8 NeuroMeeting

Napoli 12-13 maggio2016

Presidente del Congresso: Prof. Carlo Di Iorio.
Coordinamento Scientifico: D.ssa Carla Maglione, Dr. Tullio Cafiero, Dr. Antonio Frangiosa, Dr. Federico Bilotta.

IL MONITORAGGIO NEUROLOGICO NEL PAZIENTE CON DEBOLEZZA MUSCOLARE

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Paresis acquired in the intensive care unit: a prospective multicenter study

De Jonghe B, et al.

JAMA **2002**; 288:2859-2867

- Among the 95 patients who achieved satisfactory awakening, the incidence of ICU-acquired paresis was **25.3%**.

- Duration of mechanical ventilation after day 1 was significantly longer in patients with ICUAP compared with those without (**18.2 vs 7.6 days**).

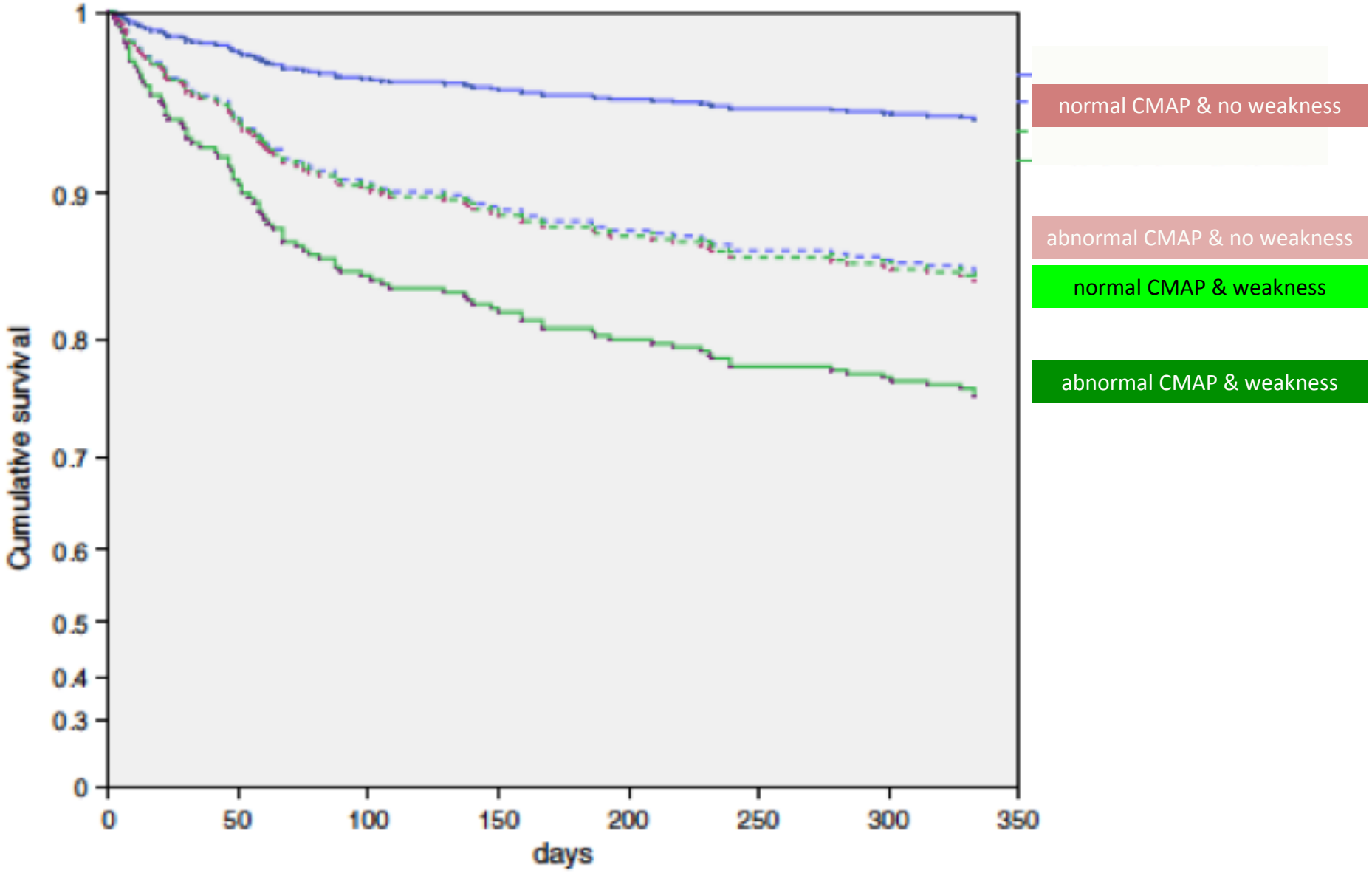
A guided approach to diagnose severe muscle weakness in the intensive care unit

Nicola Latronico and Rik Gosselink

Rev Bras Ter Intensiva **2015**; 27(3):199-201

In the ICU, severe muscle weakness is independently associated with prolonged mechanical ventilation, ICU stay, hospital stay and increased mortality.

Patients developing weakness during the ICU stay have reduced quality of life and increased mortality ***1 year*** after ICU discharge.



Hermans G, et al. Predictive value for weakness and 1-year mortality of screening electrophysiology tests in the ICU. *Intensive Care Med* **2015;** 41(12):2138-48

Intensive Care Med (2014) 40:484–495
DOI 10.1007/s00134-014-3214-y

CONFERENCE REPORTS AND EXPERT PANEL

Tarek Sharshar
Giuseppe Citerio
Peter J. D. Andrews
Arturo Chieregato
Nicola Latronico
David K. Menon
Louis Puybasset
Claudio Sandroni
Robert D. Stevens

Neurological examination of critically ill patients: a pragmatic approach. Report of an ESICM expert panel

ICUAW

- The term designates ***clinically*** detected weakness in critically ill patients in whom there is no plausible etiology other than critical illness.

Stevens R, et al. A framework for diagnosing and classifying intensive care unit-acquired weakness. *Crit Care Med* **2009**; 37[Suppl.]:S299 –S308

ICUAW

- The weakness must follow the onset of the critical illness.

Guillain-Barré Syndrome (GBS)

- GBS is often considered in the differential diagnosis of CIP, but distinction is usually obvious.
- GBS is a (rare) cause of ICU admission, whereas CIP arises as a complication of critical illness after ICU admission.
- However, the differential diagnosis can be difficult in cases of rapid progression of respiratory failure in previously undiagnosed GBS

ACUTE NEUROMUSCULAR RESPIRATORY FAILURE

Final Diagnosis	Patients, No. (%)
Myasthenia	27 (32)
GBS	12 (14)
Myopathies	12 (14)
Dermatomyositis	2
α-sarcoglycanopathy	1
Toxic necrotizing myopathy	1
Hypernatremic myopathy	1
Myotonic dystrophy	1
Myopathy with anti-SRP antibodies	1
Undetermined	5
ALS	12 (14)
Postpolio syndrome	3 (4)
CIDP	2 (2)
West Nile infection polyradiculoneuropathy	2 (2)
Amyloid polyradiculoneuropathy	1 (1)
Kennedy syndrome	1 (1)
Congenital myasthenic syndrome	1 (1)
Pseudocholinesterase deficiency	1 (1)
Myelopathy	1 (1)
Unknown	10 (12)

Cabrera Serrano M, Rabinstein AA. ***Causes and Outcomes of Acute Neuromuscular Respiratory Failure***. *Arch Neurol* **2010**;67(9):1089-1094

ICUAW

- *The weakness must follow the onset of the critical illness.*
- Physical examination shows diffuse, symmetric weakness involving all extremities and respiratory muscles.

ICUAW is usually excluded if:

1. clinical signs suggest a CNS disease (i.e. Babinski signs, increased deep tendon reflexes, spasticity, widespread muscle fasciculation, and focal neurological signs);
2. facial muscles are involved (i.e. drooping of the eyelids, weakness of extraocular muscles with diplopia, facial nerve palsy with altered patient's expression, and difficulty in speech, chewing or swallowing);
3. distribution of muscle weakness is asymmetrical (i.e. monoparesis or hemiparesis);
4. progression of muscle weakness suggests a specific diagnosis, for example, the pattern is ascending (Guillain–Barre´ syndrome) or descending (botulin intoxication);
5. muscle weakness is fluctuating and worsens after brief exercise indicating muscle fatigability and neuromuscular transmission defect (myasthenia gravis) or improves after exercise indicating pre-synaptic neuromuscular defect (Lambert–Eaton syndrome);
6. there are associated abnormalities such as skin rash or abdominal pain pointing to dermatomyositis, vasculitis, porphyria, or diabetes;
7. there are dysautonomic signs (i.e. dilated pupils poorly reactive to light suggesting botulin intoxication, and cardiac arrhythmias or fluctuations in blood pressure as seen in GBS);
8. pharmacological side effects are suspected (i.e. after prolonged administration of neuromuscular blocking agents, steroids, or cancer chemotherapy).

Sharshar T, et al. Neurological examination of critically ill patients: a pragmatic approach. Report of an ESICM expert panel. *Intensive Care Med* **2014**;40:484-495

ICUAW

- *The weakness must follow the onset of the critical illness.*
- *Physical examination shows diffuse, symmetric weakness involving all extremities and respiratory muscles.*
- MRC sumscore <48/60, or mean MRC score 4 in all testable muscle groups noted on 2 occasions separated by 24 hrs.

Medical Research Council - MRC

0 complete paralysis
1. minimal contraction
2. active movement with gravity eliminated
3. weak contraction against gravity
4. active movement against gravity and resistance
5. normal strength

MRC-sumscore (0-60)

Abduction of the arm (0-5)
Flexion of the forearm (0-5)
Extension of the wrist (0-5)

Flexion of the leg or hip flexion (0-5)
Extension of the knee (0-5)
Dorsal flexion of the foot (0-5)

Muscle groups (right and left) assessed in the measurement of the MRC-sumscore

Significant weakness
MRC <48

Severe weakness
MRC <36

Kleyweg RP, et al. *Muscle Nerve* **1991**; 14: 1103–09.

De Jonghe B, et al. *JAMA* **2002**; 288: 2859–67.

Hermans G, et al. *Muscle Nerve* **2012**; 45: 18–25

MRC SUM SCORE

Abduction of the arm (0-5) 

Flexion of the forearm (0-5) 

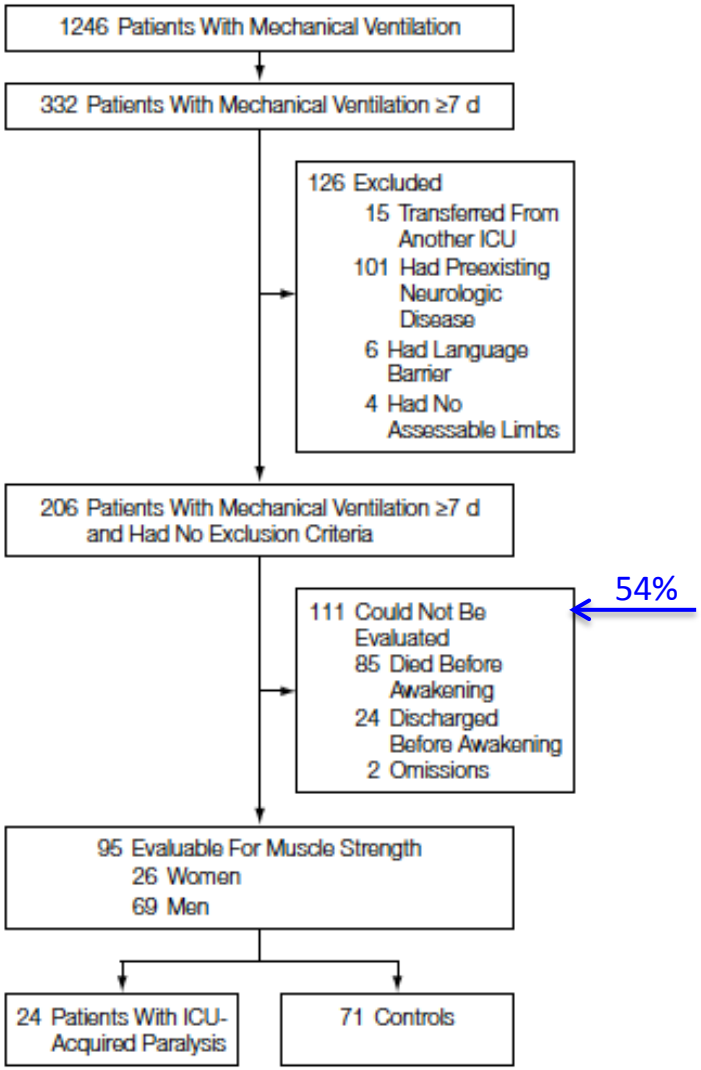
Extension of the wrist (0-5) 

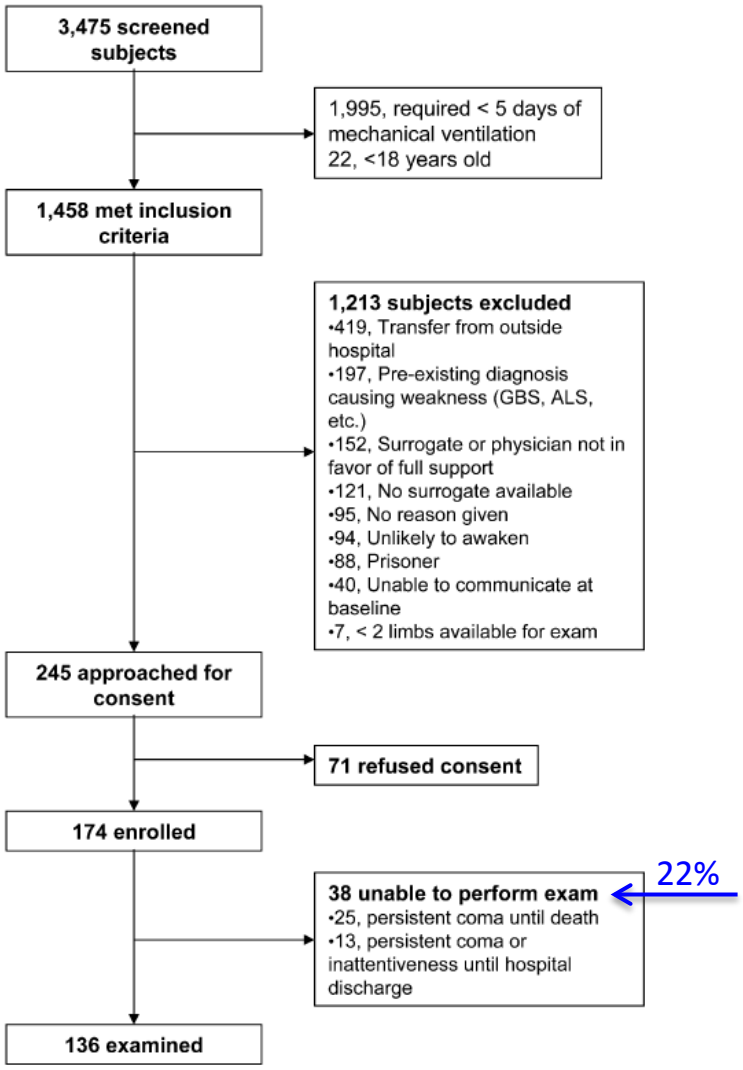
Flexion of the leg (0-5)  or hip flexion 

Extension of the knee (0-5) 

Dorsal flexion of the foot (0-5) 

Muscle groups (right and left) assessed in the measurement of the MRC-sumscore





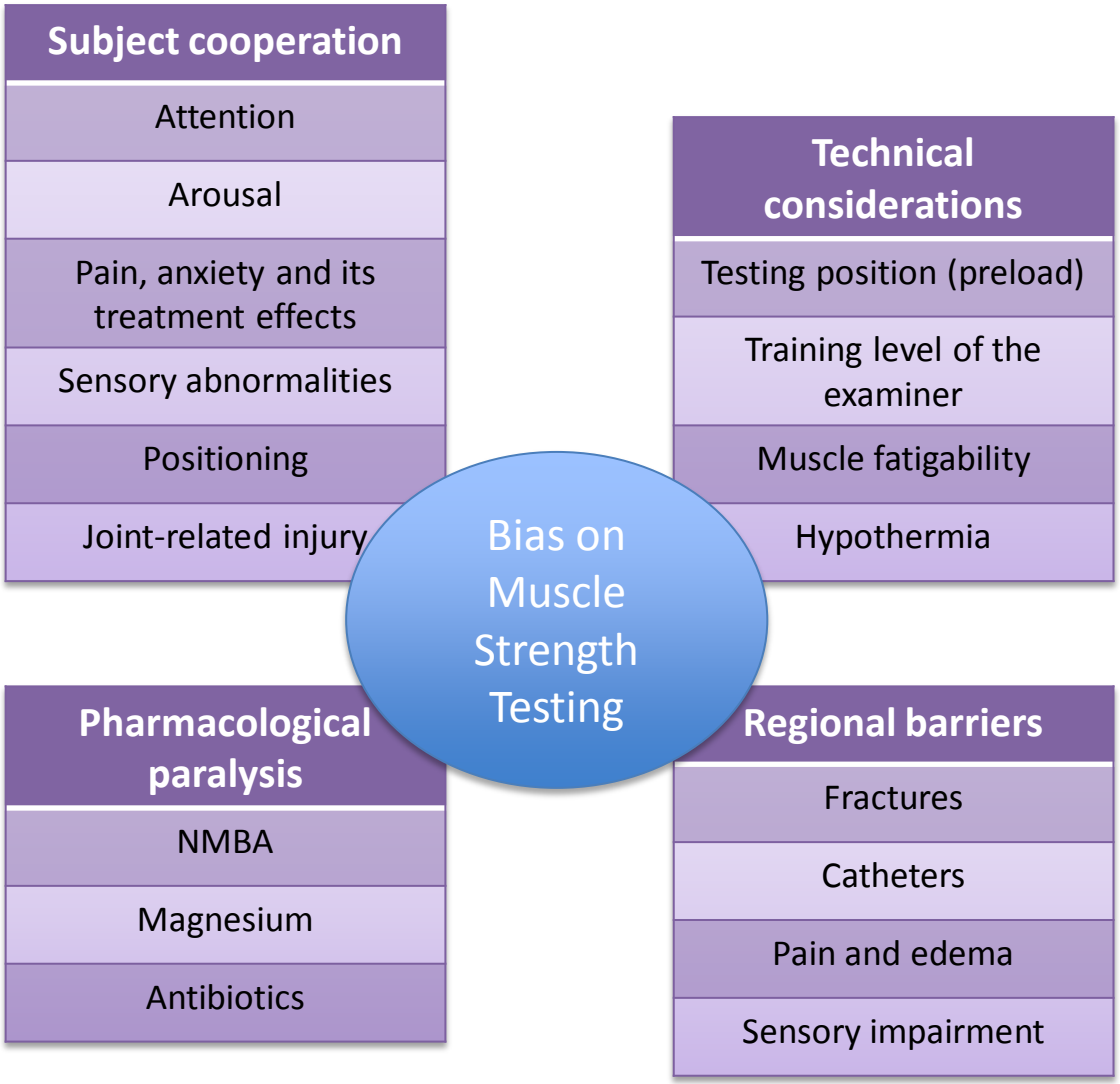
De Jonghe B , *et al*. *JAMA* **2002**; 288:2859-67

Ali NA , *et al*. *Am J Respir Crit Care Med* **2008**; 178:261-268

Acquired Muscle Weakness in the Surgical Intensive Care Unit. Nosology, Epidemiology, Diagnosis, and Prevention

Farhan H, et al.

Anesthesiology **2016**; 124:207-34



Acquired Weakness, Handgrip Strength, and Mortality in Critically Ill Patients

Ali NA, et al.

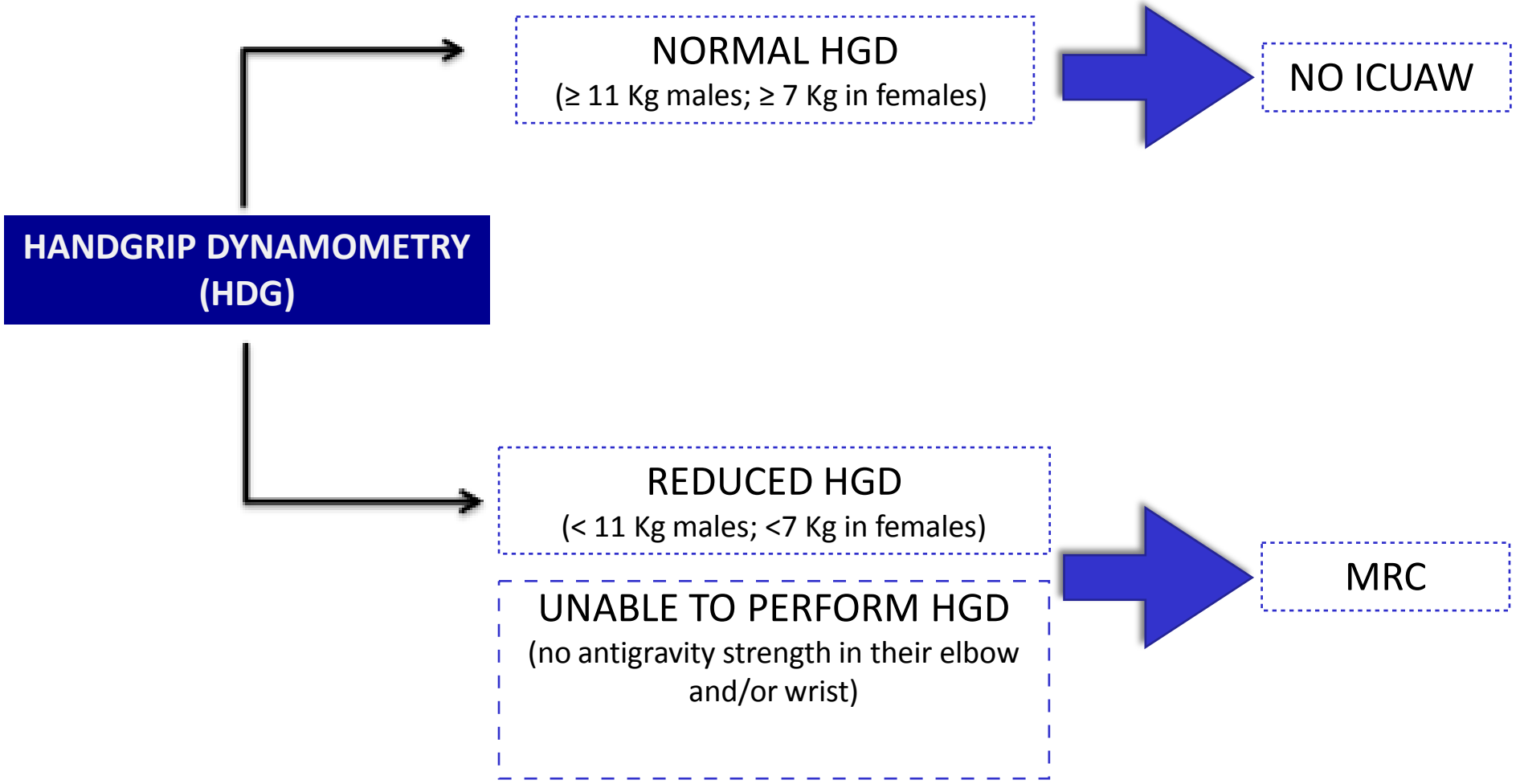
Am J Respir Crit Care Med **2008**; 178:261-268

- MRC & handgrip dynamometry.
- **25.7%** had ICUAP (a force value of <11 kg-force for males and <7 kg-force for females).
- ICUAP was independently associated with **hospital mortality** (OR 7.8; 95% CI 2.4 - 25.3).
- **ICU- and hospital-free days** were also significantly reduced in ICUAP subjects.

		Grip strength normative values at age shown (kg)						
Age (years)	Observations *	Centiles					Mean (SD)	
		10th	25th	50th	75th	90th		
Males								
5	730	6	7	8	9	10	7.7	(2.9)
10	3222	12	15	17	20	22	17.2	(4.1)
15	288	21	25	29	33	38	29.6	(5.6)
20	354	30	35	40	46	52	41.5	(7.3)
25	574	36	41	48	55	61	48.8	(8.7)
30	984	38	44	51	58	64	51.6	(9.6)
35	1380	39	45	51	58	64	51.6	(10.1)
40	880	38	44	50	57	63	50.3	(10.3)
45	798	36	42	49	56	61	48.8	(10.3)
50	820	35	41	48	54	60	47.6	(10.1)
55	3743	34	40	47	53	59	46.2	(9.8)
60	2683	33	39	45	51	56	44.6	(9.2)
65	3947	31	37	43	48	53	42.3	(8.6)
70	3286	29	34	39	44	49	39.1	(8.1)
75	1883	26	31	35	41	45	35.6	(7.6)
80	1115	23	27	32	37	42	32.2	(7.3)
85	1134	19	24	29	33	38	28.5	(7.0)
90	431	16	20	25	29	33	24.7	(6.8)
95+	5 †							
(Total)	(28,257)							
Females								
5	700	6	7	8	9	10	8.0	(3.1)
10	3339	12	14	16	19	21	16.7	(3.8)
15	345	17	20	24	27	30	23.9	(4.5)
20	463	21	24	28	32	36	28.4	(5.1)
25	870	23	26	30	35	38	30.6	(5.6)
30	1423	24	27	31	35	39	31.4	(6.0)
35	1785	23	27	31	35	39	31.3	(6.2)
40	968	23	27	31	35	39	30.7	(6.3)
45	952	22	26	30	34	38	29.9	(6.4)
50	1019	21	25	29	33	37	28.7	(6.4)
55	4250	19	23	28	32	35	27.5	(6.4)
60	2943	18	22	27	31	34	26.5	(6.2)
65	4171	17	21	25	29	33	25.3	(6.0)
70	3473	16	20	24	27	31	23.5	(5.7)
75	2135	14	18	21	25	28	21.4	(5.4)
80	1361	13	16	19	23	26	19.1	(5.1)

Dodds RM, et al. Grip Strength across the Life Course: Normative Data from Twelve British Studies. *PLoS ONE* **2014**; Dec 4;9(12):e113637

TWO-TIER STRENGTH ASSESSMENT APPROACH



Parry SM, et al. A new two-tier strength assessment approach to the diagnosis of weakness in intensive care: an observational study. *Crit Care* **2015**; 19:52

AWAKENING TIME

Days 9 [IQR 5-12]

Parry SM, et al. *Crit Care* **2015**; 19:52

ICUAW

- *The weakness must follow the onset of the critical illness.*
- *Physical examination shows diffuse, symmetric weakness involving all extremities and respiratory muscles.*
- *MRC sumscore <48/60, or mean MRC score 4 in all testable muscle groups noted on 2 occasions separated by 24 hrs.*
- Dependence on mechanical ventilation.

Journal of Neurology, Neurosurgery, and Psychiatry 1984;**47**:1223–1231

Polyneuropathy in critically ill patients

CHARLES F BOLTON, JOSEPH J GILBERT, ANGELIKA F HAHN, WILLIAM J SIBBALD

From Departments of Clinical Neurological Sciences, Pathology and Medicine, and The Critical Care/Trauma Unit, Victoria Hospital, University of Western Ontario, London, Ontario, Canada

SUMMARY Five patients developed a severe motor and sensory polyneuropathy at the peak of critical illness (sepsis and multiorgan dysfunction complicating a variety of primary illnesses). Difficulties in weaning from the ventilator as the critical illness subsided and the development of flaccid and areflexic limbs were early clinical signs. However, electrophysiological studies, especially needle electrode examination of skeletal muscle, provided the definite evidence of polyneuropathy. The cause is uncertain, but the electrophysiological and morphological features indicate a primary axonal polyneuropathy with sparing of the central nervous system. Nutritional factors may have played a role, since the polyneuropathy improved in all five patients after total parenteral nutrition had been started, including the three patients who later died of unrelated causes. The features allow diagnosis during life, and encourage continued intensive management since recovery from the polyneuropathy may occur.

Brain (1987), **110**, 819–842

CRITICAL ILLNESS POLYNEUROPATHY

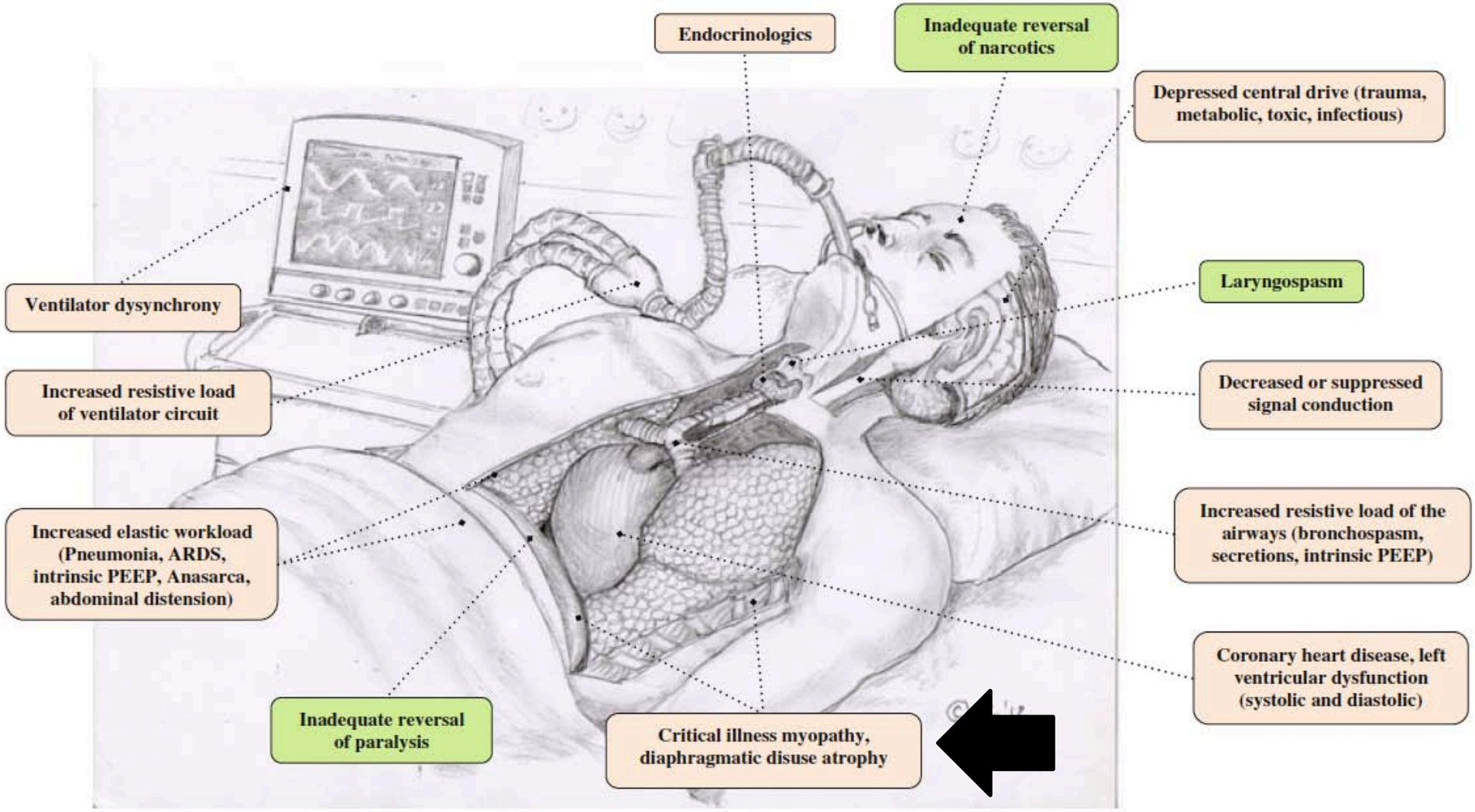
A COMPLICATION OF SEPSIS AND MULTIPLE ORGAN FAILURE

by DOUGLAS W. ZOCHODNE,[1] CHARLES F. BOLTON,[1] GEORGE A. WELLS,[4] JOSEPH J. GILBERT,[1,2] ANGELIKA F. HAHN,[1] JOHN D. BROWN[1] *and* WILLIAM A. SIBBALD[3]

SUMMARY

Nineteen patients developed polyneuropathy complicating critical illness. They had been admitted to a critical care unit following intubation for cardiac or pulmonary disease and had developed sepsis and multiple organ failure. Approximately one month following intubation, failure to wean from the ventilator and limb weakness prompted neurological referral. Examination disclosed weakness and wasting of muscles and impaired tendon reflexes in most, but not all, patients. Electrophysiological studies in 17 patients revealed attenuated compound muscle and sensory nerve action potential amplitudes and widespread denervation on needle electromyography. Autopsy in 9 patients who died of their critical illness revealed widespread primary axonal degeneration of motor and sensory fibres, with extensive denervation atrophy of limb and respiratory muscles. Survivors recovered from the polyneuropathy three to six months following discharge.

Seventeen of the patients were segregated by electrophysiological criteria into mild (8) and severe (9) polyneuropathy categories. An analysis of these two groups failed to reveal putative metabolic, drug, nutritional or toxic factors that might be responsible for the polyneuropathy. Our studies suggest that the mechanism may be a fundamental defect, still unknown, which causes dysfunction of all organ systems in this syndrome.



Perren A, Brochard L. *Intensive Care Med* **2013**;39(11):1885-95

WEANING & MV

- CIP is an independent risk factor for failed weaning from the ventilator and prolonged mechanical ventilation

Latronico N, Bolton CF. *Lancet Neurol* **2011**; 10: 931-41

ICUAW

- *The weakness must follow the onset of the critical illness.*
- *Physical examination shows diffuse, symmetric weakness involving all extremities and respiratory muscles.*
- *MRC sumscore <48/60, or mean MRC score 4 in all testable muscle groups noted on 2 occasions separated by 24 hrs.*
- *Dependence on mechanical ventilation.*
- Causes of weakness not related to the underlying critical illness have been excluded.

Question 7: how should patients be evaluated for ICU-acquired muscle weakness?

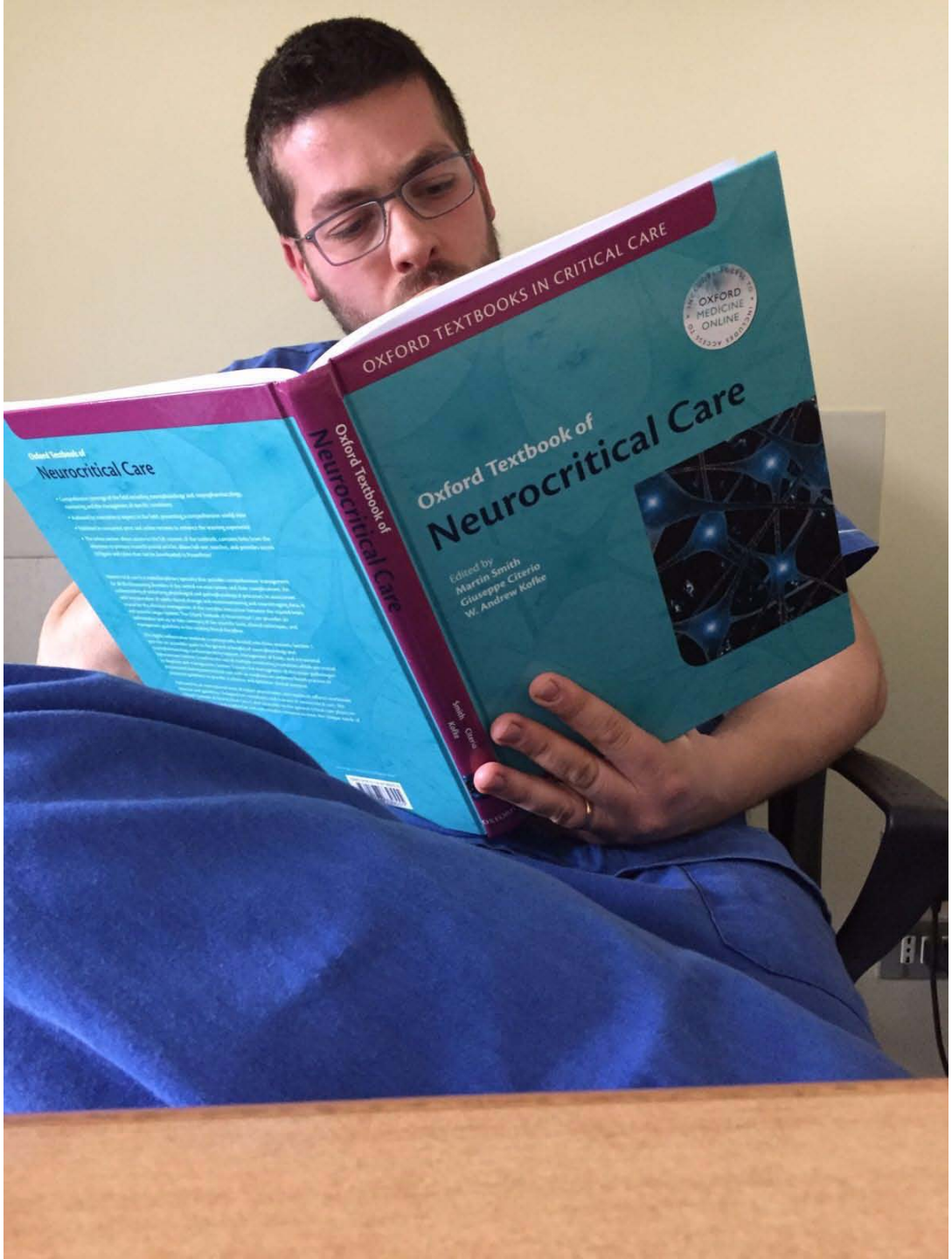
Differential diagnosis includes concurrent complications:

- electrolyte abnormalities
- rhabdomyolysis
- nerve compression or entrapment
- status epilepticus
- surgery
- use of drugs

Sharshar T, et al. Neurological examination of critically ill patients: a pragmatic approach. Report of an ESICM expert panel. *Intensive Care Med* **2014**;40:484-495

Localisation	Pre-existing	Previously Undiagnosed/New Onset	ICU Complication
Brain cortex and brainstem	Encephalitis Epilepsy Multiple sclerosis Vascular causes (brainstem infarction or haemorrhage; cerebral haemorrhage; ischaemic stroke)	Acute disseminated encephalomyelitis Encephalitis (including paralytic form of rabies) Multiple sclerosis Post-cardiac arrest encephalopathy Status epilepticus Tetanus Vascular causes	Post-cardiac arrest encephalopathy Status epilepticus (including nonepileptic) Vascular causes
Spinal cord (including anterior horn cells)	Amyotrophic lateral sclerosis Ischaemia Malformations (Arnold-Chiari) Poliomyelitis Post-polio syndrome Spinal muscular atrophy Trauma	Compression (tumour, infection, haematoma) Herpes zoster Ischaemia Transverse myelitis Surgery Tetanus Trauma West Nile virus poliomyelitis	Hopkins syndrome
Peripheral nerve	Alcohol abuse Chronic inflammatory demyelinating polyneuropathy Drugs* (bortezomib, cisplatin, dichloroacetate, epothilone, isoniazid, ixabepilone, leflunomide, linezolid, nitrofurantoin, oxaliplatin, pyridoxine, reverse transcriptase inhibitors, statins, taxanes, thalidomide, tumour necrosis factor-alpha blockers, vincristine) Guillain-Barré syndrome Hormonal disorders (acromegaly, hypothyroidism) Infections (diphtheria, HIV, Lyme disease) Tumours (carcinoma, lymphoma, multiple myeloma) Metabolic (diabetes, porphyria, tyrosinaemia, uraemia) Nutritional (thiamine deficiency) Sarcoidosis Toxic (acrylamide; heavy metals: arsenic, thallium, lead, gold; organophosphates; hexacarbons) Vasculitis (polyarteritis nodosa, lupus erythematous, rheumatoid arthritis, Churg-Strauss)	Acute intermittent porphyria Entrapment neuropathy Guillain-Barré syndrome HIV Tetanus Tick paralysis Toxic Vasculitis	Entrapment neuropathy Critical illness polyneuropathy
Neuromuscular junction	Botulism. Lambert-Eaton syndrome. Myasthenia gravis Drugs*. ANESTHETIC AGENTS (desflurane, enflurane, halothane, isoflurane, nitrous oxide, opioids, propofol, sevoflurane). ANTIBIOTICS: *amynoglycosides*+ (amikacin, clindamycin, gentamycin, kanamycin, lincomycin, neomycin, streptomycin, tobramycin); *fluoroquinolones* (ciprofloxacin, gemifloxacin, levofloxacin, lomefloxacin, moxifloxacin, norfloxacin, ofloxacin, and trovafloxacin); *macrolides* (azithromycin, erythromycin, telithromycin), *other antibiotics* (ampicillin, bacitarcin, polymyxins, tetracyclin, imipenem/cilastatin, penicillin, vancomycin). ANTI-ARRHYTHMIC AGENTS (etafenone, peruvoside, procainamide, propafenone). ANTIEPILEPTICS (carbamazepine, gabapentin, phenytoin, trimethadione). BETA-BLOCKERS** (atenolol, nadolol, oxprenolol, practolol, propranolol, sotalol, ophthalmic timolol). CALCIUM-CHANNEL BLOCKERS** (amlodipine, felodipine, nifedipine, verapamil). CORTICOSTEROIDS***. CHEMOTHERAPICS (doxorubicin, etoposide, cisplatin). H-2 RECEPTOR ANTAGONISTS (cimetidine, ranitidine, roxatidine). QUINOLONE DERIVATIVES (chloroquine, quinidine, quinine). NONCOMPETITIVE NEUROMUSCULAR BLOCKING AGENTS+. Psychotropic medications (amitriptyline, chlorpromazine, haloperidol, imipramine, lithium). OTHER DRUGS (interferon, penicillamine).	Hypermagnesaemia Myasthenia gravis Snake, scorpion and spider bites fish, shellfish, jellyfish and crab toxins Tetanus	Hypermagnesaemia Prolonged neuromuscular blockade
Muscle	Metabolic/congenital Mitochondrial myopathies Muscular dystrophies Periodic paralyses (muscle channelopathiyes) Polymyositis	Adult-onset acid maltase deficiency Hypo- and hyperkalaemia Hypophosphataemia Muscular dystrophies Polymyositis Pyomyositis Rhabdomyolysis Tetanus Toxic myopathies	Corticosteroid myopathy Critical illness myopathy Hypo- and hyperkalaemia Hypophosphataemia Propofol infusion syndrome Disuse atrophy Rhabdomyolysis

Latronico N, Fagoni N. Neuromuscular Disorders & Acquired Neuromuscular Weakness. In: Oxford Textbook of Neurocritical Care. Smith M, CITERIO G, Kofke A W (Eds). Oxford University Press, 2016


OXFORD TEXTBOOKS IN CRITICAL CARE
OXFORD MEDICINE ONLINE
Oxford Textbook of
Neurocritical Care
Edited by
Martin Smith
Giuseppe Citerio
W. Andrew Kofke
Oxford Textbook of
Neurocritical Care
Smith
Citerio
Kofke

ICU-ACQUIRED WEAKNESS (ICUAW)

Disuse atrophy
(with muscle deconditioning)

Critical Illness Polyneuropathy

Critical Illness Myopathy

ICU-ACQUIRED WEAKNESS (ICUAW)

Disuse atrophy
(with muscle deconditioning)

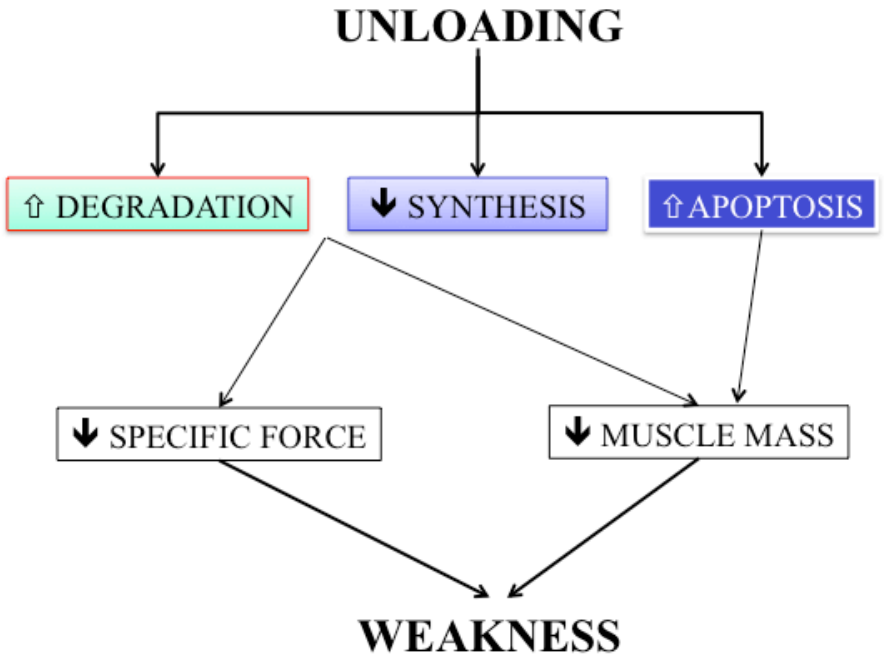
Critical Illness Polyneuropathy

Critical Illness Myopathy

DISUSE MUSCLE ATROPHY

In healthy adults, muscle strength declines by 1% per day of strict bed rest.

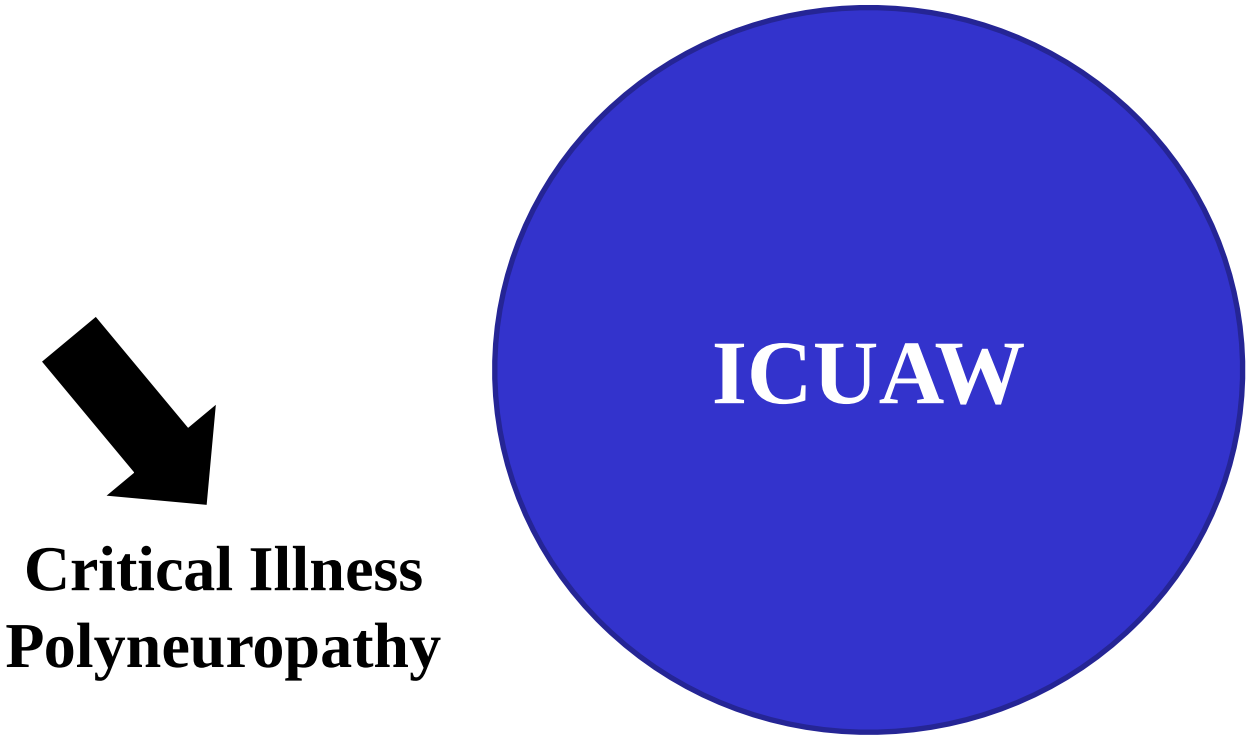
Limb immobilization by casting results in a faster decline in muscle strength — about 25% within 7 days.



Chambers MA, et al. Physical inactivity and muscle weakness in the critically ill. *Crit Care Med* **2009**; 37(10 Suppl.): S337-46

ICU-ACQUIRED WEAKNESS (ICUAW)

Disuse atrophy
(with muscle deconditioning)



Critical Illness Polyneuropathy

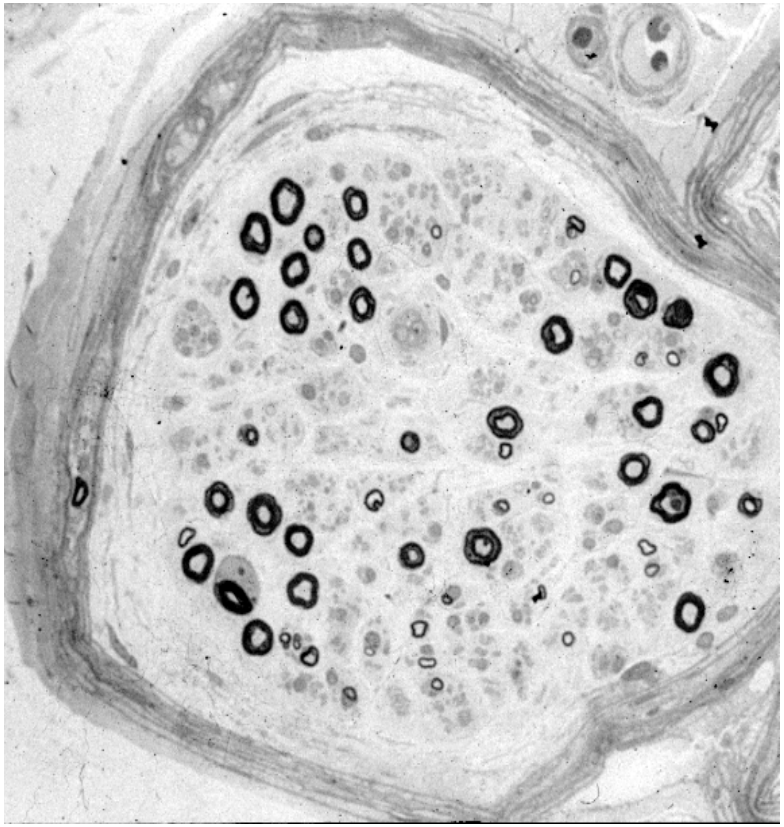
Critical Illness Myopathy

CRITICAL ILLNESS POLYNEUROPATHY

- CIP is a distal axonal sensory-motor polyneuropathy affecting limb and respiratory muscles

Latronico N, Bolton CF. *Lancet Neurol* **2011**; 10: 931-41

CRITICAL ILLNESS POLYNEUROPATHY



Latronico N, et al. *Lancet* **1996**; 347:1579-1582

ICU-ACQUIRED WEAKNESS (ICUAW)

Disuse atrophy
(with muscle deconditioning)

Critical Illness Polyneuropathy

Critical Illness Myopathy

CRITICAL ILLNESS MYOPATHY

- CIM is a primary myopathy that is not secondary to muscle denervation, with distinctive electrophysiological and morphological findings

Latronico N, Bolton CF. *Lancet Neurol* **2011**; 10: 931-41

CRITICAL ILLNESS MYOPATHY

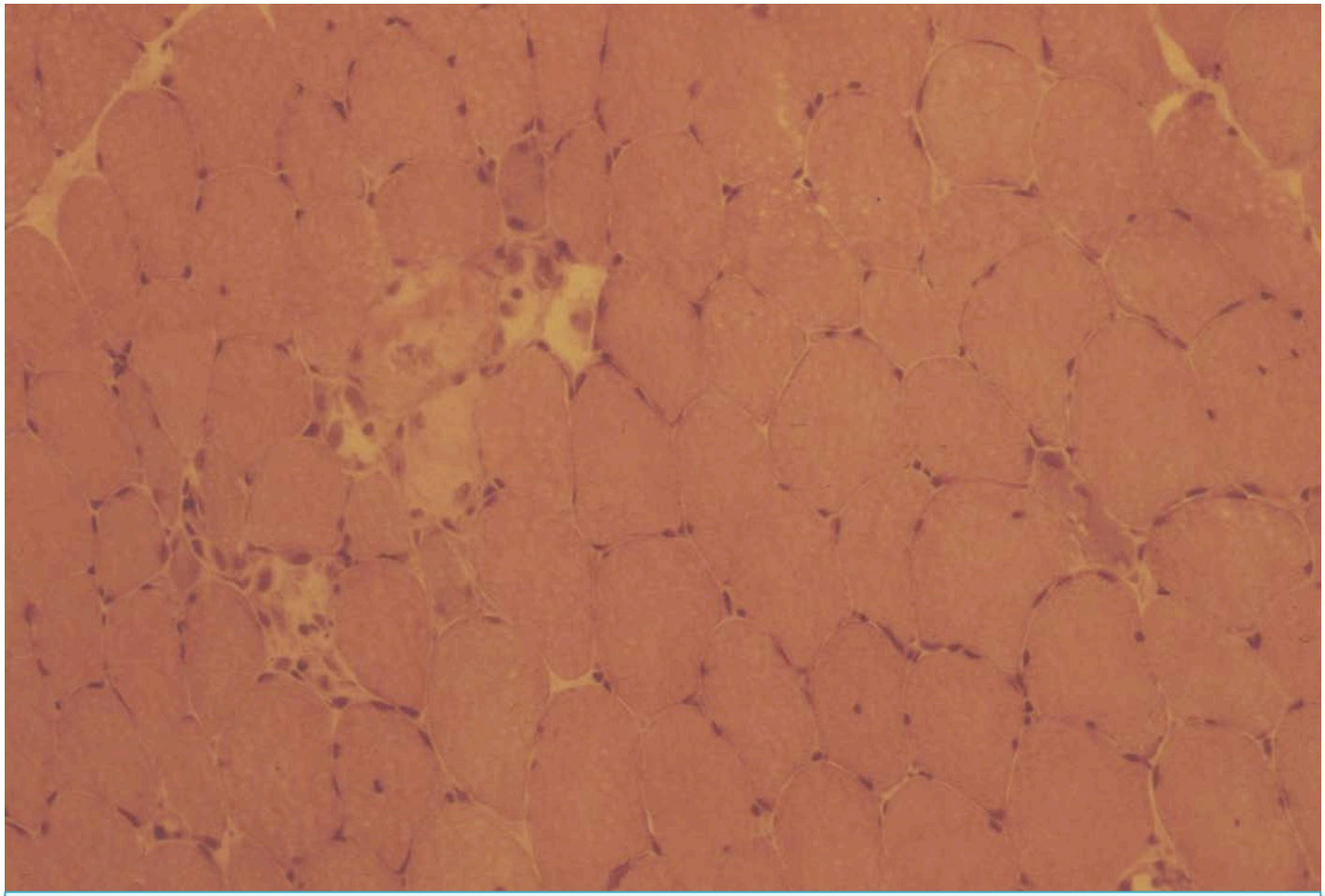
Condition	Incidence	Clinical features	Electrophysiologic findings	Serum creatine kinase	Muscle biopsy	Prognosis
Thick-filament myopathy	Common with steroids, neuromuscular blocking agents, and sepsis	Flaccid limbs; respiratory weakness	Abnormal spontaneous activity	Mildly elevated	Loss of thick (myosin) filaments	Good
Acute myopathy with scattered necrosis	Common	Flaccid limbs; respiratory weakness	Myopathy	Mildly or moderately elevated	Scattered necrosis	Variable
Acute myopathy with diffuse necrosis (necrotising myopathy of intensive care)	Rare	Flaccid weakness; myoglobinuria	Severe myopathy	Markedly elevated, myoglobinuria	Marked necrosis	Poor
Disuse (cachectic) myopathy	Common	Muscle wasting	Normal	Normal	Normal or type II fibre atrophy	Variable
RHABDOMYOLYSIS	Rare	Flaccid limbs	Near normal	Markedly elevated (myoglobinuria)	Normal or mild necrosis	Good

Latronico N, Bolton CF. *Lancet Neurol* **2011**; 10: 931-41

THICK FILAMENT MYOPATHY

Latronico N, Tomelleri G, Filosto M. *Curr Opin Rheumatol* **2012**; 24:616–622

MUSCLE FIBER NECROSIS



Latronico N, Tomelleri G, Filosto M. *Curr Opin Rheumatol* **2012**; 24:616–622

CRIMYNE

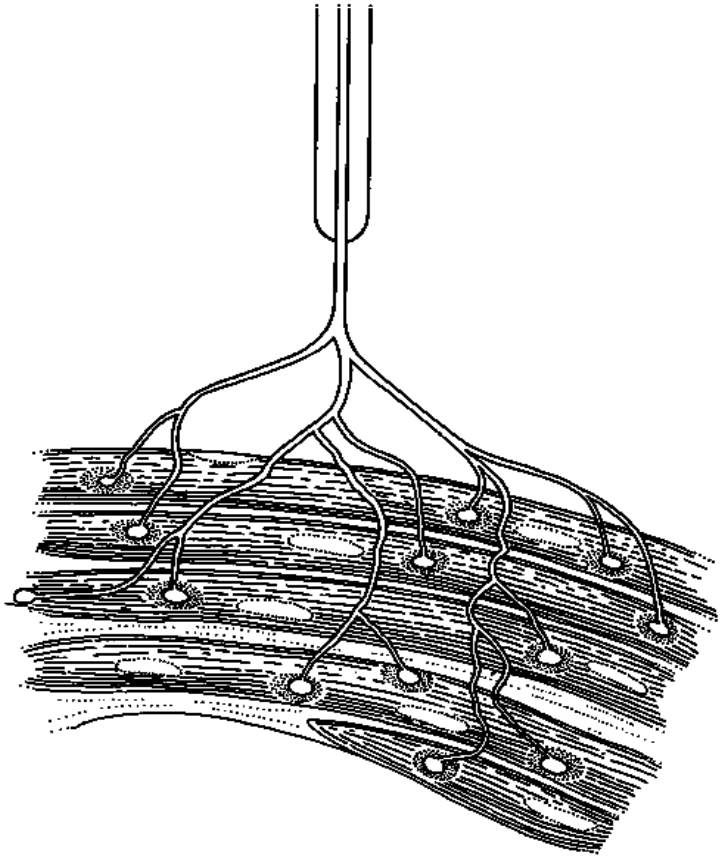
- Combined CIP and CIM could be the most common manifestation of neuromuscular weakness in the ICU.

Latronico N, Bolton CF. *Lancet Neurol* **2011**; 10: 931-41

CRIMYNE

CIP (denervation)

CIM (primary myopathy)



Latronico N, et al. *Curr Opin Crit Care* **2005**; 11:381-390

SMALL NERVE FIBER PATHOLOGY IN CRITICAL ILLNESS

- Of the 14 patients recruited, 13 (93%) had infections, sepsis or multiple organ failure.
- All had degeneration of small nerve fibers, and reduced sweat gland innervation.
- Of the 7 patients available for follow-up visit, three complained of diffuse sensory loss and burning pain, and another three showed clinical dysautonomia.

Latronico N, et al. *PLoS One* **2013** Sep 30;8(9):e75696

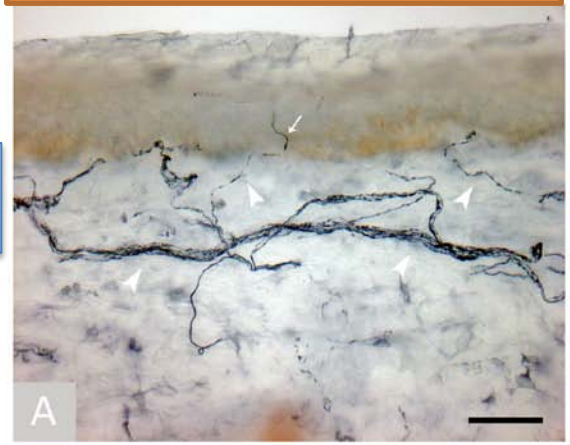
Critically ill patient

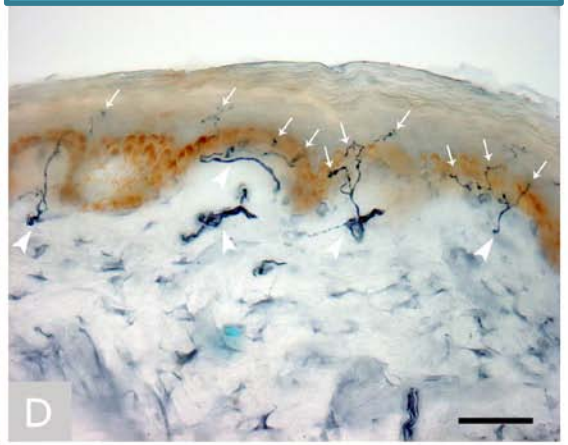
Healthy control

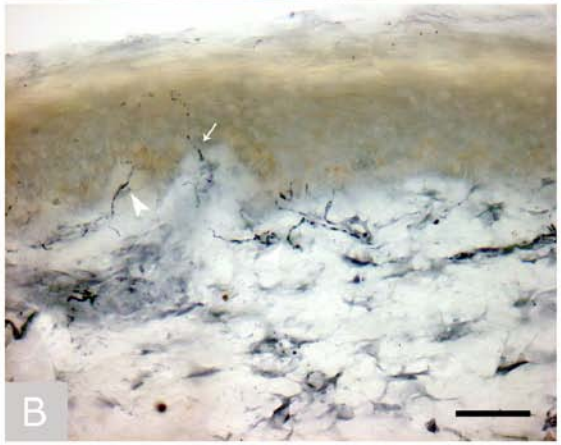
Severe depletion of intra-***epidermal*** nerve fibers

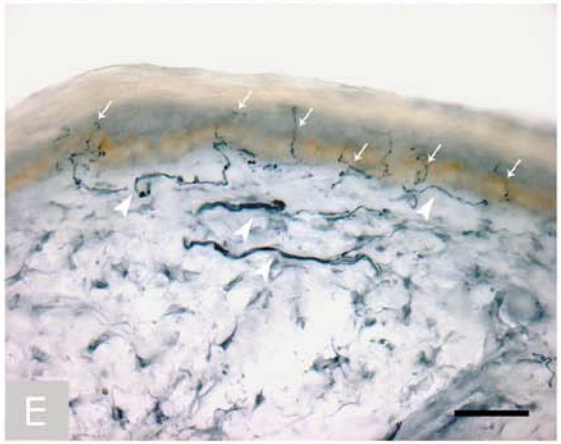
Severe reduction in the density of ***dermal*** nerve bundles

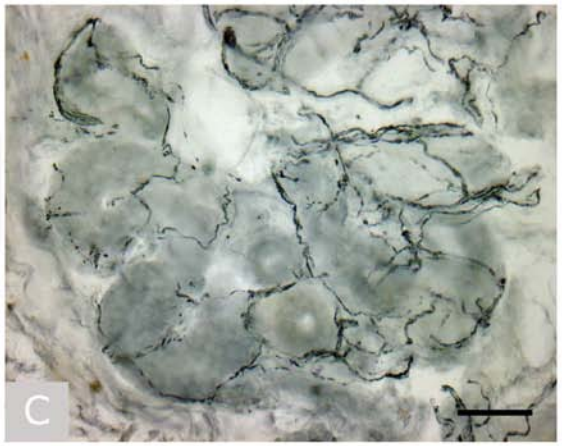
Severe reduction ***of sweat gland*** innervation

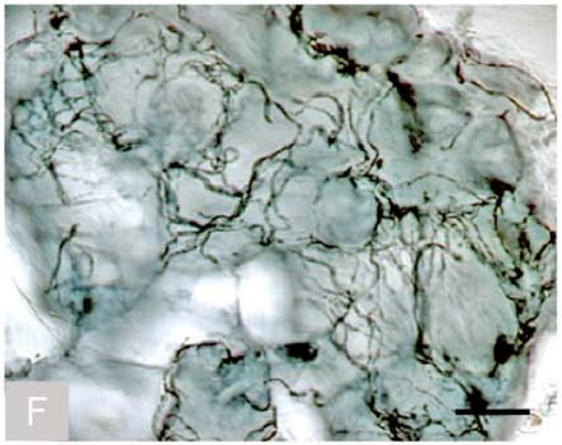




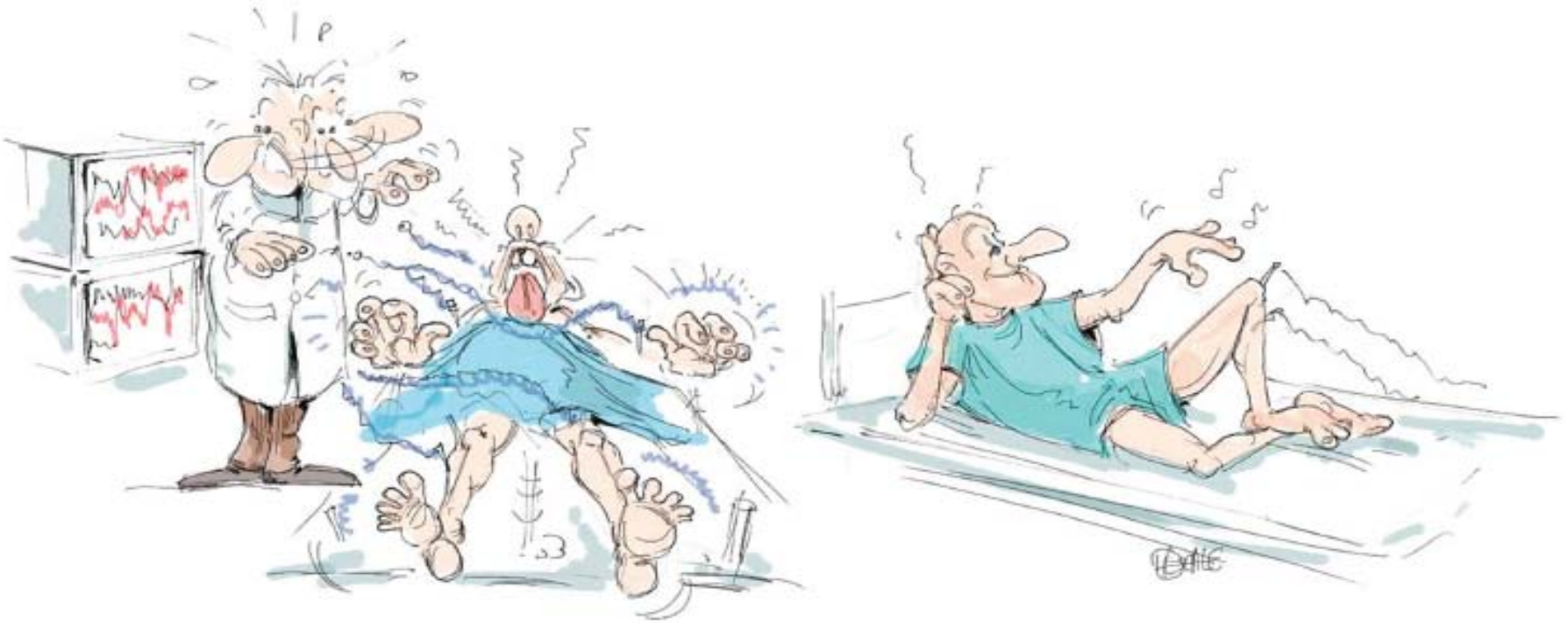








SIMPLIFIED ELECTROPHYSIOLOGICAL TESTS



Latronico N, Smith M. *Intensive Care Med* **2014**;40(5):746-8

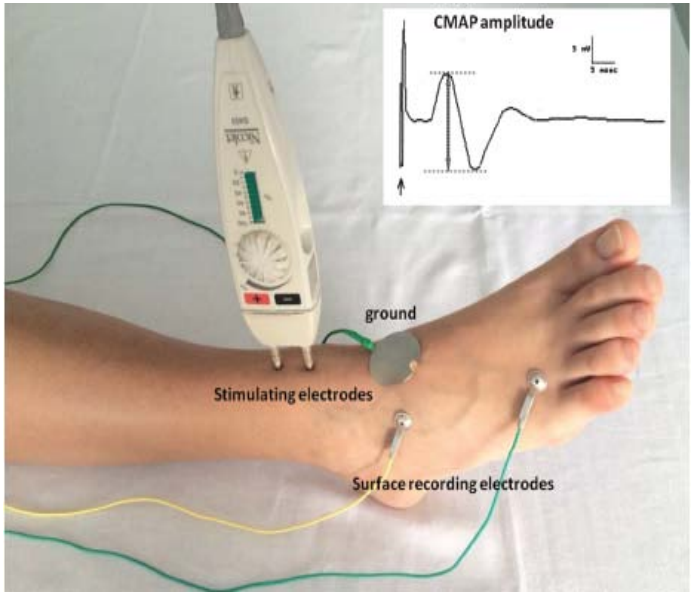
Validation of the peroneal nerve test to diagnose critical illness polyneuropathy and myopathy in the intensive care unit: the multicentre Italian CRIMYNE-2 diagnostic accuracy study.

Latronico N, Nattino G, Guarneri B, Fagoni N, Amantini A, Bertolini G, and *GiVITI* Study Investigators
F1000Research **2014;** 3:127

Objectives: accuracy of PENT compared to complete NCS-EMG.

Patients: 121 pts (neurologic 106 and non-neurologic 15, LOS ≥ 3 days in 9 Italian ICUs.

Measurements and main results: Same day, independent clinicians, blind assessment.



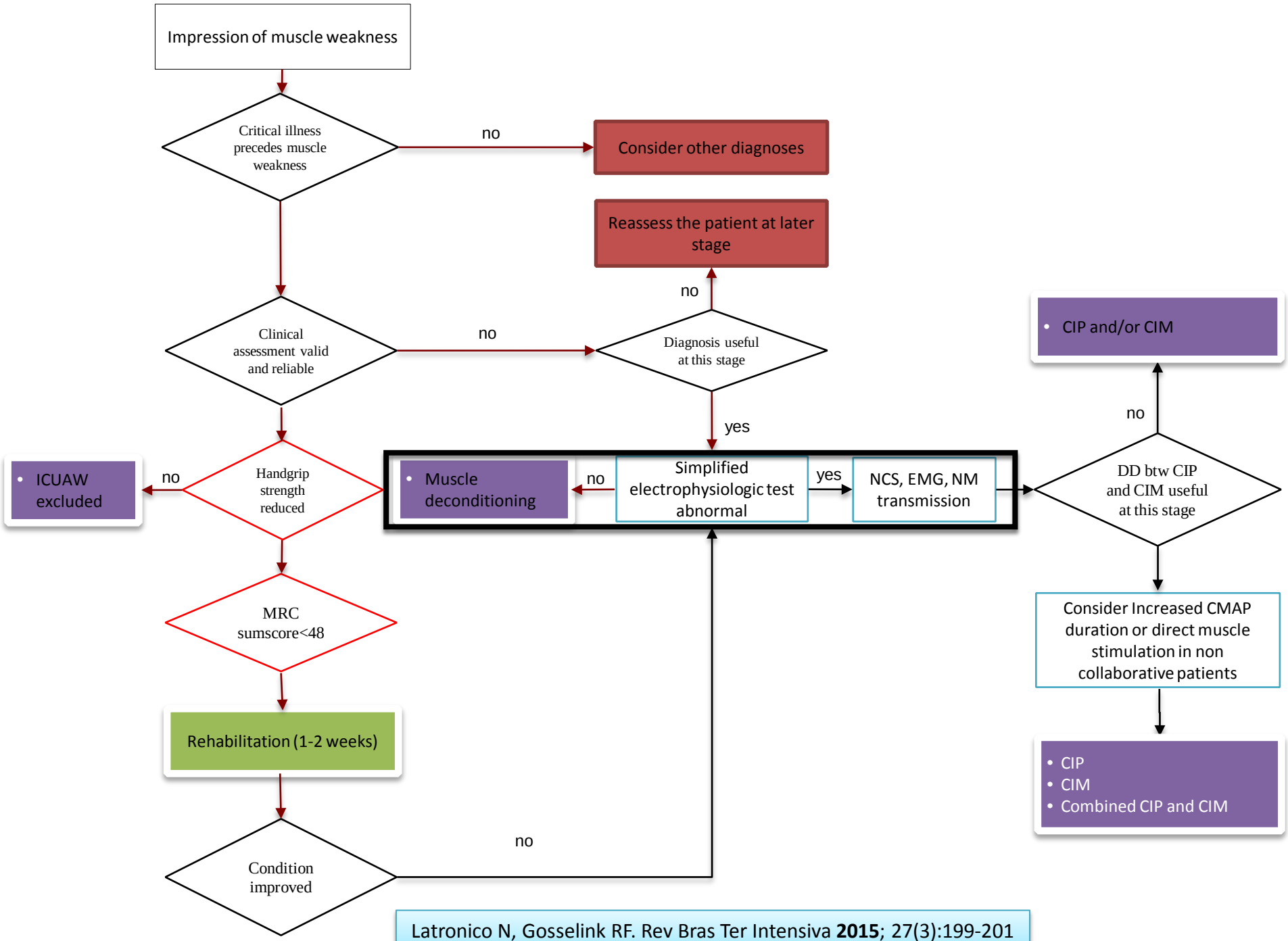
CRIMYNE-2

- Sensitivity and specificity of PENT were **100%** (95% CI 96.1-100.0) and **85.2%** (95% CI 66.3-95.8).

- The median time needed to perform PENT and the complete NCS-EMG was **10 minutes** (IQR 8.0-10.5) and 50 minutes (40-60)

SIMPLIFIED ELECTROPHYSIOLOGICAL TESTS

1. van den Berghe G, et al. Intensive insulin therapy in the critically ill patients. *N Engl J Med* **2001**; 345:1359-1367
2. Van den Berghe G, et al. Intensive insulin therapy in the medical ICU. *N Engl J Med* **2006**; 354:449-461
3. Latronico N, et al. Simplified electrophysiological evaluation of peripheral nerves in critically ill patients: the Italian multi-centre CRIMYNE study. *Crit Care* **2007**; 11(1):R11
4. Weber-Carstens S, et al. Nonexcitable muscle membrane predicts intensive care unit-acquired paresis in mechanically ventilated, sedated patients. *Crit Care Med* **2009**; 37:2632-2637
5. Moss M, et al. Screening for critical illness polyneuromyopathy with single nerve conduction studies. *Intensive Care Med* **2014**; 40(5):683-90
6. Latronico N, et al. Validation of the peroneal nerve test to diagnose critical illness polyneuropathy and myopathy in the intensive care unit: the multicentre Italian CRIMYNE-2 diagnostic accuracy study. *F1000Research* **2014**; 3:127
7. Hermans G, et al. Predictive value for weakness and 1-year mortality of screening electrophysiology tests in the ICU. *Intensive Care Med* **2015**; 41(12):2138-48



Latronico N, Gosselink RF. Rev Bras Ter Intensiva **2015**; 27(3):199-201

THE END