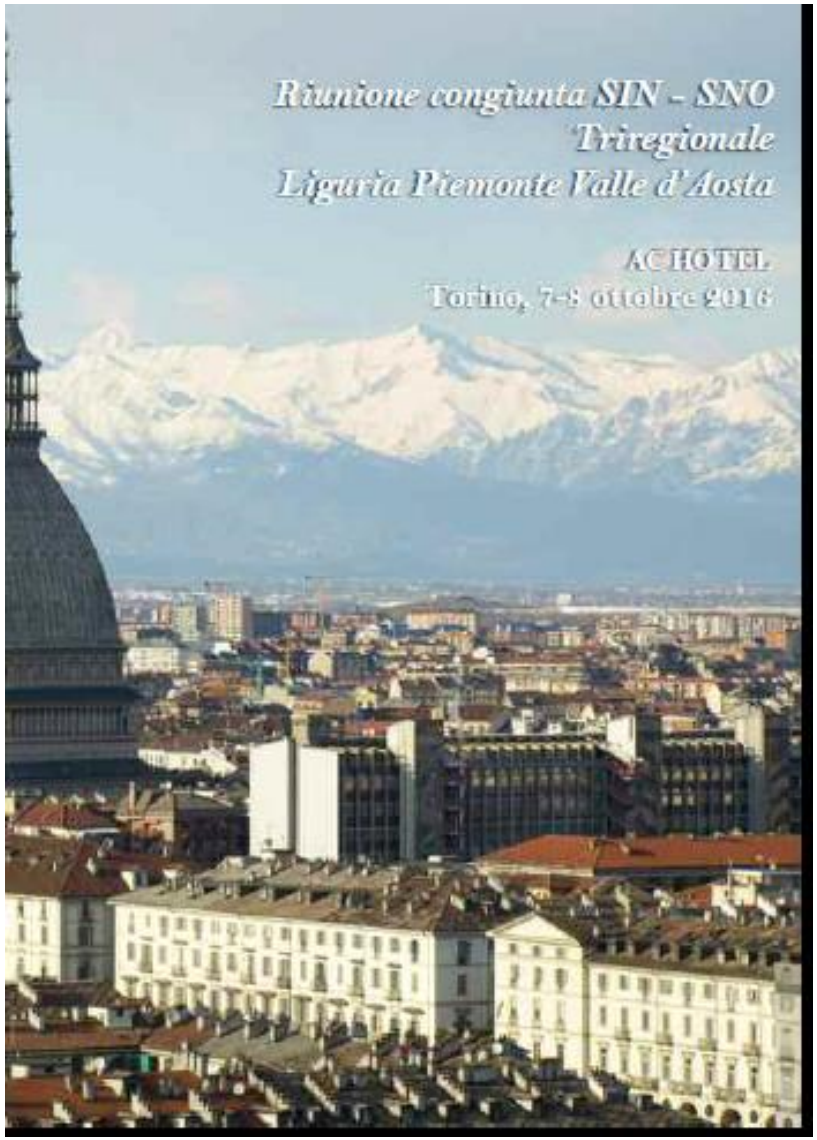
Sindrome di Guillain Barrè:

Classificazione
e
diagnosi differenziale



Dott.ssa Luana Benedetti

DINOGMI, Università di Genova

Responsabile Ambulatorio

Malattie Rare Neuromuscolari, La Spezia

Guillain-Barrè Syndrome (GBS)

In **1916**, Guillain, Barrè and Strohl described a previously unrecognized type of polyneuritis characterized by definite changes in the cerebrospinal fluid. The essential of the disease were *widespread flaccid paralysis*, *loss of tendon reflexes*, preservation of cutaneous reflexes, conservation of idiomuscular contraction on percussion, *minimal changes in the electrical reactions of the muscle and nerves*, muscle tenderness, paresthesias with little disturbances of objective sensibility and, most specifically, *increase in the protein content of the CSF* which was not accompanied by a proportionate degree of pleocytosis. Preceding infection; progression <4 weeks.

Dr. Guillain, G.

Dr. Barrè, J.A.

Dr. Strohl, A.

G. Guillain, J. Barré, and A. Strohl, “Sur un syndrome de radiculo-nevrite avec hyperalbuminose du liquide cephalorachidien sans reaction cellulaire. Remarques sur les characteres clinique et graphique des reflexes tendinaux,” *Bulletins et Memories de la Societe Medicale des Hopitaux de Paris*, vol. 40, pp. 1462–1470, 1916.

Criteri Diagnostici

(from Asbury A.K. And Cornblath DR. Ann Neurol, 1990,

modified by Bosh E.P. and Smith B.E., in Neurology in Clinical Practice, eds Bradley W.G. et al, 2000)

- Criteri clinici indispensabili

  Ipostenia progressiva in almeno 2 arti

  Areflessia

- Criteri clinici che supportano la diagnosi

  Progressione (4 settimane)

  Disturbi simmetrici

  Deficit sensitivi modesti

  Nervi cranici coinvolti

  Miglioramento dopo 2-4 weeks dal termine della progressione dei sintomi

  Disfunzioni autonomiche

  Assenza di febbre all'esordio

- Criteri di laboratorio che supportano la diagnosi

  Elevate CSF proteine, <10 cells/μL

  ENG: 80% rallentamento VDC e blocchi di conduzione motoria

Nel 2016……..

Classificazione GBS

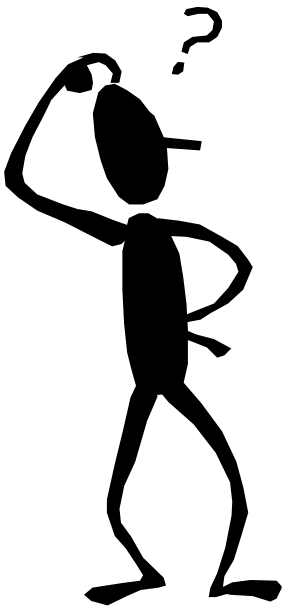
Classificazione
in base alla clinica

Classificazione
in base alla elettrofisiologia

Perché è importante saper distinguere la clinica e le sue varianti ?

L'ENG ed il CSF non sono dirimenti nelle fasi precoci di malattia

- CSF normale: >50% pz nella prima settimana dall'esordio sintomi
- ENG non utile: 40% pz nella prima settimana dall'esordio sintomi



La diagnosi e soprattutto il trattamento non DEVONO essere ritardati

Classificazione in base alla clinica

OPINION

Guillain–Barré and Miller Fisher syndromes—new diagnostic classification

Benjamin R. Wakerley, Antonino Uncini and Nobuhiro Yuki for the GBS Classification Group

Wakerley, B. R. *et al. Nat. Rev. Neurol.* **10**, 537–544 (2014); published online 29 July 2014; corrected online 7 October 2014; doi:10.1038/nrneurol.2014.138

Box 1 | The GBS Classification Group

- Shahram Attarian, Centre Hospitalier de la Timone, Marseille, France
- Jong Seok Bae, Hallym University College of Medicine, Seoul, South Korea
- Amilton A. Barreira, University of São Paulo, São Paulo, Brazil
- Yee-Cheun Chan, National University Hospital, Singapore
- Alain Créange, Université Paris-Est Créteil, Créteil, France
- Sung-Tsang Hsieh, National Taiwan University Hospital, Taipei, Taiwan
- Badrul Islam, International Centre for Diarrhoeal Disease Research, Bangladesh, Dhaka, Bangladesh
- Meena A. Kannan, Nizam's Institute of Medical Sciences, Hyderabad, India
- Nobuo Kohara, Kobe City Medical Centre General Hospital, Kobe City, Japan
- Norito Kokubun, Dokkyo Medical University, Tochigi, Japan
- Jean-Pascal Lefaucheur, Université Paris-Est Créteil, Créteil, France
- Osvaldo Nascimento, Federal Fluminense University, Rio de Janeiro, Brazil
- Nortina Shahrizaila, University of Malaya, Kuala Lumpur, Malaysia
- Umapathi Thirugnanam, National Neuroscience Institute, Singapore
- Antonino Uncini, University G. d'Annunzio, Chieti, Italy
- Peter Van den Bergh, University Hospitals St-Luc, Brussels, Belgium
- Benjamin R. Wakerley, Gloucestershire Royal Hospital, Gloucester, UK
- Einar Wilder-Smith, National University Hospital, Singapore
- Rawiphan Witoonpanich, Ramathibodi Hospital, Bangkok, Thailand
- Nobuhiro Yuki, National University of Singapore, Singapore

OPINION

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GBS “classica”:
paralisi flaccida acuta ai 4 arti

Sottotipi di GBS:
“forme localizzate”

MFS “classica”:
Acuta atassia, areflessia, oftalmoplegia

Sottotipi di MFS:
- quadri clinici “more extensive”: ipersonnia
- quadri clinici “less extensive”: forme incomplete

Table 1 | Clinical features of GBS, MFS and their subtypes

Category	Clinical features		
	Pattern of weakness	Ataxia	Hypersomnolence
GBS			
Classic GBS	Four limbs	No or minimal	No
Pharyngeal–cervical–brachial weakness*	Bulbar, cervical and upper limbs	No	No
Acute pharyngeal weakness‡	Bulbar	No	No
Paraparetic GBS*	Lower limbs	No	No
Bifacial weakness with paraesthesias*	Facial	No	No
MFS			
Classic MFS	Ophthalmoplegia	Yes	No
Acute ophthalmoparesis§	Ophthalmoplegia	No	No
Acute ataxic neuropathy§	No weakness	Yes	No
Acute ptosis§	Ptosis	No	No
Acute mydriasis§	Paralytic mydriasis	No	No
BBE‖	Ophthalmoplegia	Yes	Yes
Acute ataxic hypersomnolence¶	No weakness	Yes	Yes

GBS MFS

in comune:

- **Antecedente infezione (3 gg - 6 sett prima)**
- **Decorso monofasico**
  (intervallo tra esordio e nadir ipostenia 12h-28 gg seguito da plateau)
- **(Areflessia)……10% ROT normali o vivaci**

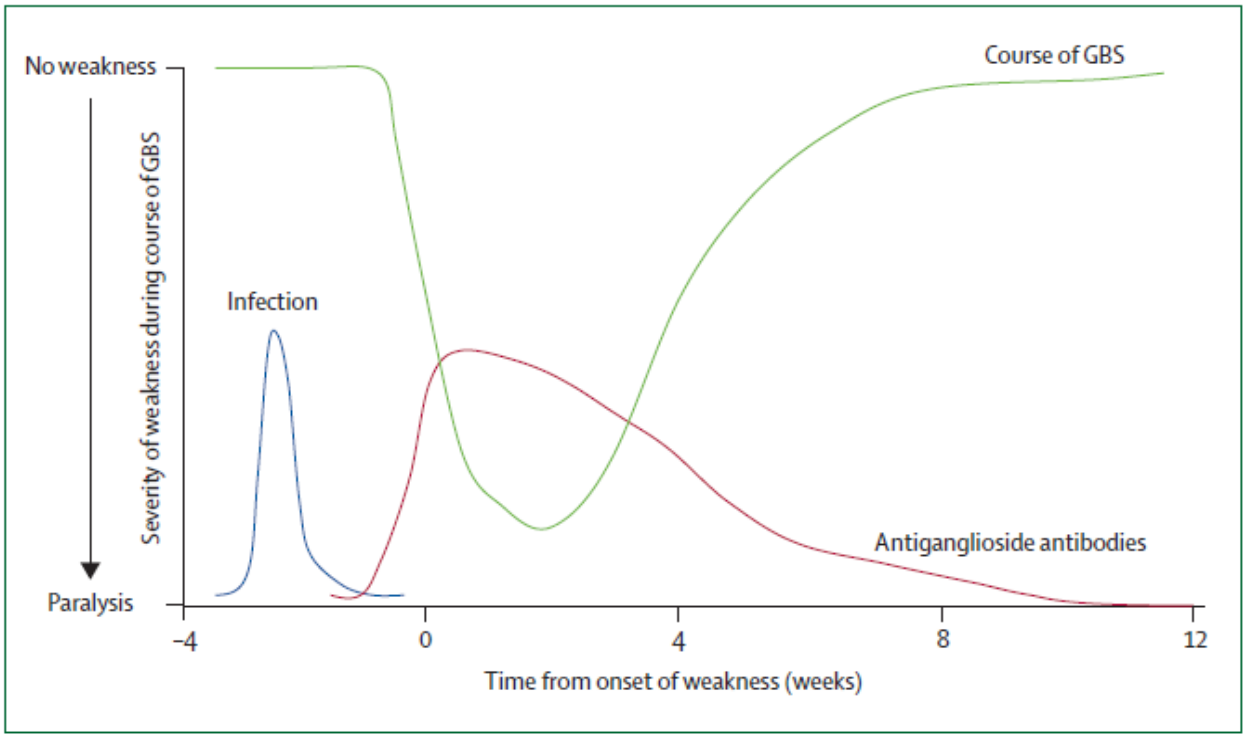

Figure 1: Relation between infections, antiganglioside antibodies, and clinical course of GBS

van Doorn PA et al., Lancet Neurol 2008;7: 939-950

GBS «classica» e forme «localizzate»

Classic GBS	Weakness* and areflexia/ hyporeflexia in all four limbs	Weakness usually starts in the legs and ascends but may start in the arms Weakness may be mild, moderate or complete paralysis Cranial-nerve-innervated muscles or respiratory muscles may be involved Muscle stretch reflexes may be normal or exaggerated in 10% of cases
Pharyngeal–cervical–brachial weakness	Oropharyngeal, neck and arm weakness*‡ and arm areflexia/ hyporeflexia Absence of leg weakness	Absence of certain features indicates incomplete pharyngeal–cervical–brachial weakness: patients without arm and neck weakness have 'acute oropharyngeal palsy'; patients without pharyngeal palsy have 'acute cervicobrachial weakness' Some leg weakness may be present, but oropharyngeal, neck and arm weakness should be more prominent Presence of additional features indicates overlap with other GBS variants: ataxia with ophthalmoplegia suggests overlap with MFS; ataxia without ophthalmoplegia suggests overlap with acute ataxic neuropathy; ataxia, ophthalmoplegia and disturbed consciousness suggests overlap with BBE
Paraparetic GBS	Leg weakness* and leg areflexia/ hyporeflexia Absence of arm weakness	Typically, bladder function is normal and there is no well-defined sensory level
Bifacial weakness with paraesthesias	Facial weakness* and limb areflexia/hyporeflexia Absence of ophthalmoplegia, ataxia and limb weakness	In some patients, limb paraesthesias may be absent and muscle stretch reflexes may be normal

Wakerley R, Uncini A. *Nat Rev. Neurol.* 2014

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Wakerley R, Uncini A. *Nat Rev. Neurol.* 2014

Journal of Neurology, Neurosurgery, and Psychiatry 1994;57:1121–1123

SHORT REPORT

Two species of antiganglioside antibodies in a patient with a pharyngeal-cervical-brachial variant of Guillain-Barré syndrome

A patient with a pharyngeal-cervical-brachial variant of Guillain-Barré syndrome had anti-GT1a and anti-GD1a antibodies (IgG) in the serum. The activ-

Particolarmente espresso nel nervo glossofaringeo e vago

Journal of Neurology, Neurosurgery, and Psychiatry 1996;61:649–651

SHORT REPORT

Acute oropharyngeal palsy is associated with antibodies to GQ1b and GT1a gangliosides

C P O'Leary, J Veitch, W F Durward, A M Thomas, J H Rees, H J Willison

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Wakerley R, Uncini A. *Nat Rev. Neurol.* 2014

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Sufficiente per far diagnosi!

Wakerley R, Uncini A. *Nat Rev. Neurol.* 2014

MFS «classica» e sottotipi

MFS	Ophthalmoplegia, ataxia*‡ and areflexia/hyporeflexia Absence of limb weakness# and hypersomnolence	Absence of certain features indicates incomplete MFS: patients without ataxia have 'acute ophthalmoparesis'; patients without ophthalmoplegia have 'acute ataxic neuropathy' Presence of a single feature indicates incomplete MFS: ptosis suggests 'acute ptosis'; mydriasis suggests 'acute mydriasis'
BBE	Hypersomnolence and ophthalmoplegia and ataxia*‡ Absence of limb weakness#	Patients without ophthalmoplegia have the incomplete form of BBE known as 'acute ataxic hypersomnolence'

Sottotipi o forme incomplete

Acute ophthalmoparesis§	Ophthalmoplegia
Acute ataxic neuropathy§	No weakness
Acute ptosis§	Ptosis
Acute mydriasis§	Paralytic mydriasis

«Ataxic GBS» (forma incompleta di MFS)
e
«acute sensory ataxic neuropathy» (variante GBS)

= ***Acute Ataxic Neuropathy***

Ab anti-GQ1b 83% MFS
68% BBE
particolarmente espressi nn oculomotori
formazione reticolare

Quadri clinici “overlap” o “sequenziali”

Relationship of bacterial strains to clinical syndromes of *Campylobacter*-associated neuropathies

K. Kimoto, MD*; M. Koga, MD, PhD*; M. Odaka, MD, PhD; K. Hirata, MD, PhD; M. Takahashi, DVM, PhD; J. Li, PhD; M. Gilbert, PhD; and N. Yuki, MD, PhD

MFS che sviluppano tetraplegia

Two species of ant
patient with a phar
of Guillain-Barré s

Kouichi Mizoguchi, Asako Hase,
Yoshirou Nishimura, Fumitoshi I

«SPECTRUM OF GBS»

-cervico-brachiale
luppa ipostenia AAII

Eur Neurol 2002;48:53–54

Guillain-Barré Syndrome Presenting Pharyngeal-Cervical-Brachial Weakness in the Recovery Phase

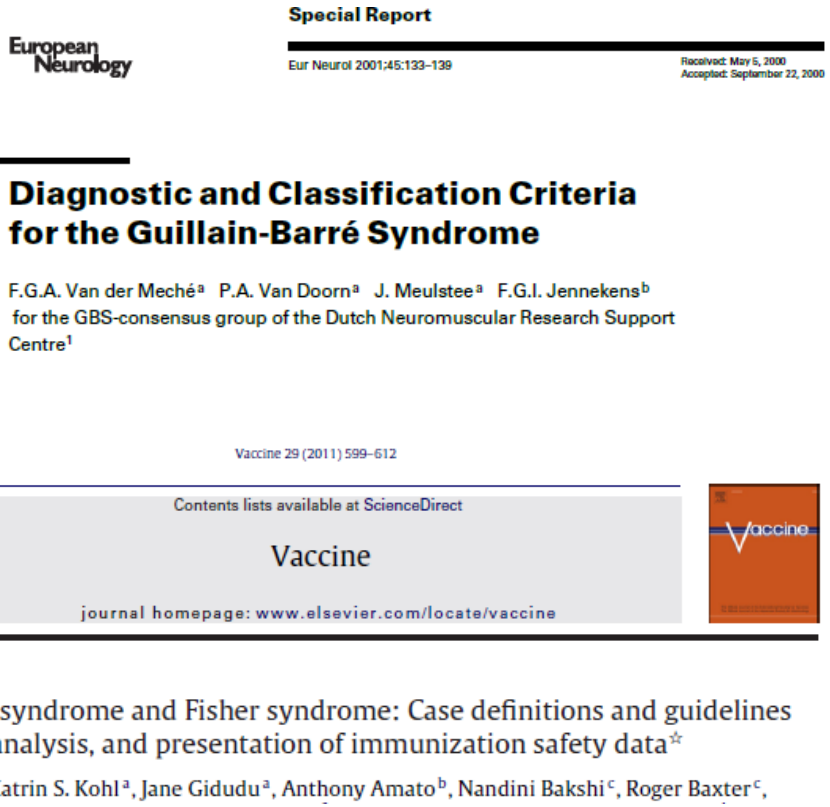
Yumiko Miura[a], *Keiichiro Susuki*[b], *Nobuhiro Yuki*[b], *Mitsuyoshi Ayabe*[a], *Hiroshi Shoji*[a]

GBS classica
che nella fase di recupero
sviluppa una ipostenia
faringo-cervico-brachiale

Acute sensory small-fibre neuropathy
Acute sensory and autonomic neuropathy
Acute pandysautonomia

- Sono condizioni autoimmuni post-infettive simili alla GBS

- ma in linea con altri autori……



European Neurology

Special Report

Eur Neurol 2001;45:133–139

Received: May 5, 2000
Accepted: September 22, 2000

Diagnostic and Classification Criteria for the Guillain-Barré Syndrome

F.G.A. Van der Meché[a] P.A. Van Doorn[a] J. Meulstee[a] F.G.I. Jennekens[b]
for the GBS-consensus group of the Dutch Neuromuscular Research Support Centre[1]

Vaccine 29 (2011) 599–612

Contents lists available at ScienceDirect

Vaccine

journal homepage: www.elsevier.com/locate/vaccine

ELSEVIER

Guillain–Barré syndrome and Fisher syndrome: Case definitions and guidelines for collection, analysis, and presentation of immunization safety data☆

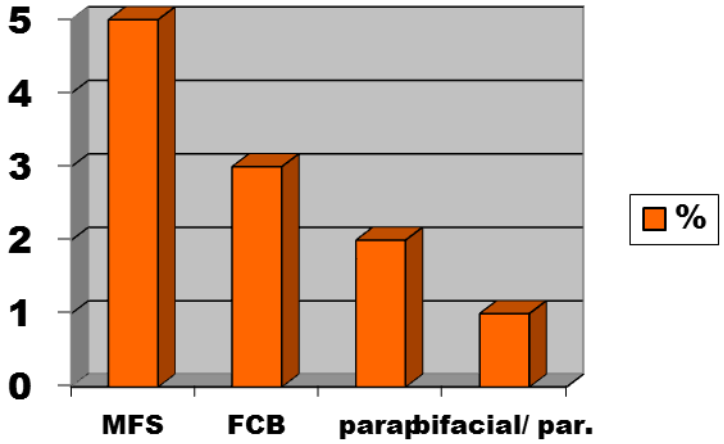
James J. Sejvar[a,*], Katrin S. Kohl[a], Jane Gidudu[a], Anthony Amato[b], Nandini Bakshi[c], Roger Baxter[c],

- **non vengono incluse** in questa nuova classificazione

Frequenza delle varianti/sottotipi

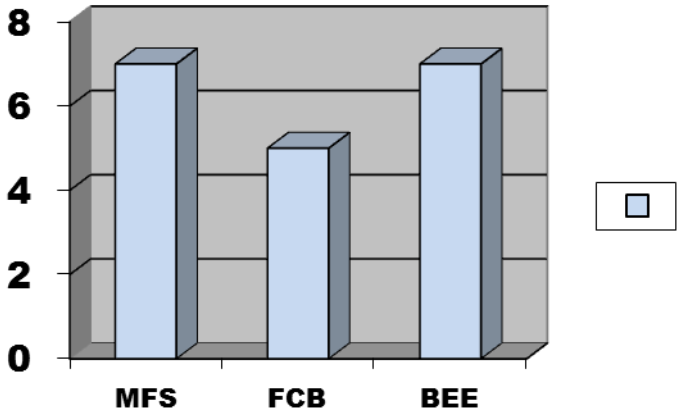
Ropper 1990

- Studio prospettico-USA
- **250** GBS



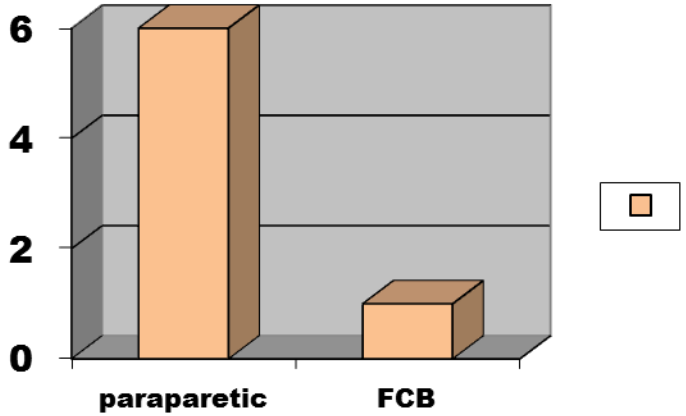
Lin 2012

- Studio retrospettivo-Taiwan
- **43** GBS



Fokke 2014

- Netherlands
- **500** GBS

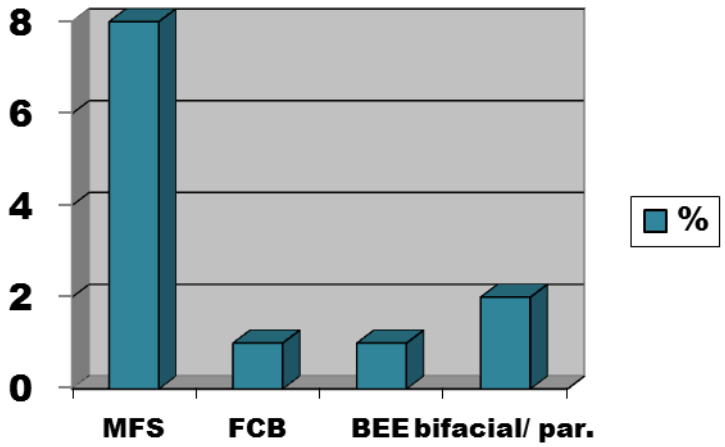


GUILLAIN-BARRE' SYNDROME:
A THIRTEEN-YEARS RETROSPECTIVE STUDY IN PROVINCE OF LA SPEZIA

Benedetti L, Beronio A, Giorli E, Parodi S, Sani C, Derchi A, Amodeo C, Rollandi F, Delia P, Massa F, Mannironi A

ASNP. Mondello 2016

- Studio retrospettivo-La Spezia
- **86** GBS



Varianti < 10%

Classificazione GBS

Classificazione
in base alla clinica

Classificazione
in base all’ elettrofisiologia

Classicamente ……

- **Forme demielinizzanti**: **AIDP**
- **Forme assonali**

  - grave e precoce danno assonale
  - +++ C. Jejuni
  - iperacute, insuff respiratoria < 7 gg
  - prognosi peggiore

AMSAN

AMAN Ab anti GM1

Ab anti GD1a

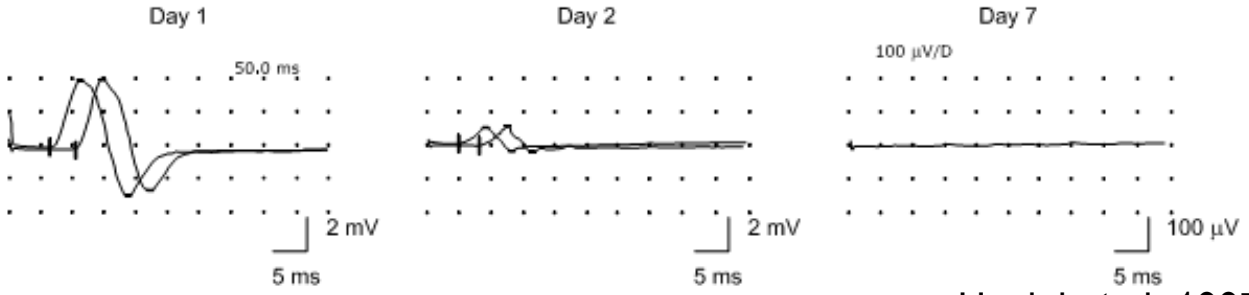
5% Europa

30-47% Cina, Giappone, America centro-sud

Classicamente ……

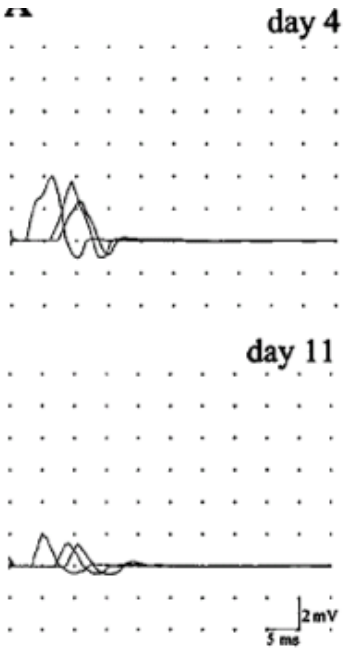
Electrophysiological criteria of AMAN

Median nerve



Uncini et al. 1997

Ulnar nerve



AMAN

(C) Ho *et al.* (1995) [22]

- No evidence of demyelination as defined in (a)
- dCMAP < 80% LLN in at least two nerves

(D) Hadden *et al.* (1998)

- None of the features of demyelination in any nerve as defined in (b) (except one demyelinating feature allowed in one nerve if dCMAP < 10% LLN)
- dCMAP < 80% LLN in at least two nerves

Ora si parla di ……

Review

Nodo-paranodopathy: Beyond the demyelinating and axonal classification in anti-ganglioside antibody-mediated neuropathies

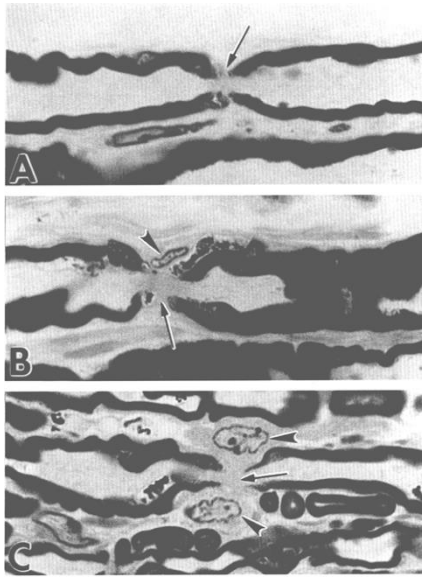
Antonino Uncini [a,*], Keiichiro Susuki [b], Nobuhiro Yuki [c]

Clinical Neurophysiology 124 (2013) 1928–1934

Journal of Neurocytology **25**, 33–51 (1996)

Early nodal changes in the acute motor axonal neuropathy pattern of the Guillain-Barré syndrome

J. W. GRIFFIN[1,2,*], C. Y. LI[3], C. MACKO, T. W. HO[1], S.-T. HSIEH[1], P. XUE[3], F. A. WANG[3], D. R. CORNBLATH[1], G. M. McKHANN[1,5] and A. K. ASBURY[4]

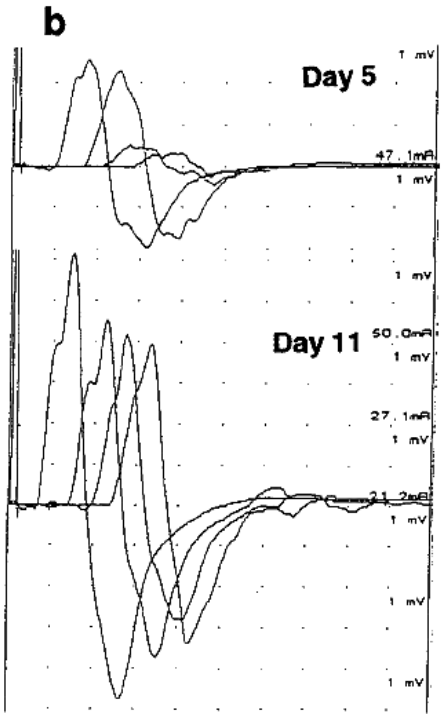


Evidence in humans and animal models point to dysfunction/ disruption of the nodal region as a common pathway in acute, and possibly chronic, neuropathies associated with antibodies to gangliosides. The term nodo-paranodopathy better reflects, in our opinion, the current understanding of the pathophysiology and better systematizes these conditions than the traditional classification into demyelinating or axonal.

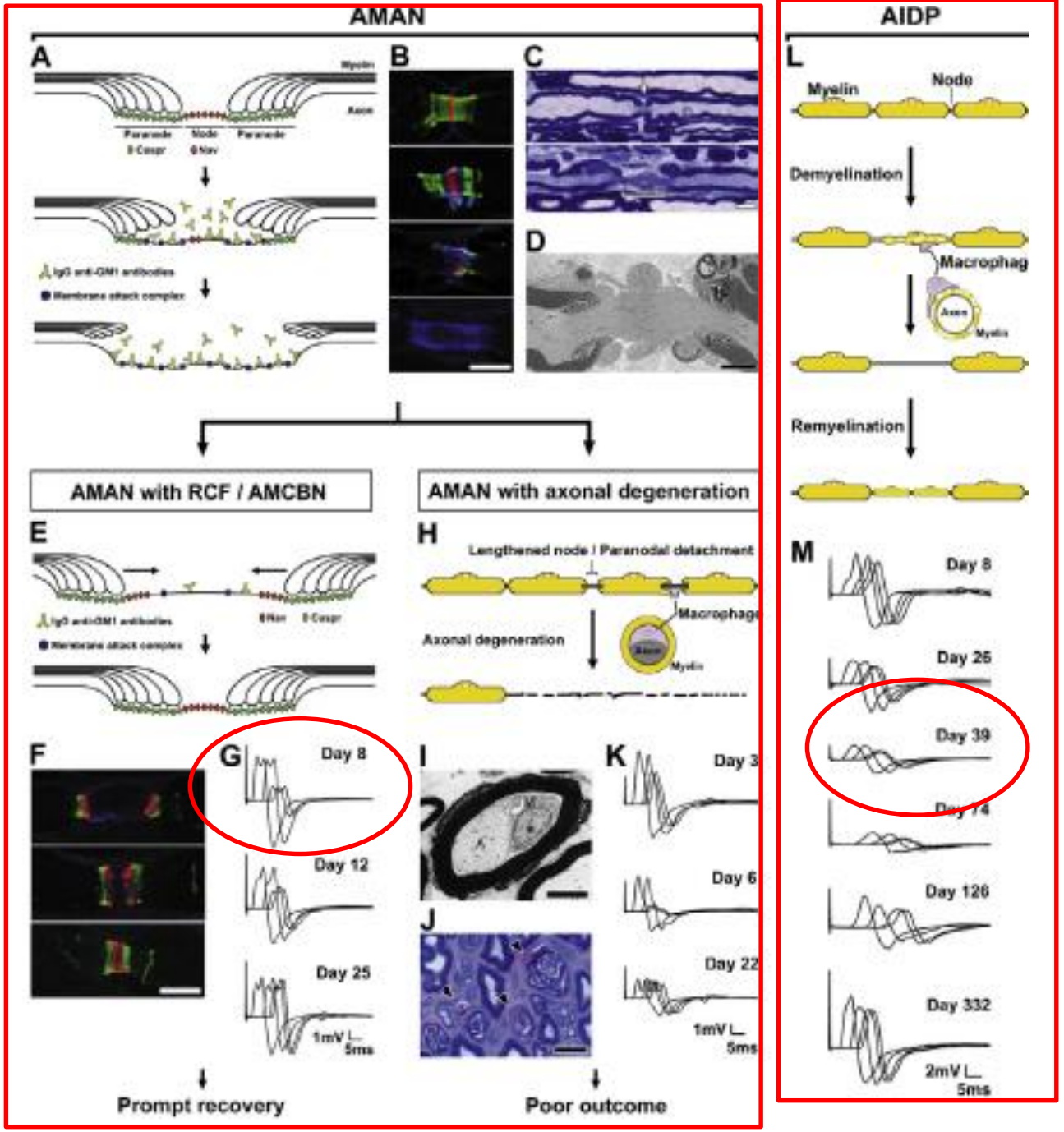
Ann Neurol 1998;44:202–208

IgG Anti-GM1 Antibody Is Associated with Reversible Conduction Failure and Axonal Degeneration in Guillain-Barré Syndrome

Satoshi Kuwabara, MD,* Nobuhiro Yuki, MD, PhD,† Michiaki Koga, MD,† Takamichi Hattori, MD,* Daisuke Matsuura, PhD,‡ Masami Miyake, PhD,‡ and Masatoshi Noda, PhD‡



"Besides axonal degeneration AMAN is characterized by a reversibly reduced safety factor of transmission, possibly due to a reduction of functioning Na-channels at Ranvier nodes"


AMAN
A
B
C
D
IgG anti-GM1 antibodies
Membrane attack complex
AMAN with RCF / AMCBN
AMAN with axonal degeneration
E
H
Lengthened node / Paranodal detachment
Macrophage
Axonal degeneration
Axon
Myelin
F
G
Day 8
Day 12
Day 25
1mV
5ms
I
J
K
Day 3
Day 6
Day 22
1mV
5ms
Prompt recovery
Poor outcome
AIDP
L
Myelin
Node
Demyelination
Macrophag
Remyelination
M
Day 8
Day 26
Day 39
Day 74
Day 126
Day 332
2mV
5ms

CME

Acute motor conduction block neuropathy

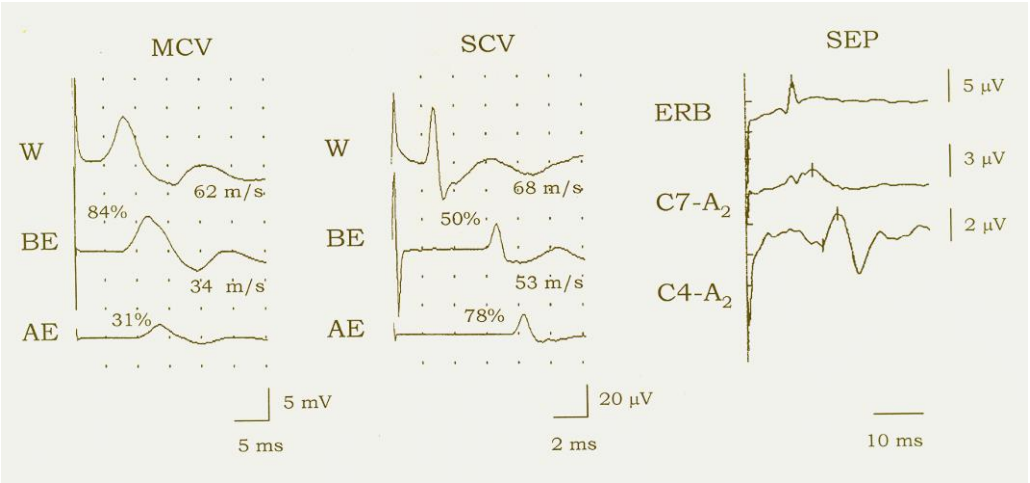
Another Guillain–Barré syndrome variant

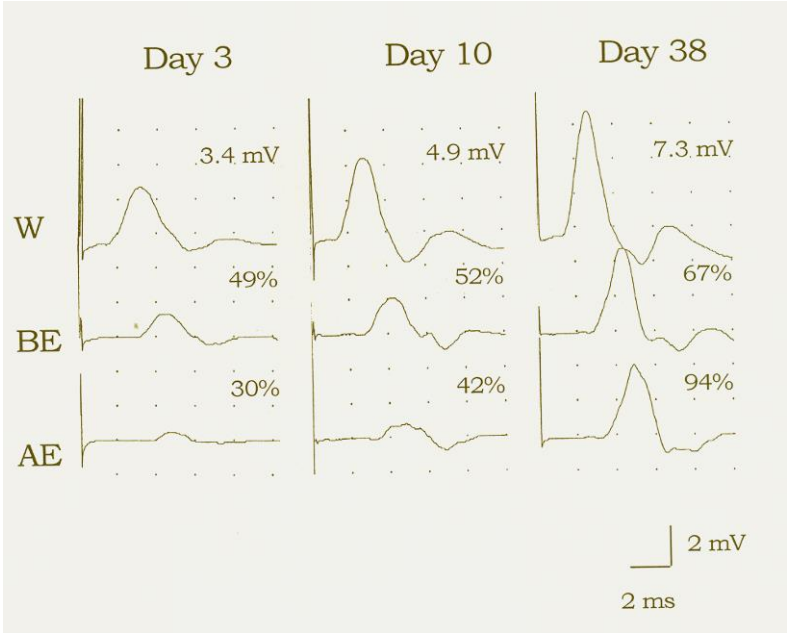
M. Capasso, MD; C.M. Caporale, MD; F. Pomilio, DVM; P. Gandolfi, DSc; A. Lugaresi, MD, PhD; and A. Uncini, MD

NEUROLOGY 2003;61:617–622

ab anti-GM1,-GD1a,-GD1b

Ulnar nerve





Acute Motor Conduction Block Neuropathy is an "arrested" AMAN and it is equivalent to AMAN with reversible conduction failure

Editor's choice
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REVIEW

Nodopathies of the peripheral nerve: an emerging concept

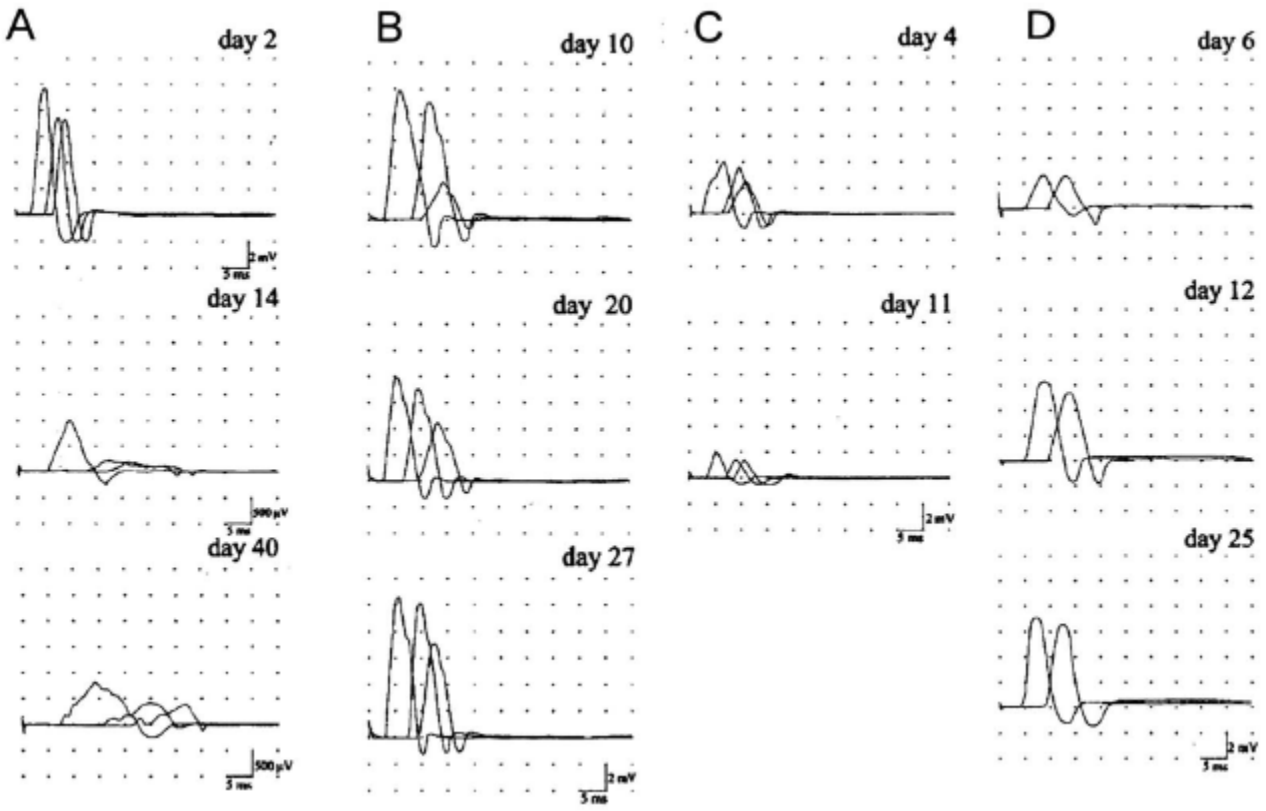
Antonino Uncini,[1] Satoshi Kuwabara[2]

AIDP

AMAN con «RCF»

AMAN con deg.assonale

AMAN con «RCF» distale



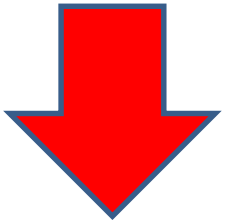
Prompt recovery

Poor outcome

Prompt recovery

Implicazioni pratiche

- RCF is different from demyelinative CB in AIDP that is characterized in the recovery phase by increased CMAP duration due to excessive temporal dispersion
- Not recognizing distal RCF may induce on the basis of a single test to diagnose axonal degeneration and to formulate a bad prognosis
- Recovery in AMAN may be either very rapid and complete or prolonged with poor outcome according to the relative amount of RCF and axonal degeneration in each patient
- The lack of distinction among demyelinating CB and RCF may fallaciously classify axonal GBS as AIDP



Sequential recordings are mandatory for a correct electrodiagnosis

Research paper

Pitfalls in electrodiagnosis of Guillain—Barré syndrome subtypes

Antonino Uncini,[1,2] Claudia Manzoli,[1] Francesca Notturno,[1,2] Margherita Capasso[1]

J Neurol Neurosurg Psychiatry 2010;**81**:1157—1163.

Clinical Neurophysiology 124 (2013) 1456–1459

Contents lists available at SciVerse ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

Two sets of nerve conduction studies may suffice in reaching a reliable electrodiagnosis in Guillain–Barré syndrome

Nortina Shahrizaila[a,*], Khean Jin Goh[a], Suhailah Abdullah[a], Rishikesan Kuppusamy[a], Nobuhiro Yuki[b]

doi:10.1093/brain/awq260 Brain 2010: 133; 2897–2908 | 2897

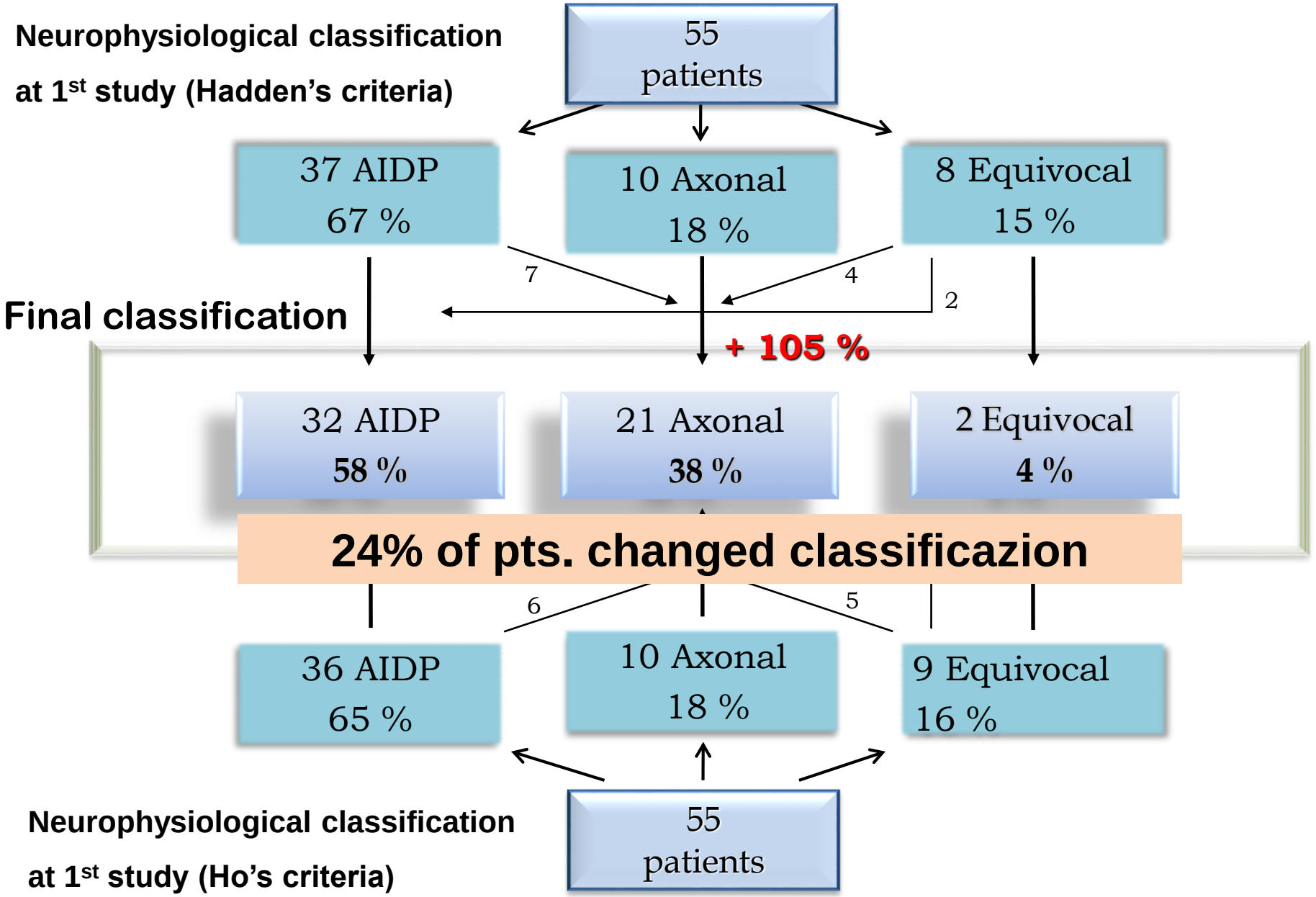
BRAIN
A JOURNAL OF NEUROLOGY

Conduction block in acute motor axonal neuropathy

Norito Kokubun,[1] Momoka Nishibayashi,[1] Antonino Uncini,[2,3] Masaaki Odaka,[1] Koichi Hirata[1] and Nobuhiro Yuki[4,5]

- 3 studies, 120 pts
- 24-38% pts changed classification after serial studies
- No patients changed from AMAN to AIDP
- 2 NCS performed within the first 2 weeks and between 3–8 weeks of disease onset suffice in elucidating the true electrodiagnosis of GBS subtypes

Confronto 1° studio ENG (media 9 gg) e 2° studio ENG (media 28 gg)



Uncini A. *JNNP* 2010

Axonal Guillain-Barrè syndrome is understimated in Europe?

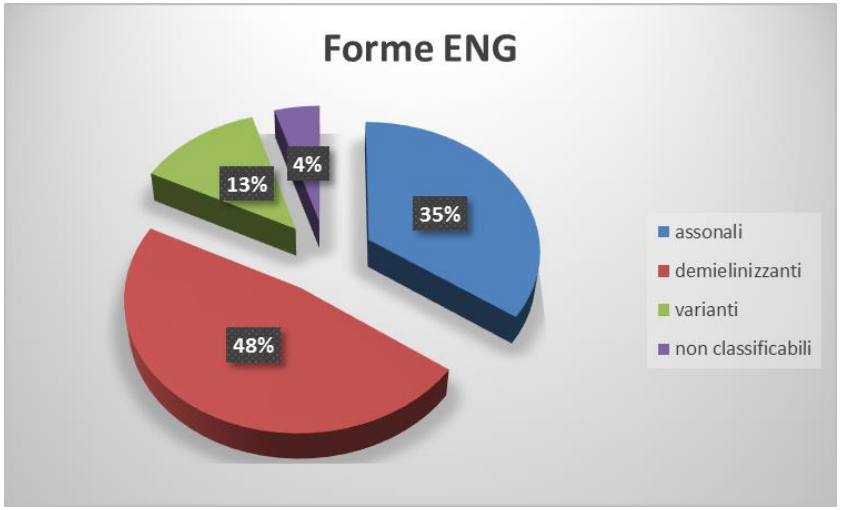
Kuwabara et al. JNNP 2010

GUILLAIN-BARRE' SYNDROME:
A THIRTEEN-YEARS RETROSPECTIVE STUDY IN PROVINCE OF LA SPEZIA

Benedetti L, Beronio A, Giorli E, Parodi S, Sani C, Derchi A, Amodeo C, Rollandi F, Delia P, Massa F, Mannironi A

ASNP. Mondello 2016

Classificazione basata
su studi ripetuti nel 88% pz



Elettrofisiologia nelle varianti

- MFS
- Faringo-cervico-brachiale
- GBS paraparetica

} ASSONALI

REVERSIBLE CONDUCTION FAILURE IN PHARYNGEAL-CERVICAL-BRACHIAL VARIANT OF GUILLAIN-BARRÉ SYNDROME

MARGHERITA CAPASSO, MD, PhD,[1] FRANCESCA NOTTURNO, MD,[1,2] CLAUDIA MANZOLI, MD,[1] NOBUHIRO YUKI, MD, PhD,[3] and ANTONINO UNCINI, MD[1]

Muscle Nerve 42: 608–612, 2010

Muscle Nerve 37: 265–268, 2008

ACUTE SENSORY ATAXIC NEUROPATHY WITH ANTIBODIES TO GD1b AND GQ1b GANGLIOSIDES AND PROMPT RECOVERY

FRANCESCA NOTTURNO, MD, CHRISTINA M. CAPORALE, MD, and ANTONINO UNCINI, MD

RCF restricted to sensory fibres

Journal of the Neurological Sciences 303 (2011) 35–38

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Motor and sensory conduction failure in overlap of Guillain–Barré and Miller Fisher syndrome: Two simultaneous cases

Yusuf A. Rajabally [a,b,*], Ghaniah Hassan-Smith [a], Francesca Notturno [c,d], Penelope J. Eames [a], Thomas Hayton [a], Margherita Capasso [c], Antonino Uncini [c,d]

- Deficit bilat. VII + parestesie → DEMIELINIZZANTE

Journal of Critical Care 37 (2017) 19–23

Contents lists available at ScienceDirect

Journal of Critical Care

journal homepage: www.jccjournal.org

Guillain-Barré syndrome in patients with a recent history of Zika in Cúcuta, Colombia: A descriptive case series of 19 patients from December 2015 to March 2016

Arturo Arias, MD [a], Lilian Torres-Tobar, MSc [b], Gualberto Hernández, MD [b], Deyanira Paipilla, MD [c], Eduardo Palacios, MD [d], Yahaira Torres, MD, MSc [c], Julian Duran, MD [e], Sebastian Ugarte U, MD [f], Adriana Ardila-Sierra, MD, PhD [g], Gabriel Castellanos, MD, PhD [g,*]

- 19 pz tutte AMAN
- 15/19 insuff. Respiratoria
- 79% GBS disability scale 4-5

Diagnosi Differenziale

OPINION

Guillain–Barré and Miller Fisher syndromes—new diagnostic classification

Benjamin R. Wakerley, Antonino Uncini and Nobuhiro Yuki for the GBS Classification Group

GBS
- Acute spinal cord disease
- Carcinomatous or lymphomatous meningitis
- Myasthenia gravis
- Critical illness neuropathy
- Thiamine deficiency
- Periodic paralysis
- Corticosteroid-induced myopathy
- Toxins (such as neurotoxic shellfish poisoning)
- Acute hypophosphataemia
- Prolonged use of neuromuscular junction blocking drugs
- Tick paralysis
- West Nile poliomyelitis
- Acute intermittent porphyria
- Functional paralysis

MFS, BBE, and pharyngeal–cervical–brachial weakness
- Brainstem stroke
- Myasthenia gravis
- Wernicke encephalopathy
- Botulism
- Brainstem encephalitis
- Diphtheria
- Tick paralysis

Bifacial weakness with paraesthesias
- Lyme disease
- Sarcoidosis

Paraparetic GBS
- Lumbosacral plexopathy
- Diabetic neuropathy
- Neoplasms
- Inflammatory conditions (such as sarcoidosis)
- Infections (such as cytomegalovirus, Lyme disease)
- Lesions of the cauda equina

Debolezza muscolare acuta (neurogena e non traumatica)

- Singolo nervo
  - *Clinica o NFC*
    - Mononeuropatia
      - Vasculite
      - Diabete
      - Rallentamenti multipli
        - HNPP
- Radicolare
  - *Clinica o NFC*
    - Radicolopatia
- Plessi
  - Arto sup. e dolore
    - S. Parsonage Turner
  - Arto inf.
    - S. Garland
- Bilaterale simmetrica
  - Distribuzione, Dolore, Dist. sfinteri
    - Cauda equina
  - Confusione, Convulsioni, Dolori addominali
    - Porfiria
  - Pancitopenia, diarrea, vomito, parestes. dolor.
    - Arsenico Tallio
  - Parestesie
    - S. Guillain Barrè
      - *NFC*
        - AIDP
        - AMSAN
        - AMAN
  - Sepsi, insuff. resp.
    - Critical illness neuropathy
  - Turbe elettrolitiche
    - Ipopotassiemia Ipofosfatemia

1ª Conferenza Nazionale "Neurologia d'Urgenza"

Vita G.

L. Ruts, MD
J. Drenthen, MD
B.C. Jacobs, MD, PhD
P.A. van Doorn, MD, PhD
On behalf of the Dutch GBS Study Group

Distinguishing acute-onset CIDP from fluctuating Guillain-Barré syndrome

A prospective study

Neurology® 2010;74:1680-1686

Background: The distinction between Guillain-Barré syndrome (GBS) with fluctuations shortly after start of treatment (treatment-related fluctuations, or GBS-TRF) and chronic inflammatory demyelinating polyneuropathy with acute onset (A-CIDP) is difficult but important because prognosis and treatment strategy largely differ.