CRITICAL ILLNESS POLYNEUROMYOPATHY – A WELL-KNOWN BUT STILL CONTROVERSIAL ENTITY

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ABSTRACT

Two main clinical, pathological and electrophysiological types of acquired neuromuscular involvement in critically ill patients have been described in the past two decades: critical illness polyneuropathy and critical illness myopathy. However, there, still exist many controversies and unresolved questions regarding definition, terminology, diagnosis, and differentiation of what appears to be a spectrum of more or less overlapping neuromuscular disorders rather than distinct entities, and recently a new term – critical illness polyneuromyopathy (CIPM) has been coined. CIPM may present with muscle weakness and failure to wean from mechanical ventilation, but is discovered more often and earlier by electrophysiological examination. In this review, the incidence, clinical, electrophysiological and histopathological features, and risk factors of CIPM will be described. Among the most important risk factors for CIPM are sepsis or systemic inflammatory response syndrome and the severity of multi-organ failure. Acquired neuromuscular weakness in critically ill patients should be regarded as a part rather than a complication of critical illness – dysfunction or failure of a further (neuromuscular) system.

THE ICU WEAKNESS: TERMINOLOGY, DIAGNOSTIC CRITERIA AND DIFFERENTIAL DIAGNOSIS

Neuromuscular weakness is often encountered in patients in the intensive care unit (ICU). Patients can be admitted to the ICU because of increasing muscle weakness and threatening respiratory failure due to an underlying neuromuscular disorder, such as Guillain-Barré syndrome (GBS) and myasthenia gravis (MG). More frequently, a new weakness appears during the ICU stay. If weakness of central origin due to encephalopathy and rarely to myelopathy is excluded, a neuromuscular weakness could be caused by exacerbation of an underlying neuromuscular disease (such as MG, motor neuron disease or muscular dystrophy), or a weakness due to persistent neuromuscular blockade after administration.
of non-depolarising muscle blocking agent (NDMBA) must be taken into consideration. By far the most frequent cause of new acquired weakness of neuromuscular origin in ICU patients is, however, critical illness polyneuropathy (CIP), critical illness myopathy (CIM), or both [1,2]. In this review, we will focus mainly on those aspects of acquired neuromuscular weakness in critically ill patients that we addressed in our previous research: epidemiology, diagnosis, and aetiology. CIP was first systematically described in the early 1980s [3], while myopathy in critically ill patients was first reported in 1977 in association with the administration of corticosteroids [4]. The first paper focused on this problem in the Czech or Slovak literatures was published in 2000 [5], and subsequently also in paediatric patients [6]. The differentiation between CIP and CIM is based on the typical clinical, electrophysiological and histopathological signs of acute axonal sensorimotor polyneuropathy and myopathy, and on the assumption that most cases can be categorised as one type or the other [7,8]. There is, however, increasing evidence that myopathy may co-exist with neuropathy [9,10], and that the final diagnosis – CIP, CIM or both – is critically dependent on the method used [10]. Another factor complicating diagnosis is the fact that neither CIP nor CIM are homogenous entities. Although CIP is typically sensorimotor acute axonal polyneuropathy, pure motor and pure sensory forms of CIP have also been described [10–12]. CIM is an even more heterogeneous group of muscle disorders. At least four main types of CIM have been reported: myopathy with selective loss of myosin filaments (‘myosin loss myopathy’, ‘thick filament myopathy’), acute necrotising myopathy, non-necrotising ‘cachectic’ myopathy, and decreased sarcolemmic excitability [13,14]. The term ‘critical illness myopathy’ has been coined for myopathy with loss of myosin [8]; for non-necrotising ‘cachectic myopathy’ with dominant type II atrophy [15]; or as a descriptive counterpart to CIP covering all types of myopathy in critically ill patients [16]. It is not known whether all these changes share the same aetiology and represent one disease. CIP has been thought of as an easy diagnosis to make. Clinical diagnosis of CIP is based on the presence of flaccid areflexic quadriparesis with sensory involvement; this presence is, however, difficult to prove in comatose or unco-operative patients. Although some authors have reported different distributions of muscle weakness in CIP and CIM, differentiation between neuropathy and myopathy based on clinical grounds in comatose, sedated or encephalopathic critically ill patients is highly unreliable [8,17].

**ELECTROPHYSIOLOGY**

An electrophysiological pattern of acute axonal sensorimotor polyneuropathy is not difficult to discern, but differentiation between CIP and CIM is difficult or even impossible for numerous reasons not associated in other conditions [11,18]. Routine conduction studies and needle electromyography (EMG) provide only non-specific data. Abnormal spontaneous activity and decreased amplitudes of compound muscle action potentials can occur in different lesions of a motor unit, including myopathy. Diffuse tissue oedemas of the extremities of critically ill patients could lead to false-positive sensory conduction abnormalities. The assessment of recruitment and interference in the voluntary EMG pattern is obscured by severe weakness or poor voluntary effort in most patients. Some authors have reported the usefulness of sophisticated electrophysiological methods, such as single-fibre EMG [19], quantitative electromyography (QEMG), and motor unit number estimation [18,20]. CIM is a difficult diagnosis to make using electrophysiological methods [16]. If such diagnosis is based only on electrophysiological criteria, it is considerably underdiagnosed [10]. There are several reasons for this underestimation. Motor involvement in acute axonal neuropathy and myopathy shares the same pattern on conventional electroneurography (ENG) and needle EMG. In the presence of an abnormal sensory neurogram, motor abnormalities have invariably been attributed to neuropathy, irrespective of the possibility of concomitant myopathy associated with sensory or sensorimotor polyneuropathy [12]. Some additional techniques useful in the detection of myopathy, such as quantitative EMG, require patient cooperation, which is lacking in most cases. Nevertheless, some studies utilising these techniques have reported predominant myopathic involvement in critically ill patients [13,18,20]. Trjaborg et al. [18] carried out electrophysiological studies on 22 consecutive patients with critical illness-associated weakness and found electrophysiological signs of myopathy in all cases (confirmed by biopsy in nine of them), while only one patient showed electrophysiological signs of polineuropathy. Lacomis et al. [20], studying 100 patients with new weakness, reported that electrophysiological signs of acute myopathy were three times more frequent (42 %) than those of acute polyneuropathy (13 %). Rich et al. [13] reported direct muscle stimulation (DMS) findings, indicative of decreased sarcolemmic excitability, in 11 out of 14 critically ill weak patients. The newly introduced DMS method [5,13,21] enables the investigator to distinguish decreased muscle membrane excitability from other causes of muscle weakness. It has, however, some methodological pitfalls [18,21]. One of them is the use of the ratio of compound muscle action potentials (CMAPs) obtained with direct muscle stimulation (dmCMAP) and nerve stimulation (neCMAP); where the ratio ne/dmCMAP is >0.5 it is seen as a sign of decreased muscle membrane excitability [13]. This value can be found in normal muscle, and electroclinical correlation would be rendered difficult or impossible by lack of co-operation or central palsy. The absolute value of
dmCMAP amplitude is a more valuable indicator of decreased sarcolemmic excitability [18,21], but it is an extremely variable parameter, prone to false-positive findings [21,22]. Another pitfall in the interpretation of DMS signs of decreased muscle membrane excitability is that the histopathological correlates of this condition are unknown in man. Recently, decreased muscle membrane excitability caused by increased fast inactivation of sodium channels was documented in an animal model of myosin loss myopathy in steroid-denervated rats [23]. In our material [21], decreased muscle membrane excitability was associated with various histopathological myopathic changes in all seven patients with this particular DMS pattern in whom muscle biopsy was performed. One can speculate that decreased muscle membrane excitability is probably a part or a phase of a complex pathological process involving muscle fibres during critical illness that does not usually remain isolated as a cause of muscle weakness. The interpretation of isolated electrophysiological motor abnormalities has been a matter of discussion for the last 15 years. A substantial proportion of weak, critically ill patients (46% in our study sample, 30% in a group studied by Coakley et al. [11]) show a pure motor abnormality on conventional ENG/EMG examination. Coakley [11] introduced the term ‘pure motor syndrome’ for this electrophysiological pattern. There are several reports of ‘pure motor neuropathy’, mostly attributed to the administration of non-depolarising muscle-blocking agents [23–25], but the evidence of neuropathy is based on non-specific electrophysiological motor abnormalities [24], on the absence of myopathic changes [23], or on the presence of neurogenic atrophy on muscle biopsy [25]. Interpretation of histopathological signs of denervation atrophy is, however, somewhat controversial (see discussion below). Lacomis et al. [20] interpreted isolated motor abnormalities as signs of myopathy and we confirmed this assumption in our study: myopathic changes on muscle biopsy were found in all six patients with pure motor syndrome chosen for biopsy and decreased muscle excitability DMS pattern in eight out of 12 cases with pure motor syndrome in our group [21]. We can thus conclude that a motor form of critical illness polyneuropathy has not been adequately proved by pathological or electrophysiological studies. Recently, early electrophysiological detection of neuromuscular involvement in critically ill patients has been reported [26].

**BIOPSY**

Histopathological changes on muscle biopsy are used as a benchmark in the diagnosis of critical illness myopathy [4] and as a gold standard for evaluation of the validity of electrophysiological parameters [7,8,10,11,18]. Latronico et al. [10] studied 24 acutely ill neurological patients with clinical and ENG-EMG signs of CIP with muscle and nerve biopsy. They found that 23 patients (96%) had a myopathy, and that 15 of these would have been diagnosed as having only a critical illness polyneuropathy, had they not performed muscle biopsy. Similar results were obtained in a study by DeJonghe et al. [27], in which all 10 patients with an ENG-EMG diagnosis of CIP also had signs of myopathy on muscle biopsy. The pathological features of myopathy in critically ill patients are somewhat complex, but most authors classify them into three main types:

- non-necrotising changes with atrophy of myofibres predominantly involving type II fibres (Figure 1), abnormal variation of muscle fibre size, angulated fibres, internalised nuclei, rimmed vacuoles, fatty degeneration of muscle fibres and fibrosis;
- necrotic changes with signs of regeneration (Figure 2);
- selective loss of thick myosin myofilaments (Figure 3) [8,10,15].

This classification is, however, somewhat arbitrary, with significant overlap of different changes in the same patient [8,18,28]. In contrast to the loss of myosin, considered to be a change that is pathognomonic for CIPM, atrophy of myofibres is less specific. Selective atrophy of type II myofibres is reported in myopathy, neuropathy, and disuse atrophy. Angular atrophic myofibres found multifocally or in small groups are traditionally interpreted as signs of denervation (Figure 4) [7,29,30]. Bolton and Breuer [7] stated that muscle biopsy in critically ill patients usually discloses ‘denervation atrophy and mild muscle necrosis’ and they interpret these findings as signs of critical illness polyneuropathy. Others, however, have described atrophic angular fibres as being characteristic signs of critical illness myopathy [15,31]. These discrepancies have resulted in striking differences in the classification of muscle biopsy changes in critically ill patients. Most authors who have systematically examined histopathological muscle changes in critically ill patients reported dominant myopathic changes [10,18,21,28,30]. The study carried out by Sander et al. [32] in eight quadriplegic areflexic patients with electrophysiological findings suggestive of CIP found normal nerve biopsy, but myopathic changes on muscle biopsy including myosin loss in all cases. Moreover, a significant proportion of cases examined has been interpreted as having a ‘mixed’ myopathic and neuropathic pattern. In particular, non-specific changes with dominant type II fibre atrophy were reported to be associated with CIP [15], but we found all types of myopathic changes to be associated with neuropathy [21]. Histopathological and immunopathological approaches are extremely valuable in the detection of various myopathic changes, but are less specific in the detection of acute denervation, especially in association with myopathic changes. The presence of muscle histopathological changes indicative of myosin loss has been proposed as one of four
major diagnostic features necessary to meet research diagnostic criteria for the diagnosis of definite critical illness myopathy [4]. Biopsy is, however, not a practical screening tool for larger observational epidemiological studies [18,21,33]. The methodological difficulties and reported coincidence of both types of neuromuscular involvement in the same critically ill patients led to the introduction of the descriptive term ‘critical illness polyneuromyopathy’ (CIPM) [5,6], critical illness polyneuropathy and myopathy [10,28,34], or CRIMYNE [35]. Some authors even maintain that the concept of critical illness polyneuropathy is outdated and have suggested the use of the clinical descriptive term ‘critical illness weakness’ followed by a description of myopathic, neuropathic, neuromuscular junction, metabolic and encephalopathic components [36]. Others have used a further descriptive term, ‘ICU-acquired paresis’ [27].

**Epidemiology**

At the beginning, the CIP–CIM interface seemed no more than a scientific curiosity, but research over the past 20 years has shown that CIP and CIM are the most frequent acute polynuropathies and myopathies encountered in critically ill patients [37]. Although the exact incidence is unknown, due to wide variation in diagnostic criteria and patient case-mix, available data regarding the incidence of neuromuscular involvement in critically ill patients are rather impressive (Table 1): 25–57% of critically ill patients (usually defined as >7 days in ICU (most or all mechanically ventilated, most or all with associated sepsis and multiple organ failure)) show clinically symptomatic weakness [21,28,34,38,39], and electrophysiological methods reveal abnormalities suggestive of CIP or CIPM in 21–100% of these patients [11,21,26,34,38–43]. Critical illness myopathy is a primary myopathy that has only been characterised in recent years [10]. Data on its incidence are lacking; evidence is, however, mounting that CIM is at least as frequent as CIP [10,13,18,27,28]. Douglass et al. [44] reported myopathy detected clinically in 36% of patients with status asthmaticus treated with corticosteroids and mostly with non-depolarising muscle-blocking agents (NDMBA). Campellone et al. [45], in a series of 100 consecutive patients after liver transplantation, detected clinical and electrophysiological signs of myopathy in seven cases (7%); biopsy confirmed myosin loss myopathy in five cases.

**Aetiology**

The concept of critical illness myopathy and neuropathy leads to a dichotomy in aetiological considerations of both types of involvement. Recently some authors have tended to replace this approach with the more descriptive concept of ‘critical illness polyneuromyopathy’ or ‘ICU-acquired paresis’ [27,28,34], and significant risk factors or predictors of CIPM have been sought. Sepsis or systemic inflammatory response syndrome (SIRS) is thought to be a leading cause of CIP [3,34,43,46,47]. Factors responsible for the systemic effects of sepsis, i.e. release of tumour necrosis factor (TNF), histamines and arachidonic acid metabolites, activation of the complement and cell adhesion systems, and formation of local free radicals may cause axonal degeneration [48]. As a mechanism, Bolton [48] suggested disturbances in the microcirculation of peripheral nerves. Using a bioassay by which toxic effects of patients’ sera on motor neurons could be determined quantitatively, Hund et al. [49] demonstrated the presence of a low-molecular weight factor (<3 kDa) in the serum of patients with CIP that kills cultured motor neurons. There exists a growing body of evidence that sepsis could also be responsible, at least in association with other factors, for the development of CIM or a myopathic component of CIPM [10,50–52]. Recently, the presence of SIRS has been found to increase the risk of clinically symptomatic CIPM development leading to artificial ventilation and this, together with the APACHE III score, has been used to estimate the risk [34]. Critical illness myopathy was originally reported as a complication of corticosteroid administration, either alone or in association with NDMBA [4,53,54]. Corticosteroids and NDMBA may serve as triggers, especially in necrotising and myosin-loss myopathies [15]. NDMBA may play a potentiating role, by virtue of a pharmacological denervation, that facilitates the toxic effect of other agents such as corticosteroids or inflammatory mediators. The functional denervation in CIP may provide a link between CIP and CIM. Pure motor neuropathy, on the other hand, has been attributed to the administration of NDMBA [55]. The causal role of NDMBA and corticosteroids in the development of CIM has been supported by numerous case reports, as well as by experimental data [56,57]. Recently, prospective studies reported controversial data on the significant independent influence of corticosteroids or NDMBA upon CIPM or ICU-acquired paresis development [27,34,40,58,59]. The differences between the studies might be explained by variable dosage of the corticosteroids administered, depending on the spectrum of diagnoses leading to critical illness. Another more general pitfall in the assessment of the aetiological influence of corticosteroids (in neuromuscular involvement of critically ill patients) could be a possibly predominant impact on the development of a myopathic component that is difficult to assess reliably in larger studies. Although administration of corticosteroids and NDMBAs has come to be avoided – or the drugs have been administered at the lowest possible doses – this practice does not seem to reduce the occurrence of neuromuscular involvement in critically ill patients. The influence of these drugs upon the development of CIPM and particular pathological subtypes of neuromuscular involvement thus remains to be established.
Table 1
Incidence of acquired neuromuscular involvement in critically ill patients (other than prolonged neuromuscular blockade): prospective observational studies

<table>
<thead>
<tr>
<th>Incidence of neuromuscular involvement (%)</th>
<th>Group definition (inclusion criteria)</th>
<th>Diagnostic criteria</th>
<th>Study sample (No of cases)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>57%</td>
<td>&gt;7 days in ICU, failure of at least 2 organs</td>
<td>Electrophysiological criteria of CIPM after 4 weeks Clinical signs of flaccid quadriplegia + electrophysiological criteria of CIPM after 4 weeks</td>
<td>61</td>
<td>Bednarik et al. 2005 [63]</td>
</tr>
<tr>
<td>28%</td>
<td>&gt;7 days in ICU, failure of at least 1 organ</td>
<td>Electrophysiological abnormalities</td>
<td>44</td>
<td>Coakley et al. 1998 [11]</td>
</tr>
<tr>
<td>84%</td>
<td>&gt;7 days in ICU, failure of at least 1 organ</td>
<td>Electrophysiological abnormalities</td>
<td>44</td>
<td>Coakley et al. 1998 [11]</td>
</tr>
<tr>
<td>25%</td>
<td>Mechanical ventilation &gt;7 days</td>
<td>Severe muscle weakness on day 7</td>
<td>95</td>
<td>DeJonghe et al. 2002 [27]</td>
</tr>
<tr>
<td>75%</td>
<td>Mechanical ventilation, SIRS, MOF</td>
<td>Electrophysiological criteria of CIPM after 3 weeks</td>
<td>73</td>
<td>Garnacho-Montero et al. 2001 [40]</td>
</tr>
<tr>
<td>33%</td>
<td>Mechanical ventilation</td>
<td>Clinical and electrophysiological criteria of CIPM</td>
<td>98</td>
<td>DeLetter et al. 2001 [34]</td>
</tr>
<tr>
<td>57%</td>
<td>Mechanical ventilation &gt; 3 days</td>
<td>Clinical and electrophysiological criteria of CIP on day 14</td>
<td>28</td>
<td>Druschky et al. 2001 [39]</td>
</tr>
<tr>
<td>100%</td>
<td>Mechanical ventilation, sepsis, MOF</td>
<td>Electrophysiological abnormality (decrease in CMAP amplitudes and/or fibrillations on days 2–5</td>
<td>9</td>
<td>Tennilä et al. 2000 [26]</td>
</tr>
<tr>
<td>21%</td>
<td>MOF</td>
<td>Electrophysiological signs of CIP on discharge from ICU</td>
<td>33</td>
<td>Mohr et al. 1997 [41]</td>
</tr>
<tr>
<td>70%</td>
<td>Sepsis, MOF</td>
<td>Electrophysiological signs of CIP</td>
<td>43</td>
<td>Witt et al. 1991 [42]</td>
</tr>
<tr>
<td>41% 82%</td>
<td>Sepsis, MOF</td>
<td>Clinical signs of CIP Electrophysiological signs of CIP</td>
<td>22</td>
<td>Berek et al. 1996 [38]</td>
</tr>
<tr>
<td>52% 29% (conventional treatment/treated with insulin)</td>
<td>&gt; 7 days in ICU</td>
<td>Electrophysiological signs of CIPM</td>
<td>206 157</td>
<td>Van den Berghe et al. 2001 [43]</td>
</tr>
<tr>
<td>36%</td>
<td>Status asthmaticus, corticosteroid treatment</td>
<td>Clinical signs of CIM</td>
<td>25</td>
<td>Douglass et al. 1992 [44]</td>
</tr>
<tr>
<td>7%</td>
<td>Liver transplantation</td>
<td>Clinical and electrophysiological signs of CIM</td>
<td>100</td>
<td>Campellone et al. 1998 [45]</td>
</tr>
<tr>
<td>30%</td>
<td>&gt; 10 days in ICU</td>
<td>Electrophysiological signs of CRIMYNE</td>
<td>92</td>
<td>Latronico et al. 2007 [35]</td>
</tr>
</tbody>
</table>

SIRS = systemic inflammatory response syndrome
MOF = multiple organ failure
CRIMYNE = critical illness myopathy and/or neuropathy
Multiple organ failure is a hallmark of systemic critical illness and failures of individual organ systems have also been proposed as possible causative factors for the development of neuromuscular involvement. A significant association between CIP development and the duration of mechanical ventilation and neurological failure was found in a cohort of 73 critically ill patients with multiple organ dysfunction [40]. In contrast, Campellone et al. [45] found a significant association between CIM development and the severity of critical illness with the presence of renal failure. These associations between CIP or CIM and other organ failures, however, raise questions as to whether neuromuscular involvement is not simply a part of systemic critical illness and whether or not it represents just another organ failure. Among other variables reported as significantly and independently associated with increased risk of CIP development were hyperosmolality, parenteral nutrition [40], hypoalbuminaemia, hyperglycaemia [27,42,43], hyperpyrexia [60], NDMBA [40,55,61], aminoglycoside antibiotics [37], catecholamines/vasopressors [43,58], increased age [40], and female sex [27]. Most of these factors are intrinsically...
related to sepsis and severity of critical illness and their causal relationship to CIPM is unclear [62,63]. In a recent prospective study [34], the development of critical illness polyneuromyopathy (CIPM) was significantly associated with APACHE III score (as a quantitative index of disease severity) and the presence of SIRS. These factors were used to estimate the risk of developing CIPM. In a prospective cohort study [63], we have demonstrated an increased risk of CIPM development in the presence of SIRS and especially in its longer duration. Another variable significantly associated with CIPM development was the severity and duration of multiple and some individual organ failures, namely neurological, cardiovascular and respiratory failures (and closely correlated duration of ventilation support).

The discrepancy between studies on the aetiology of neuromuscular disorders in critically ill patients could be explained by the varying severity of neuromuscular involvement in the cases studied and by different criteria for CIP, CIM or CIPM. Recent comparable prospective studies focused on clinically symptomatic CIPM in patients on mechanical ventilation [27,34], while others relied on more sensitive electrophysiological or histological criteria that are able some-what to extend differentiation between neuropathy and myopathy.

CONCLUSIONS

Current knowledge of the incidence and pathophysiology of acquired neuromuscular involvement in critically ill patients is limited, which is a fact that has restrictive implications for clinical practice. Studies published to date are limited by a low number of analysed patient cases; however, inconsistent eligibility criteria and inconsistent case definitions preclude the combination of results by meta-analysis. Better animal models and improved epidemiological studies on the incidence of acquired neuromuscular involvement and on all potential risk factors with complete evaluation of all ICU patients at risk are required. Acquired neuromuscular weakness as a result of critical illness is more common than previously recognised and may result in substantial excess morbidity, mortality, and costs. It is questionable whether forthcoming aetiological studies on neuromuscular involve-ment in critically ill patients should adhere to the concept of CIPM or try to differentiate different components of neuromuscular component and assess them separately. The possibility of achieving reliable differentiation of myopathic and neuropathic components in larger prospective studies is limited not only by the inherent limitations of every method, but also by the frequent association between myopathy and neuropathy in critically ill patients. The neuromuscular system is probably diffusely, although patchily, involved in critical illness. Neuromuscular involvement in critically ill patients probably represents a continuum of neurogenic and myogenic changes of varying severity and progression over time. Studies utilising clinical and electrophysiological techniques should avoid strict categorisation of cases into CIP or CIM, but might do better to employ description of the main electrophysiological syndromes: isolated sensory neuropathy, isolated motor syndrome, mixed sensorimotor involvement, and decreased sarcolemnic excitability. Forced categorisation into myopathy and polyneuropathy frequently leads to misinterpretation of abnormalities and discrepancies between studies.

Muscle biopsy, as an invasive technique, is not a suitable diagnostic tool for large epidemiological studies. Nevertheless, it would be helpful to design a large multicentre study employing biopsy and differentiating all the histopathological types of CIM to address the dilemmas of the incidence of CIP and different types of CIM; of the correlation between electrophysiological findings and histopathological subtypes; and of the risk factors for different pathological subtypes of neuromuscular involvement. Acquired neuromuscular weakness in critically ill patients should be regarded as dysfunction or failure of a further (neuromuscular) system that could be detected early during critical illness. Records of neuromuscular function should be included as measures of outcome in all interventional ICU studies. Measurements to detect early signs of neuromuscular dysfunction (such as, possibly, decrease of CMAP amplitude, as shown recently by Latronico et al. [35], and decrease of sarcolemnic excitability) should be sought and validated.

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REFERENCES


A meeting of the Department of Neurology management

Sonography laboratory
Lecture hall

Speech therapy examination