is given its correct weighting, along with Apgar scores, fetal oxygen saturation and acid base status at birth, neuro-imaging evidence of the new-born's brain along with evidence of oxygen deprivation in other fetal structures, and justice will have to struggle hard to be effected. Until this is affected, the truth about the causation and hence possible liability of the obstetrician, can never surface. Although more than half a century has elapsed since the fateful 1960's, there are many who still need to see the light. And those many include doctors, obstetricians, neo-natologists, plaintiff and defense lawyers, the injured parents and finally Court itself. It is a change of attitude which will also produce a positive loop effect on medical practice itself. Only time can tell.

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