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Hospital
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ENCEFALOPATIA HIPÒXIC-ISQUÈMICA NEONATAL
IMPLICACIONS MÈDICO-LEGALS

LA VISIÓ DEL OBSTETRA

Emilio Pérez Picañol, 2016
eperezp.germanstrias@gencat.cat

Anàlisi de les reclamacions a professionals ginecòlegs en Catalunya (1986-2010)

Procedures	Total of claims	Payment rate (among closed files)	Payment mean (€)
Oncologic diseases	97	13.19% (12/91)	91,460.2
Breast cancer	74	14.28% (10/70)	88,852.2
Uterine cancer	20	11.11% (2/18)	104,500.2
Hysterectomy	80	22.39% (15/67)	57,861.9
Histerectomy itself	27	22.72% (5/22)	
Complications	53	20% (9/45)	
Fetus death during labour and delivery	73	22.22% (14/63)	111,583.5
Neurologically impairment child	68	31.66% (19/60)	477,871.6
Foreign object	51	71.73% (33/)	15,422.1
Gauze	43	70% (28/40)	16,968.2
Others	8	83.33% (5/6)	6763.9
Tubal ligation	49	39.58% (19/48)	35,288.3
Ineffective	28	32.14%	
Complications	13	69.23%	
Ultrasound diagnosis	47	18.42% (7/38)	403,224.2
Brachial palsy	27	26.09% (6/23)	143,025.9
Ectopic pregnancy	25	9.09% (2/22)	115,654.5
Ovary surgery	25	28% (7/25)	55,961.6
Voluntary interruption of pregnancy	23	30% (6/20)	87,579.2
Fertility treatments	20	15.79% (3/19)	69,365.3
IUD problems	16	21.43% (3/14)	12,329.3

E L Gómez-Durán, JA Mulà-Rosías y cols

Journal of Forensic and Legal Medicine 20 (2013) 442-446

ENCEFALOPATIA NEONATAL

LA VISIÓ DE LA PART ACTORA

- **Interpretació errònia del registre de la FCF,antenatal o intrapart**
- **Retard en la decisió de finalitzar el embaràs/part**
- **Creença de que una intervenció obstètrica en el moment adequat hagués evitat el dany fetal (rol de la cesària)**

Suport argumental

Aportació d'un dictamen pericial defensant la existència de MALA PRAXI o error assistencial

El dictamen pot haver estat elaborat per un especialista en obstetrícia i ginecologia, un metge forense o un metge especialista en valoració del dany corporal

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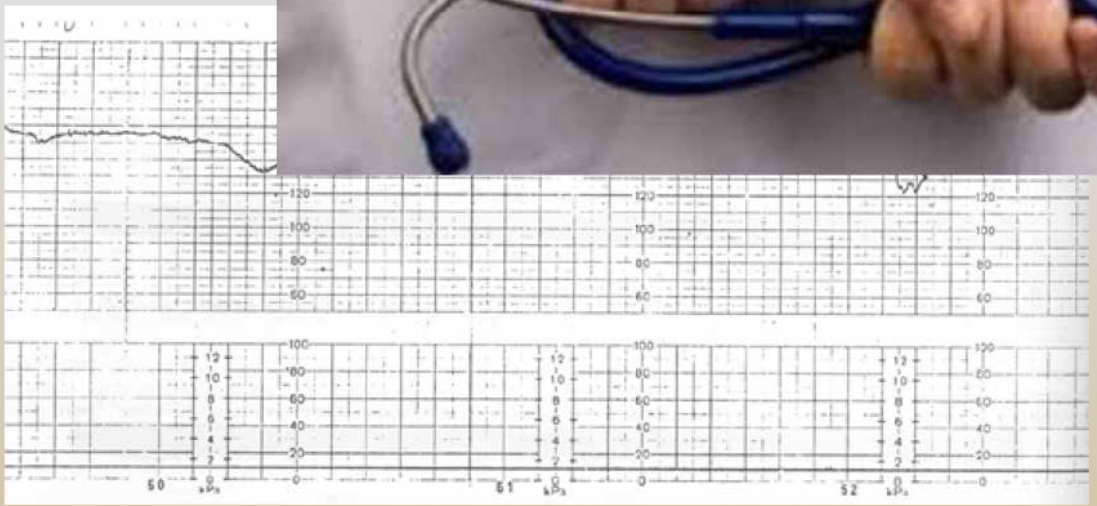
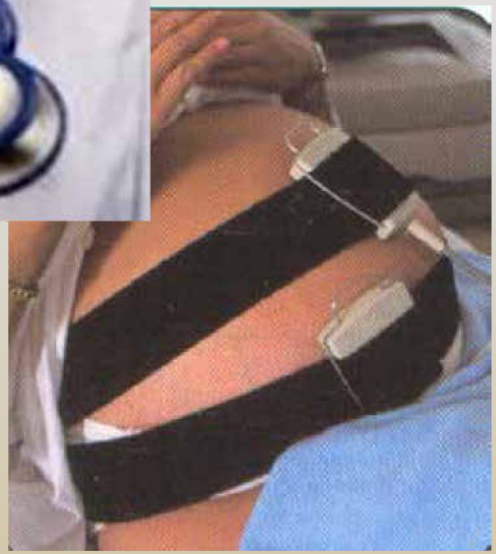
PROTAGONISTES DEL EPISODI JUDICIAL





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CO-PROTAGONISTA, HABITUALMENT NO DEMANDAT, PERÒ CLÍNICA I JURÍDICAMENT MOLT RELLEVANT:

EL NEONATÒLEG

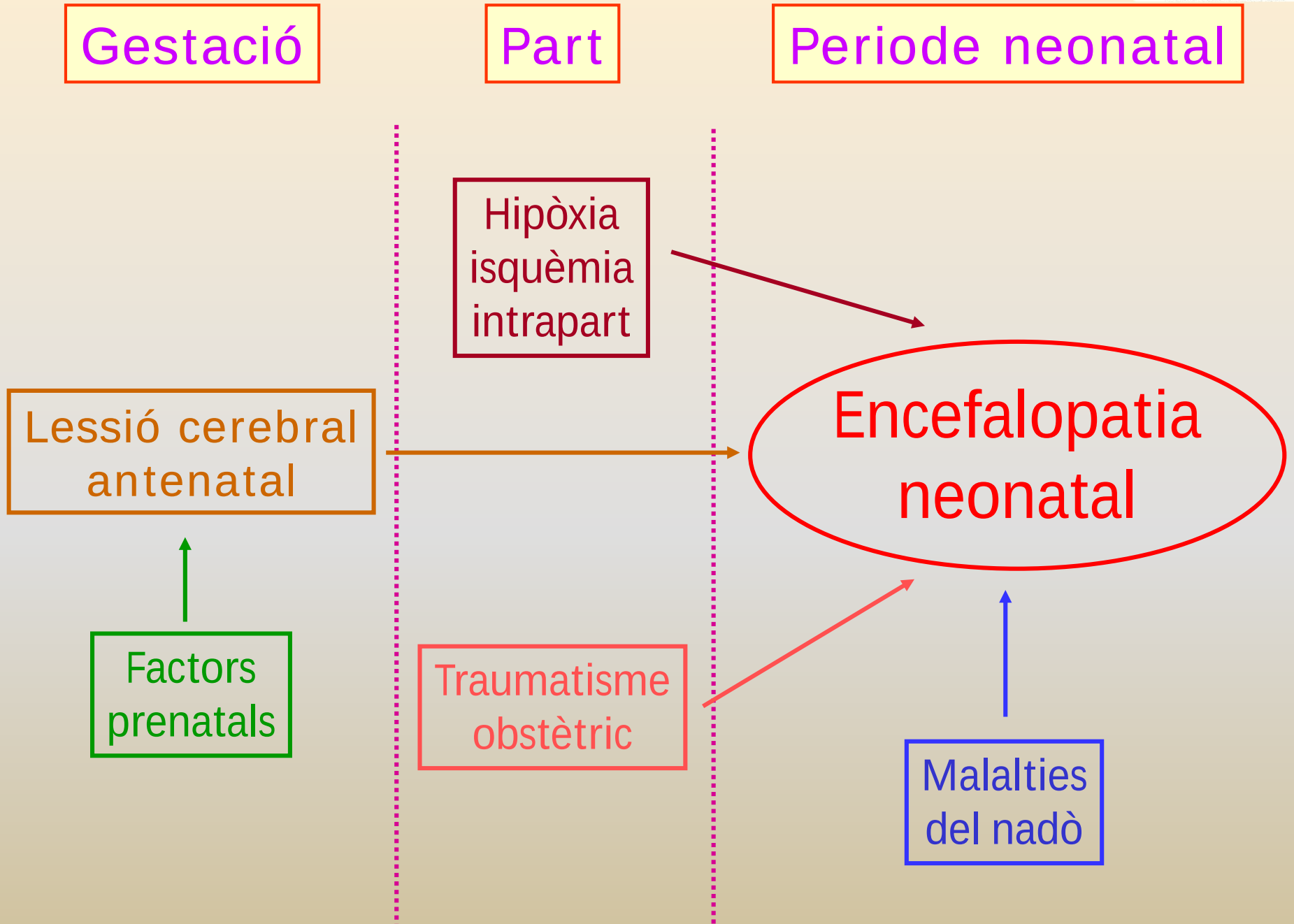
Paper fonamental del informe neonatal : els diagnòstics

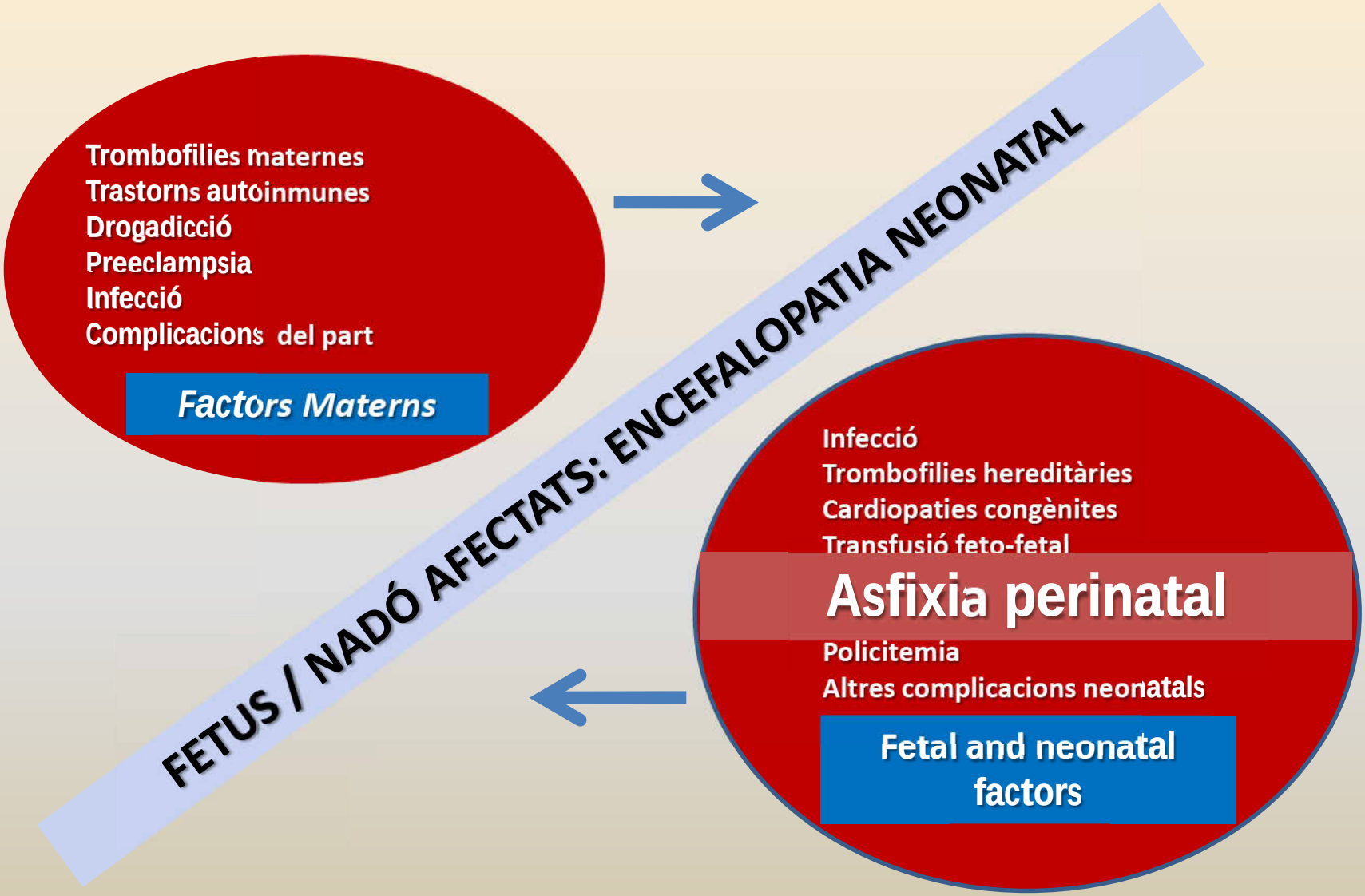
PATIMENT FETAL EN EL PART
ASFIXIA PERINATAL
ENCEFALOPATIA HIPÒXIC-ISQUÈMICA PERINATAL

ENCEFALOPATIA NEONATAL

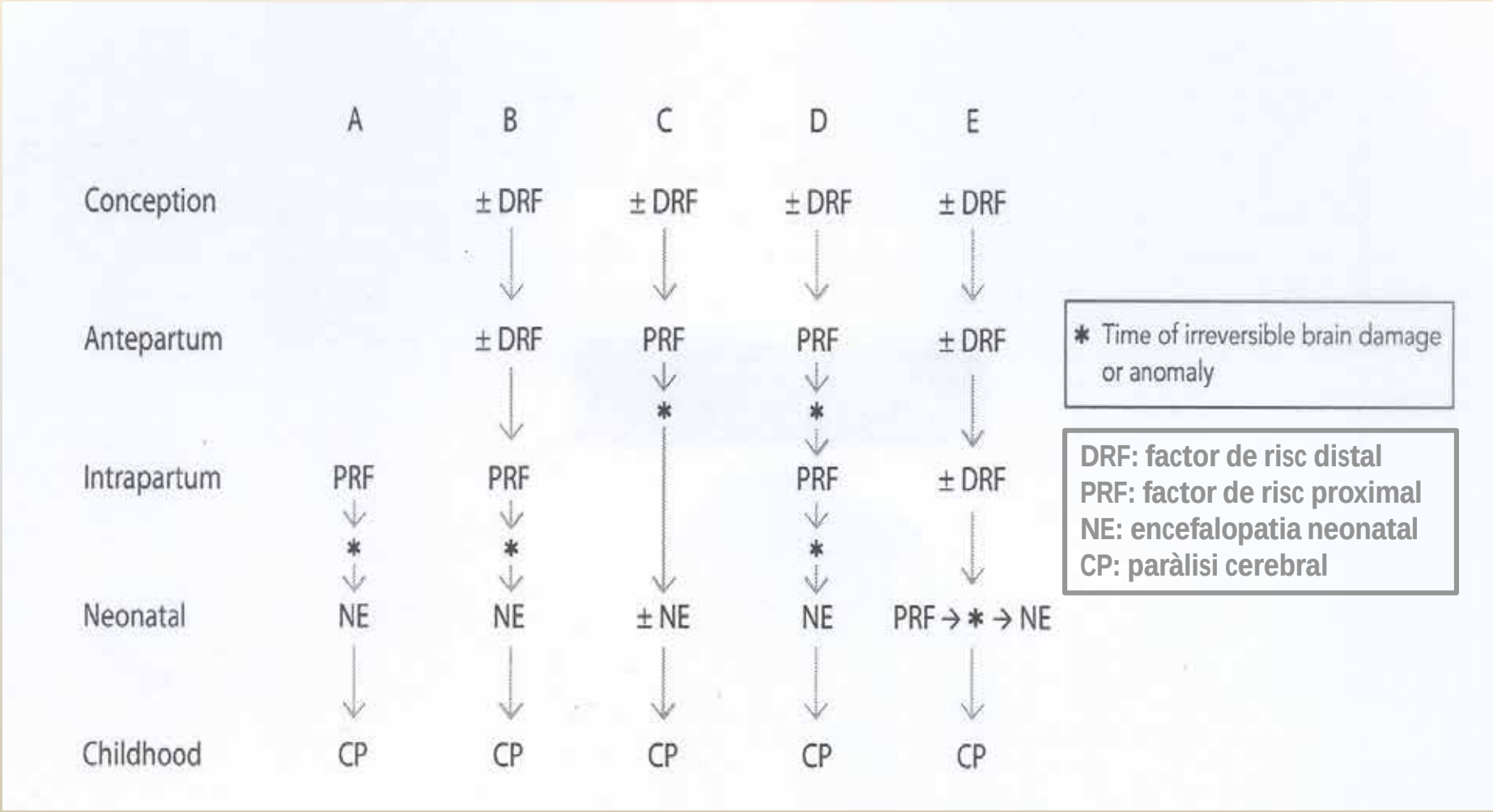
DEFINICIÓ

La encefalopatia neonatal es una síndrome heterogènia clínicament definida com a un trastorn de la funció neurològica en els primers dies de vida en un nounat nascut a partir de les 35 setmanes de gestació i manifestat per un nivell de consciència subnormal o convulsions i amb freqüència acompanyat de dificultats en iniciar i mantenir la respiració, en la alimentació i depressió del to i dels reflexes.



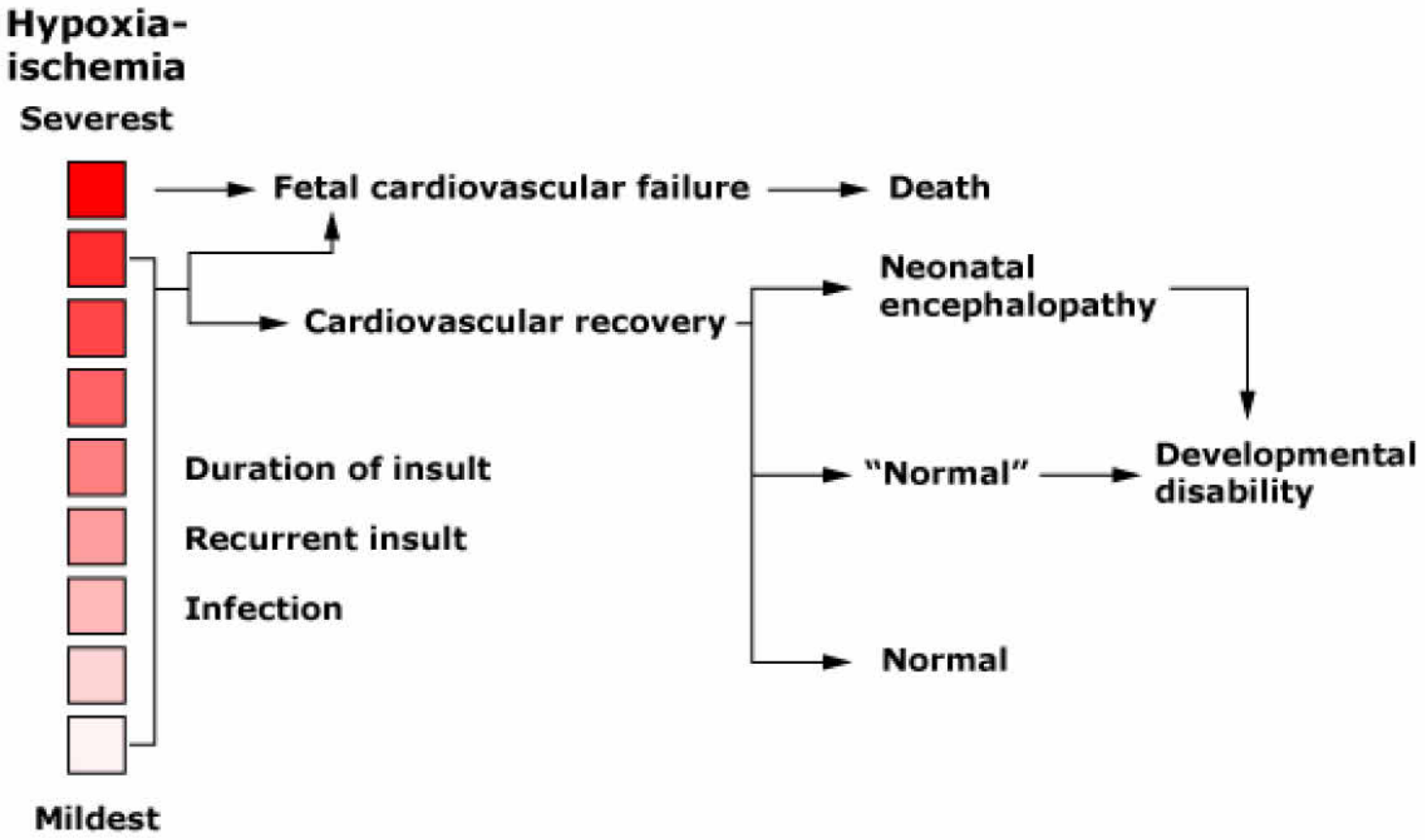

Trombofilies maternes
Trastorns autoinmunes
Drogadicció
Preeclampsia
Infecció
Complicacions del part
Factors Materns
FETUS / NADÓ AFECTATS: ENCEFALOPATIA NEONATAL
Infecció
Trombofilies hereditàries
Cardiopaties congènites
Transfusió feto-fetal
Asfixia perinatal
Policitemia
Altres complicacions neonatals
Fetal and neonatal factors

Etapes gestacionals, intrapart i neonatals en que determinats factors son causa de paràlisi cerebral en nascuts a terme



Task Force on Neonatal Encephalopathy , Obstet & Gynecol, vol 123, nº 4, April 2014

Outcome following neonatal hypoxic-ischemic encephalopathy



Sidhartha Tan, MD.

A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement

Alastair MacLennan for the International Cerebral Palsy Task Force*

Editorial by Bakketeig

Department of Obstetrics and Gynaecology, University of Adelaide Women's and Children's Hospital, North Adelaide, South Australia 5006, Australia
Alastair MacLennan *associate professor*
amaclennan@medicine.adelaide.edu.au

*The names of the task force, which consisted of 49 people from 7 countries, are given on the *BMJ*'s website

BMJ 1999;319:1054–9

website extra

Members of the International Cerebral Palsy Task Force and the references appear on the BMJ's website

www.bmj.com

Chairman's foreword

In 1997 the research committee of the Perinatal Society of Australia and New Zealand competitively funded a special initiative to bring together the modern literature on the causation of cerebral palsy, and to try to define an objective template of evidence to better identify cases of cerebral palsy where the neuropathology began or became established around labour and birth. Recently there have been many advances in a wide variety of scientific areas associated with cerebral palsy, and thus this multidisciplinary review may benefit research into the causation and prevention of cerebral palsy and may also help those who offer expert opinion when counselling in this area or giving such opinion in court.

The corresponding task force was open to anyone who could make a scientific contribution to understanding in this area. The task force had representation from a wide range of clinical and scientific specialties. Submissions were sought from the society's 1000 members, which include scientists, pathologists, obstetricians, neonatalogists, midwives, neonatal nurses, and epidemiologists. International contributions were sought from those identified from the current literature as contributing to this area through peer reviewed research. They were not preselected for their views, and they were invited to join the corresponding task force. Some international members joined later in the discussion process as word of this open debate reached them.

During 1997 and 1998 multiple online electronic conferences were held, and in March 1998 many of the task force members were able to participate in a workshop in Alice Springs, Australia to discuss the fourth draft of the statement. Drafts of the statement were circulated and debated, with the sixth draft being discussed at an international telephone conference in October 1998. The paper continued to be redrafted eight times until consensus was reached. No opinion was excluded from the debate, but the statement only includes discussion that has a reasonable scientific basis and can be referenced. All members agree that updated reports will be required within a few years when further information is published. It is hoped that other international researchers in this area will offer to assist in future statements. The final draft of the statement was sent to the professional colleges and scientific societies to which the task force members belong. To date no such group has declined to support the statement or has disputed its final content. Box 1 lists those professional bodies endorsing the statement at the time of publication.

Box 1—Supporters of the consensus statement

American College of Obstetricians and Gynecologists
American Gynecological and Obstetrical Society
Australian College of Midwives
Hong Kong Society of Neonatal Medicine
Institute of Obstetrics and Gynaecology of the Royal College of Physicians of Ireland
International Society of Perinatal Obstetricians
New Zealand College of Midwives
Paediatric Society of New Zealand
Perinatal Society of Australia and New Zealand
Royal Australasian College of Physicians, Paediatric Division
Royal Australian College of General Practitioners
Royal Australian College of Obstetricians and Gynaecologists
Royal College of Obstetricians and Gynaecologists
Royal College of Pathologists of Australasia
Royal New Zealand College of Obstetricians and Gynaecologists
Society of Obstetricians and Gynaecologists of Canada

Consensus was reached with some difficulty in two areas. Firstly, the current validity of neuroimaging in the infant to retrospectively determine the precise perinatal timing, pathology, or cause of the abnormalities seen on imaging. The task force awaits the publication of strong data, using the criteria suggested in its template, to define an acute intrapartum hypoxic event to validate specific neuroimaging appearances that occur as a result of acute intrapartum hypoxia. These images must be different from those that arise after chronic asphyxial or non-asphyxial causes of cerebral palsy in order to be of help in determining intrapartum timing.

The second area of debate was terminology, and in particular the term "non-reassuring fetal status" was debated. It has been adopted in the consensus statement rather than the term "fetal distress" as clinical signs often poorly predict a compromised fetus, and continued use of this latter term may

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Criteria to define an acute intrapartum hypoxic event

Essential criteria

1.- Evidence of a metabolic acidosis in intrapartum fetal, umbilical arterial cord, or very early neonatal blood samples (pH < 7.00 and base deficit >12 mmol/l)

2.- Early onset of severe or moderate neonatal encephalopathy in infants of >34 weeks' gestation

3.- Cerebral palsy of the spastic quadriplegic or dyskinetic type

Criteria that together suggest an intrapartum timing but by themselves are nonspecific

4.- A sentinel (signal) hypoxic event occurring immediately before or during labour

5.- A sudden, rapid, and sustained deterioration of the fetal heart rate pattern usually after the hypoxic sentinel event where the pattern was previously normal

6.- Apgar scores of 06 for longer than 5 minutes

7.- Early evidence of multisystem involvement

8.- Early imaging evidence of acute cerebral abnormality

*Alastair MacLennan for the International Cerebral Palsy Task Force**
BMJ volume 319 16 october 1999 www.bmj.com

CANVI DE PARADIGMA I GUIA DE DEFENSA EN CAS DE DEMANDA

En base a aquests criteris es va estimar que únicament el 25% del casos de EHI neonatal es podien relacionar amb una hipòxia aguda intrapart

The American College of Obstetricians and Gynecologists
WOMEN'S HEALTH CARE PHYSICIANS

Neonatal Encephalopathy and Neurologic Outcome, Second Edition

Report of the American College of Obstetricians and Gynecologists' Task Force on Neonatal Encephalopathy

Executive Summary

In the first edition of this report, the Task Force on Neonatal Encephalopathy and Cerebral Palsy outlined criteria deemed essential to establish a causal link between intrapartum hypoxic events and cerebral palsy. It is now known that there are multiple potential causal pathways that lead to cerebral palsy in term infants (see Fig. 1), and the signs and symptoms of neonatal encephalopathy may range from mild to severe, depending on the nature and timing of the brain injury. Thus, for the current edition, the Task Force on Neonatal Encephalopathy determined that a broader perspective may be more fruitful. This conclusion reflects the sober recognition that knowledge gaps still preclude a definitive test or set of markers that accurately identifies, with high sensitivity and specificity, an infant in whom neonatal encephalopathy is attributable to an acute intrapartum event. The information necessary for assessment of likelihood can be derived from a comprehensive evaluation of all potential contributing factors in cases of neonatal encephalopathy. This is the broader perspective championed in the current report. If a comprehensive etiologic evaluation is not possible, the term *hypoxic–ischemic encephalopathy* should best be replaced by *neonatal encephalopathy* because neither hypoxia nor ischemia can be assumed to have been the unique initiating causal mechanism. The title of this report has been changed from *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology* to *Neonatal Encephalopathy and Neurologic Outcome* to indicate that an array of developmental outcomes may arise after neonatal encephalopathy in addition to cerebral palsy.

In order to determine the likelihood that an acute hypoxic–ischemia event that occurred within close temporal proximity to labor and delivery contributed to neonatal encephalopathy, it is recommended that a comprehensive multidimensional assessment be performed of neonatal status and all potential contributing factors, including maternal medical history, obstetric antecedents, intrapartum factors (including fetal heart rate monitoring results and issues relating to the delivery itself), and placental pathology. A description of the items to be included in the assessment follows.

I. Case Definition

Neonatal encephalopathy is a clinically defined syndrome of disturbed neurologic function in the earliest days of life in an infant born at or beyond 35 weeks of gestation, manifested by a subnormal level of consciousness or seizures, and often accompanied by difficulty with initiating and maintaining respiration and depression of tone and reflexes. This expanded clinical definition must be put into use based on measures that can be reliably and accurately implemented by trained staff. The first mandatory step in an assessment of neonatal encephalopathy is to confirm whether a specific infant meets the case definition.

In confirmed cases of neonatal encephalopathy, the following assessment will determine the likelihood that an acute peripartum or intrapartum event was a contributor. This list is based on the premise that neonatal

Neonatal Encephalopathy and Neurologic Outcome, Second Edition, was developed by the Task Force on Neonatal Encephalopathy: Mary E. D'Alton, MD, Chair; Gary D.V. Hankins, MD, Vice Chair; Richard L. Berkowitz, MD; Jessica Bienstock, MD, MPH; Alessandro Ghidini, MD; Jay Goldsmith, MD; Rosemary Higgins, MD; Thomas R. Moore, MD; Renato Natale, MD; Karin B. Nelson, MD; Lu-Ann Papile, MD; Donald Peebles, MD; Roberto Jose Romero, MD; Diana Schendel, PhD; Catherine Yvonne Spong, MD; Richard N. Waldman, MD; Yvonne Wu, MD, MPH; and the American College of Obstetricians and Gynecologists' staff: Gerald F. Joseph Jr, MD; Debra Hawks, MPH; Alyssa Politzer, MA; Chuck Emig, MA; and Kelly Thomas.

Signes neonatals consistents amb un esdeveniment agut peripart o intrapart (I)

1.- Un test de Apgar inferior a 5 als 5'

2.- Acidosi de la arteria umbilical fetal

- pH de arteria umbilical fetal < 7.0 o DB ≥ 12 mmol/L

Quin es el risc de EHI i PC dels nadons amb pH de arteria umbilical < 7.00?

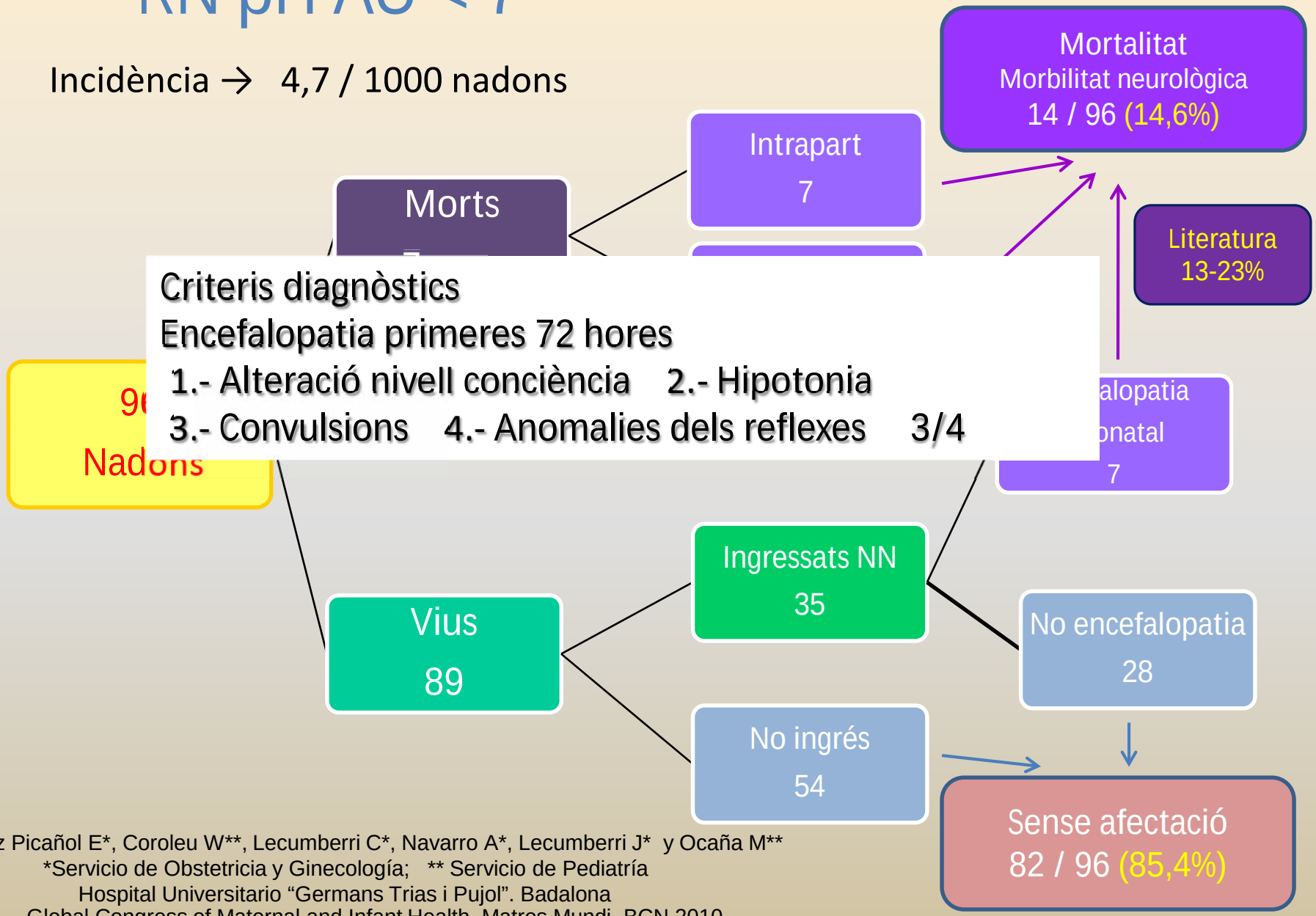
3.- Evidència de lesions cerebrals agudes per Ressonància magnètica

- *Practicada les primeres 24-96 h pot ajudar a establir el moment de la noxa cerebral*
- *Practicada als 10 dies (entre el 7-21), té valor pronòstic.*

4.- Presència de una fallida multiorgànica.

RN pH AU < 7

Incidència → 4,7 / 1000 nadons



Pérez Picañol E*, Coroleu W**, Lecumberri C*, Navarro A*, Lecumberri J* y Ocaña M**
*Servicio de Obstetricia y Ginecología; ** Servicio de Pediatría
Hospital Universitario "Germans Trias i Pujol". Badalona
Global Congress of Maternal and Infant Health. Matres Mundi. BCN 2010

RN pH AU<7

Dels 96 nadons amb un pH < 7 de arteria umbilical :
7 desenvoluparen una Encefalopatia (7,3%),
2 desenvoluparen una Paràlisi Cerebral (2%).

Entre els nadons a terme (N:75) vam observar 1 cas de Paràlisi Cerebral (1,3%), que podem considerar com el risc teòric per aquests nadons.

- Paràlisi cerebral: 1 en RN a terme (1,3%)
  1 en RN preterme (4,7%)

Ret				tard
greu	moderat	moderat	lleu	DPM
3	1	1	1	1

2 paràlisis cerebrals

DPM: desenvolupament maduratiu

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Signe b un esdever part (II)

1.- Un esdevenime mediatament abans o durant el p

- Ruptura
- DPPNI gr
- Prolapse
- Embòlia mia maternes.
- Shock ca
- Hemorrà

2.- RCTG compatib rapart.

- S'ha de c inici dels que apareixen al llarg d

- *Convers* *ia III.*
- Aparició e silent amb desacceleracions r

3.- Absència d'altre sibles causes.

NICHD criteria for category I, II, and III FHR tracings

Category I
All of the following criteria must be present. Tracings meeting these criteria are predictive of normal fetal acid-base balance at the time of observation.
Baseline rate: 110 to 160 bpm
Moderate baseline FHR variability
No late or variable decelerations
Early decelerations may be present or absent
Accelerations may be present or absent
Category III
Category III tracings are predictive of abnormal fetal acid-base status at the time of observation. Prompt evaluation is indicated and most parturients will require expeditious intervention, such as provision of supplemental oxygen, change in position, treatment of hypotension, and discontinuation of any uterotonic drugs being administered. Category III tracings include either (1) or (2) below.
(1) Absent baseline FHR variability and any of the following:
▪ Recurrent late decelerations
▪ Recurrent variable decelerations
▪ Bradycardia
(2) Sinusoidal pattern
Category II
FHR tracing does not meet criteria for either category I or III and is considered indeterminate.

NICHD: National Institute of Child Health and Human Development; FHR: fetal heart rate; bpm: beats per minute.

Data from: Macones GA, Hankins GD, Spong CY, et al. The 2008 National Institute of Child Health and Human Development Workshop Report on Electronic Fetal Monitoring: Update on Definitions, Interpretation, and Research Guidelines. Obstet Gynecol 2008; 112:661.

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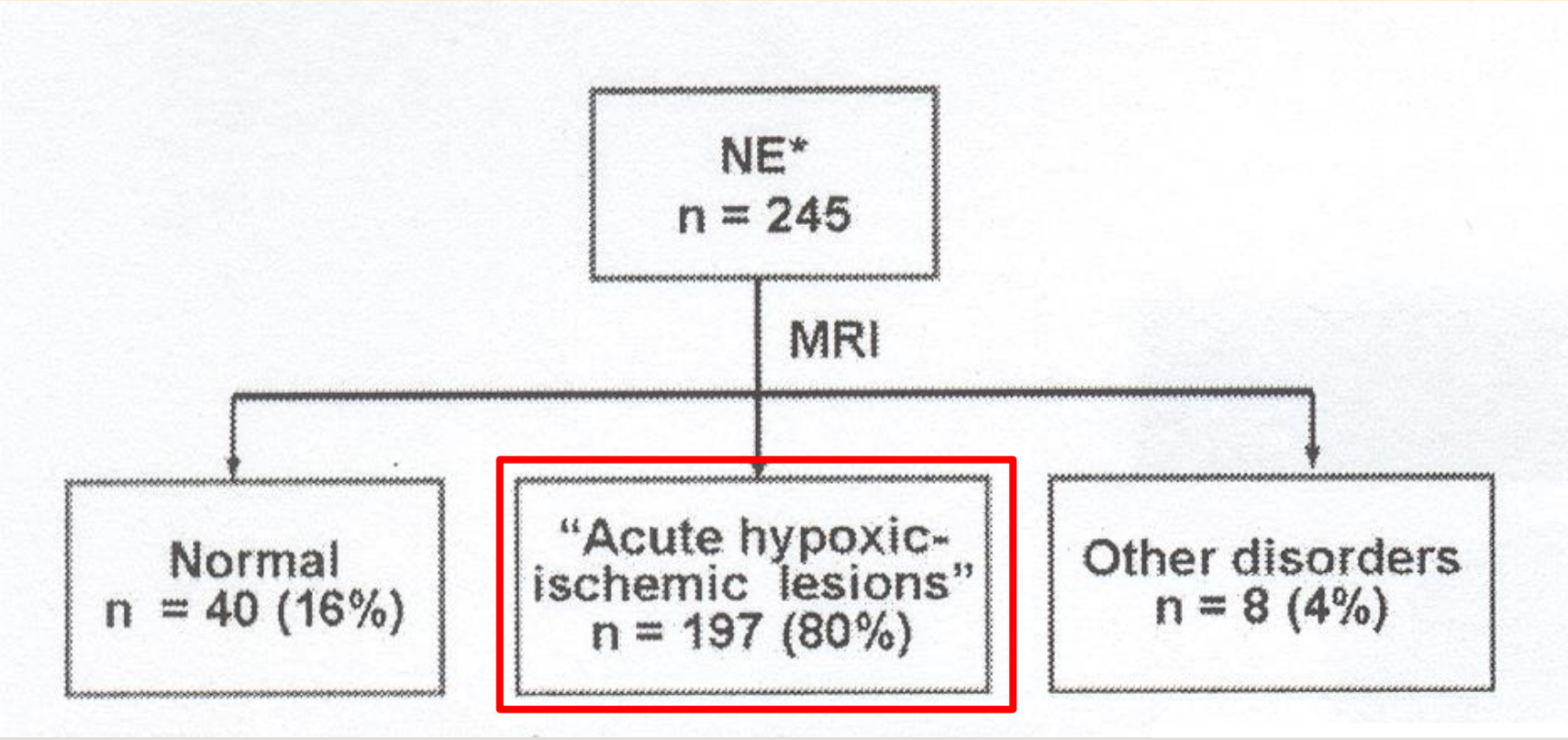
Neonatal Encephalopathy: An Inadequate Term for Hypoxic–Ischemic Encephalopathy

Joseph J. Volpe, MD

This Point of View article addresses neonatal encephalopathy (NE) presumably caused by hypoxia–ischemia and the terminology currently in wide use for this disorder. The nonspecific term NE is commonly utilized for those infants with the clinical and imaging characteristics of neonatal hypoxic–ischemic encephalopathy (HIE). Multiple magnetic resonance imaging studies of term infants with the clinical setting of presumed hypoxia–ischemia near the time of delivery have delineated a topography of lesions highly correlated with that defined by human neuropathology and by animal models, including primate models, of hypoxia–ischemia. These imaging findings, coupled with clinical features consistent with perinatal hypoxic–ischemic insult(s), warrant the specific designation of neonatal HIE.

ANN NEUROL 2012;72:156–166

UN NOU PARADIGMA MENYS FAVORABLE PER EL OBSTETRA



3 dels següents criteris d'inclusió

1.- Desacceleracions de la FCF en el part o meconi
2.- pH arteria umbilical < 7.10
3.- Retard en el inici de la respiració
4.- Apgar < 7 als 5'
5.- Fracàs multiorgànic

Cowan et al. Lancet 2003; 361: 736-742

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Medical and legal implications for necessary requirements to diagnose damaging hypoxic-ischemic encephalopathy leading to later cerebral palsy

Roger K. Freeman, MD

ROL IMPORTANT DE LA INFECCIÓ (CORIOAMNIONITIS), FEBRE INTRAPART I/O FEBRE NEONATAL PERLLONGADA.

Paper de la resposta inflamatòria fetal com a factor predisposant de la PC a través de les citoquines proinflamatòries

Impo uir el

RM i F ent no poder correl

1.- Una primera exploració practicada entre les 24 i 96 hores proporcionarà la guia més útil per establir el moment potencial del insult cerebral.

2.- Una segona exploració practicada entre els dies 10 i 21 permetria establir la natura i extensió de la lesió i per tant, el pronòstic.

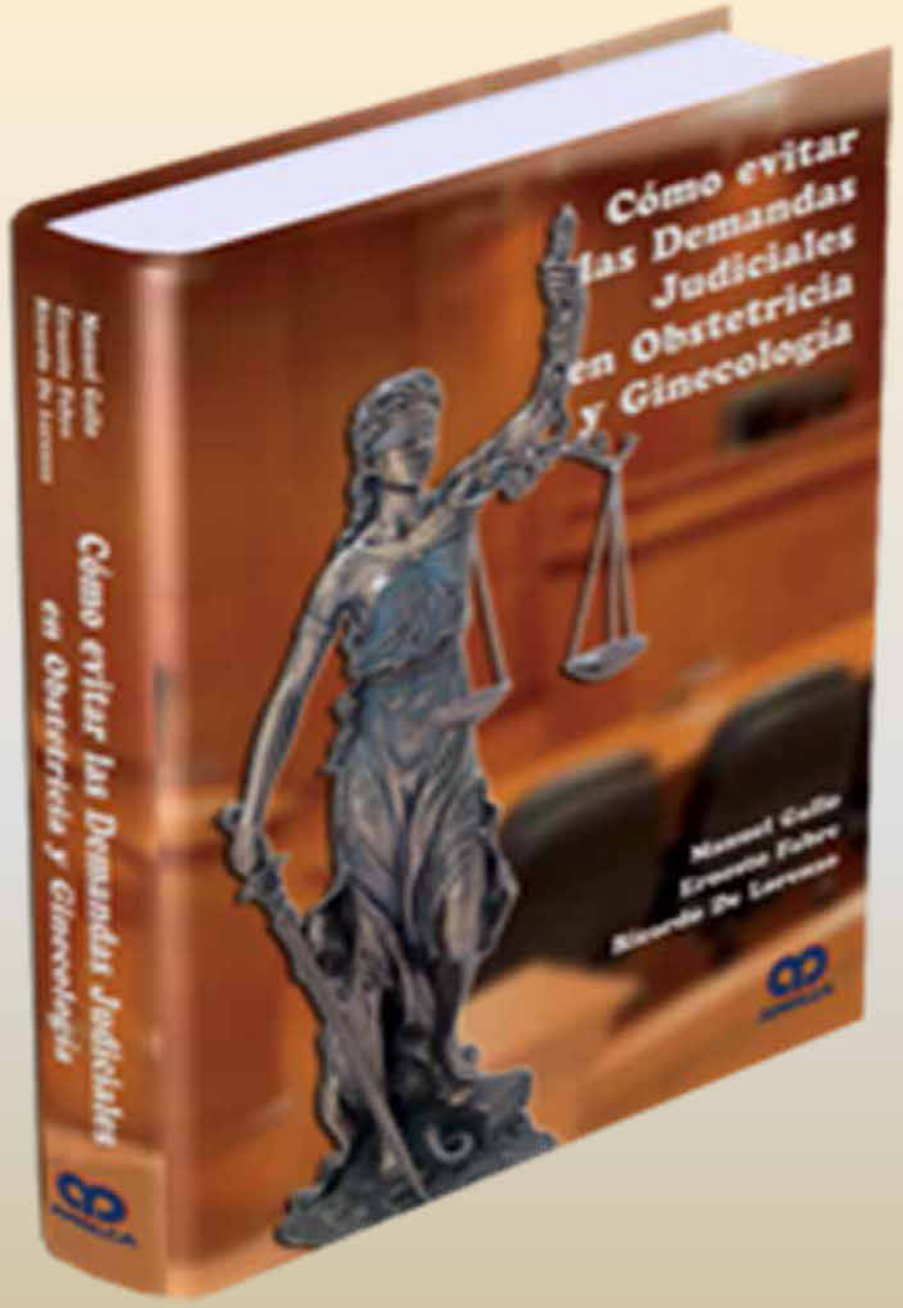
Factors que suggereixen una causa de paràlisis cerebral diferent d'una hipòxia aguda intrapart

§ Dèficit de base menor de 12 mmol/l o pH superior a 7,00 en artèria umbilical
§ Nounats amb malformacions congènites majors o metabolopaties
§ Infecció del SNC o sistèmica
§ Evidència precoç d'anormalitats neurològiques de llarga evolució per estudis d'imatge (ej. Ventriculomegàlia, porencefalia, encefalomalacia multiquística)
§ Presència de creixement intrauterí retardat
§ Absència o disminució de la variabilitat del RCTG des del inici del part o del registre
§ Microcefalia (circumferència cranial inferior al percentil 30)
§ Corioamnionitis severa
§ Trastorns congènits de la coagulació en el nadó
§ Presència d'altres factors de risc: prematuritat (< 34 s), gestació múltiple, malaltia autoimmune
§ Presència de factors de risc postnatal de paràlisi cerebral: encefalitis, hipotensió sostinguda, hipòxia secundària a destret respiratori
§ Un germà amb paràlisi cerebral del mateix tipus

*Alastair MacLennan for the International Cerebral Palsy Task Force**
BMJ volume 319 16 october 1999 www.bmj.com

50 ANYS DE MONITORITZACIÓ CARDIACA FETAL COM A MÈTODE DE CONTROL DEL

CONVINDRIA INICIAR ÀREAS DE REÇERCA AMB L'OBJECTIU DE MONITORITZAR EL CERVELL FETAL D'UNA MANERA MÉS DIRECTA QUE A TRAVÈS DE LA MONITORITZACIÓ DEL COR FETAL


Cómo evitar las Demandas Judiciales en Obstetricia y Ginecología
Manuel Gallo
Ernesto Febre
Ricardo De Lorenzo

Original Article

Journal of Child Neurology
2015, Vol. 30(7) 828-841

DOI: 10.1177/0883073814543306
jcn.sagepub.com
SAGE

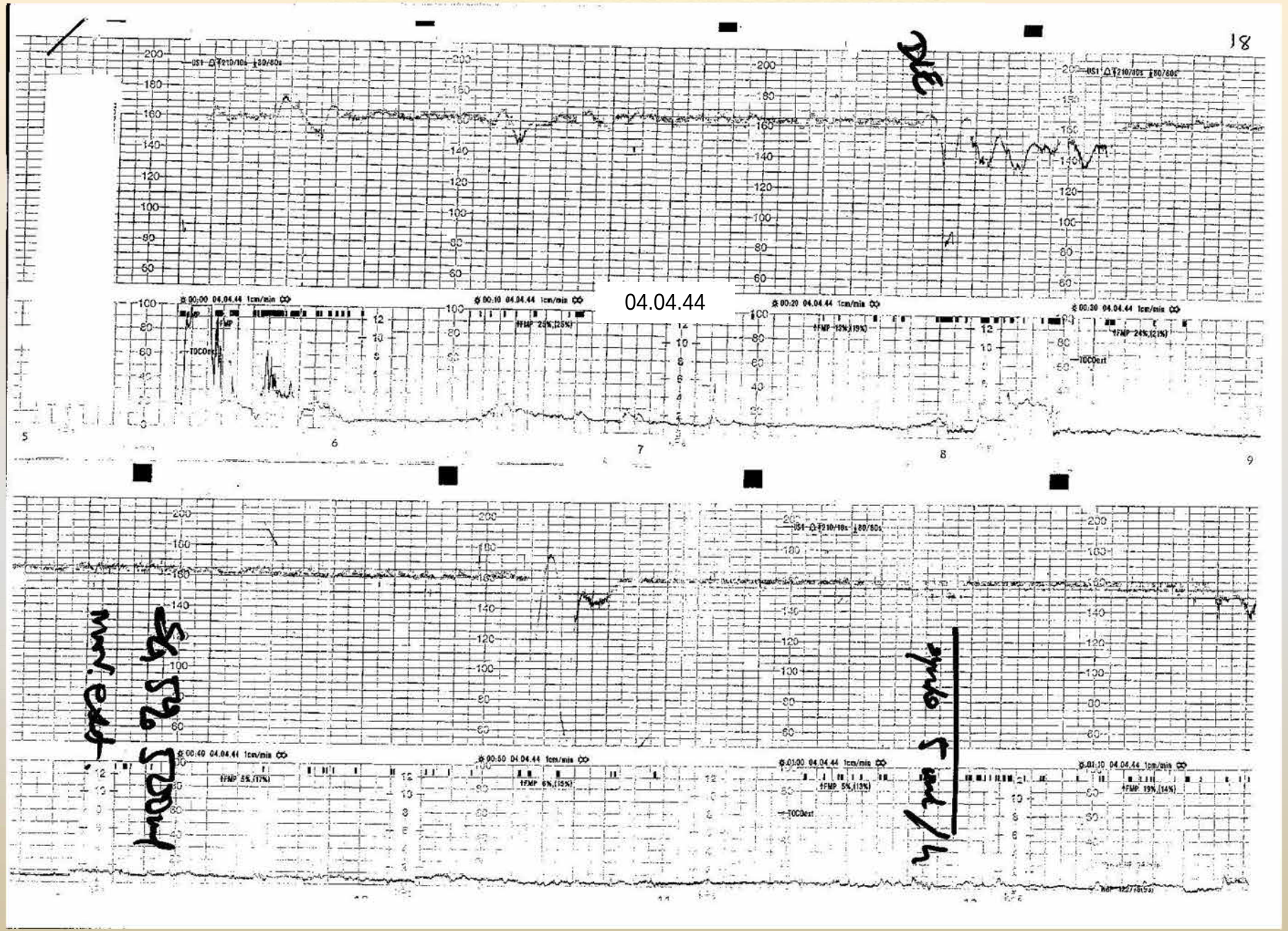
Cerebral Palsy Litigation: Change Course or Abandon Ship

Thomas P. Sartwelle, BBA, LLB[1] and James C. Johnston, MD, JD[2]

Abstract

The cardinal driver of cerebral palsy litigation is electronic fetal monitoring, which has continued unabated for 40 years. Electronic fetal monitoring, however, is based on 19th-century childbirth myths, a virtually nonexistent scientific foundation, and has a false positive rate exceeding 99%. It has not affected the incidence of cerebral palsy. Electronic fetal monitoring has, however, increased the cesarian section rate, with the expected increase in mortality and morbidity risks to mothers and babies alike. This article explains why electronic fetal monitoring remains endorsed as efficacious in the worlds' labor rooms and courtrooms despite being such a feeble medical modality. It also reviews the reasons professional organizations have failed to condemn the use of electronic fetal monitoring in courtrooms. The failures of tort reform, special cerebral palsy courts, and damage limits to stem the escalating litigation are discussed. Finally, the authors propose using a currently available evidence rule—the *Daubert* doctrine that excludes "junk science" from the courtroom—as the beginning of the end to cerebral palsy litigation and electronic fetal monitoring's 40-year masquerade as science.

ALGUNES RECOMANACIONS



GRACIES PER LA SEVA ATENCIÓ

Emilio Pérez Picañol, 2016
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