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日本医学会分科会 理事長・会長 殿

> 日本医学会 会長 髙久



ICD-10の一部改訂に係る意見について(依頼)

標記について、別紙のとおり厚生労働省大臣官房統計情報部より本職宛てに依頼がありました.

ICD-10 の平成 27 年(2015 年)の一部改訂について、日本としての意見をとりまとめるにあたり、日本医学会からの意見を求めています.

ご多忙の折とは存じますが、ご意見がありましたら別紙様式へ記入のうえ<u>9月22</u> 日(月)までに本会宛てお送りいただけますようご協力のほどお願い致します.

本件の担当: 日本医学会 長門宏子

1日103-3946-2121 (内 2041)

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事 務 連 絡 平成26年6月16日

日本医学会 会長 髙 久 史 麿 殿

> WHO 国際統計分類協力センター長 厚生労働省大臣官房統計情報部企画課 国際分類情報管理室長 谷 伸 悦

ICD-10の一部改正に係る意見について(依頼)

時下ますます御清祥のこととお慶び申し上げます。

日頃より ICD の改訂・改正に関する検討につきましては、御協力を賜り誠にありがとうございます。

さて、ICD-10 につきましては、毎年、各国からの意見をもとに、WHO において一部改正が行われているところですが、例年、年度末の3月31日が意見提出の期限となっております。

つきましては、平成27年(2015年)の一部改正に向けて、日本としての 意見をとりまとめるにあたり、貴会からの御意見を頂戴いたしたくお願い申し 上げます。

別添1の留意事項に従って、9月30日(火)</u>までに以下の担当へ御提出いただきたくお願いいたします。

なお、期日を過ぎて御提出いただいた御意見については、翌年の 2016 年改正 分としてお取り扱いいたしますことを申し添えます。

なお、ICD については、現在 ICD-11 への改訂に向けての作業が続いているところですが、ICD-11 の国内導入までは相当の期間を要すると考えられることから、当面は ICD-10 の適用が継続すると考えられます。

つきましては、一部改正の機会を活用し、適切な ICD となるよう、積極的な 御意見の提出を何卒よろしくお願い申し上げます。

【担 当】

厚生労働省大臣官房統計情報部 企画課国際分類情報管理室(及川、中濱) 〒100-8916 東京都千代田区霞が関 1-2-2

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E-mail: icdoffice@mhlw.go.jp

ICD-10 の一部改正に係る意見についての留意事項

1. 最新の ICD-10 について

ICD-10 は毎年、一部改正が行われており、2013 年の改正までが確定しております。最新の ICD-10 の内容は以下の WHO の URL をご参照ください。

http://www.who.int/classifications/icd/icd10updates/en/

2. 御意見の内容について

以下に合致したものについて、御意見として御提出ください。

- ① 医学、医療の進歩に伴い、**現在の分類が学術的な合意と食い違っているもの**。根拠(推計数、論文等)が明確であること。
- ② ICD という国際標準となる分類の改正に対する意見として、先進国だけでなく、発展途上国も含め世界的な対応が必要となるものであること。
- ③ 日本から提出する意見として、<u>その分野を専門とする学会の総意が</u> **得られているもの**であること。

3. 提出について

- ① 提出いただくもの
 - ・WHO ICD URC 提案票 (別添 2-1 (日本語)、2-2 (英語))
 - ・添付資料:御意見の根拠となる論文、統計データ等
- ② WHO ICD URC 提案票 (別添 2-1, 2-2) 記載の注意点
 - 各項目とも記載の漏れが無いようご注意ください。
 - ・WHO への提出は別添2-2 (英語)を元に行いますが、専門委員会の審議資料としては、別添2-1 (日本語)を元に行いますので、お手数ですが、英語、日本語とも作成をお願いいたします。
 - ・各項目への記載例については、別紙1-1、1-2をご参照ください。
 - WHO で行われる具体的な議論の経緯については、別紙1-3をご参照ください。

③ 提出先

以下まで電子メールにより御提出ください。

厚生労働省大臣官房統計情報部企画課国際分類情報管理室

担当:及川、中濱

E-mail: icdoffice@mhlw.go.jp

Tel:03-3595-3501

④ 期限

平成26年9月30日(火)

上記期日を過ぎて御提出いただいた御意見については、翌年の 2016 年改正分としてお取り扱いいたします。

WHO ICD URC 提案票(<u>日本語</u>)

<u>必ず、別添2-2(英語)も併せて作成してください。</u>

申請	学会名	
申請年月日		平成 年 月 日
担当	者名	
担当	者連絡先(Tel/E-Mail)	() – / @
関係	する ICD-10 コード及び	
コー	ドタイトル(分類名)	
関係	する傷病名	
提	□ 既存コードの削除	□ 既存のコードタイトル(分類名)の修正
条 内	□ 既存コードの移動	□ コードに含まれる傷病名等の追加
提案内容の概要	□ 新規コードの追加(既存コード (選択:内容例示の包含用語/除外用語/索引の用語)
概	の細分)	□ 既存の傷病名等に割り当てられているコードの修正
要		(選択:内容例示の包含用語/除外用語/索引の用語)
	□その他	
具体	的な提案内容	
提案	理由	

必ず、別添2-2(英語)も併せて作成してください。

		中 空 加 V 中 关		=
工ビ		患の概念・定義		
ビデン		症状		
ンス		病因		
	臨床所見			
		その他		
	(基礎疾患・合併症・予		症・予	
		後等の情報、診断	基準や	
		治療法の有無、関連指針		
		等についてご記載	くださ	
		(')		
	疫	学情報		
		罹患者数・率、 有病者数・率	国内	
			世界	
		死亡者数・率	国内	
			世界	
		その他(公衆衛生上の		
		重要性、性差、妨	好発年	
		齢・好発地域等に	こつい	
		てご記載ください	(\)	
	医	学的コンセンサスの	の程度	
	英	英文根拠論文		
備者	z .			
7/用4	J			

WHO ICD URC Draft Application Form (English)

Name of academic society								
Date of application		dd/mm/yy	ууу:					
Resp	onsible applicant							
Cont	act info. (tel./email)	()	_	/	/	@		
Prin	nary Code Affected							
(Rela	ated ICD-10 code and its							
title/	classification name)							
Rela	ted disease names							
lew	☐ Deletion of existing						of an existing	
Overview	☐ Movement of existing	ng code		\Box A	addition of a	an inclusion	n/exclusion to	erm
Ove	☐ Addition of new cod	le (making			[Please	e select: incl.	/ excl. of tabul	ar list, index entry]
	subdivisions of an ϵ	existing coo	de)		Correction of	r clarificati	ion of a code	assignment for
				a	n existing t	erm		
					[Please	e select: incl.	/ excl. of tabul	ar list, index entry]
	☐ Other							_
Deta	iled description of							
prop	osal							
Rationale/Background of								
proposal								

Continued

	_	0: 1:1	1.	
nce	D	efinition of the d	disease e	ntity
Evidence		Sign/Symptom		
E		Etiology		
		Clinical findings		
		Other (e.g. informa	ation	
		of underlying condi	tion,	
		complication or		
		prognosis, diagnosis	s	
		criteria, treatment,		
		related guidelines e	etc.)	
	E	pidemiological ii	nformati	on
		Number and rate	Japan	
		of incidence/	Japan	
		prevalence	World	
		Number and rate of death	Japan	
			World	
		Other (e.g. importance		
	from a public health		lth	
		perspective, gender		
		difference, common age,		
		regional aspect etc.)		
	De	egree of medical co	nsensus	
-		apporting English		
		publications		
Ren				

分類構造や統計への影響が大きいコードの削除・ 移動・追加や割り当てコードの大幅な修正は、基 本的には3年毎の改正の際に適用されます。また、 提案には特に十分な根拠が求められますのでご 留意ください。

「CD URC 提案票(日本語)(案)

別紙1-1

えば、既存のICDコードの疾患が古 概念でコードとして不要と考えられる め削除すべきと考えられる場合(他 コードの追加等を要さないもの)。	<u>必ず、別添2-2(英語)も併せて作成してください。</u>
申請 右の事例のように、既 (例) 担当 右の事例のように、既 (例) にICDコードがあるけ 183 下肢の静脈瘤 れども(例では184)、 184 痔核 185 食道静脈瘤 位置でないため修正 サ K63 腸のその他の疾合など。 (略) 関係 コー 関係 K65 腸膜炎	月 日 既存のICDコードについて、一般的に使用される名称が変わってきている場合や名称を適正化することによって概念がより適切に捉えられるようになるため英語のコードタイトルの修正を要する場合など(例) C81 ホジキン病 → C81 ホジキンリンパ腫
提 □ 既存コードの削除 案 □ 既存コードの移動 ←	□ 既存のコードタイトル (分類名) の修正 □ コードに含まれる傷病名等の追加 ≪ (選択:内容例示の包含用語/除外用語/索引の用語) □ 既存の傷病名等に割り当てられているコードの修正 ≪
要 当てはまる 選びにくい 択肢、詳細	(選却・内容例示の包含用語/除外用語/索引の用語) 選択肢がない場合や 場合は、「その他」を選 は「具体的な提案内 説明ください。
[存のコードが大まか (例) 概念であるところ、 K64 痔核 り詳細な細分コード ↓ 必要と考えられる場 K64 痔核 でか新しい疾患概念 が確立したために既 K64.1 第2度痔核 では、 K64.1 第2度痔核 では、 K64.1 第2度痔核 では、 K64.1 第2度痔核 では、 K64.9 痔核、詳細 でなど。	既存のコードに含まれる傷病を明確にする ために例示を追加する場合など (例) K64.0 第1度痔核 脱肛を伴わない痔核(出血性) K64.1 第2度痔核 脱肛を伴わない痔核(出血性)
提案理由 なぜ上記提案のような改正・修正が 必要かについて医学的・公衆衛生 的側面等から御説明ください。提案 の根拠としてURCに提示されます。	内容例示の既存の例示用語や索引の用語 等に振られているコードが間違っており、修 正が必要な場合など (例:第3巻 索引) 痔核NOS K64.0 ← 間違え ↓ 痔核NOS <u>K64.9</u>
	裏面に続く

以ず 則沃り−り (革託) t 仕れて作品してください

		必り、別称と一と(央暗)も併せて作成してください。
x	疾患の概念・定義	
ビデ	症状	
ンス	病因	
	臨床所見	エビデンスの各項目では、基本的な疾患概念や公
	その他	衆衛生状の重要性など御提案を支える種々の情 報を御記入ください。御提案をWHOや各国に対し
	(基礎疾患・合併症・予	て主張していく際に活用させていただきます。
	後等の情報、診断基準や	
	治療法の有無、関連指針	

WHO ICD URC 提案票(<u>日本語</u>)【記載例】

必ず、別添2-2(英語)も併せて作成してください。

申請学会名	〇×学会			
申請年月日	平成 \times \times 年 \times \times 月 \times \times 日			
担当者名	厚生 ふうか			
担当者連絡先(Tel/E-Ma	1) (XXX)XXX-XXX / AAA_BBB@CCC.JP			
関係する ICD-10 コード及	び G10-G14			
コードタイトル (分類名)	主に中枢神経系を障害する系統萎縮症			
関係する傷病名	ポリオ後症候群			
提 □ 既存コードの削降 案 内 □ 既存コードの移動 容 ■ 新規コードの追加 の () () () () () () () () () (□ コードに含まれる傷病名等の追加			
概 の細分) 要	(選択:内容例示の包含用語/除外用語/索引の用語)			
□ その他	(区)(-13年797.92 区日/旧町/ 例717日間/ 外月97日間/			
具体的な提案内容	第2巻 内容例示 主に中枢神経系を障害する系統萎縮症 (G10·G13G14) ・・・ G13* 他に分類される疾患における主に中枢神経系を障害する系統萎縮症 G14 ポリオ後症候群 灰白髄炎後症候群 除外:灰白髄炎<ポリオ>の続発・後遺症 (B91) < 上記に伴い必要となる除外事項の追加> B91 灰白髄炎<ポリオ>の続発・後遺症 除外:ポリオ後症候群 (G14) M89.6 急性灰白髄炎<ポリオ>の分類が必要な場合は、追加コード (B91) を使用する。 除外:ポリオ後症候群 (G14) < 上記に伴い必要となる第3巻 索引用語の追加> 灰白髄炎後-病態を参照 ・オステオパシー<骨障害> M89.6 ・症候群 G14			

	症候群-疾患<病>も参照
	• • •
	-ポリオ後(灰白髄炎後)G14
提案理由	本症候群は不可逆的で、治療できず、運動ニューロンの進行性の機能障
	害という観点から、ポリオ後遺症に分類すべきではない。つまり ICD-10
	で A80 および B91 で規定されるものと異なり、新たにカテゴリーを設け
	るべきである。

<u>必ず、別添2-2(英語)も併せて作成してください。</u>

	 長患の概念・定義		
	症状		ポリオ発症後長期間経過してから起こる、筋の脱力や疲労等
ζ	病因		最も受け入れられているのは 1875 年の Charcot による
`			Super-training 理論で、ポリオウィルスの急性感染により、ウィルス
			が脊髄全角細胞を傷害し、神経細胞の可塑性のためいくつか運動単位
			が除神経され、それらの修復が起こる。30年から40年の潜伏期の後
			神経修復の結果形成された巨大運動単位の代謝の要求の影響で運動神
			経の障害がはじまり、新たな神経萎縮等を起こす。
	臨床所見		進行性の筋萎縮、脱力、筋関節痛
	その他		UNICEFESP-EPM の研究によると、随伴症状として睡眠障害、めま
			い、記憶障害等がおこる。他の診断に重要な所見はポリオ罹患から⊄
			経過期間であり、30年から40年であることが多い。
	(·)		
疫学情報			
	罹患者数・率、	国内	北九州市でポリオ罹患者のうち 85%
	有病者数・率		北九州市では人口 10 万対 18(Takemura, 2004)
		世界	英国で罹患率 77% (Bruno, 1997)、NZ で 47% (CHETWYND, 1993)
			他
			欧州で 25 万人、世界で 2 千万人(BOSH, 2004)
	死亡者数・率	国内	
		世界	
	その他(公衆領	•	世界各国をみると、ポリオ罹患者のうちポリオ後症候群になるのは 50
	の重要性、性	差、好	\sim 80%という報告が多い。 2003 年に欧州で PPS に関する団体と欧州
	発年齢•好発均		議会メンバー20名がポリオに関する欧州連合を創設しており、ポリス
	についてご記	載く	後症候群が疾病分類として認識されることが喫緊の課題となってい
	ださい)		る。
医	学的コンセンサスの科	呈度	
英文根拠論文			別添参照
英	- 又恨拠論又		りは多いの

ICD Update Platform

Home | ICD-10 | Search/Filter/Report | All Groups

User logged in :icdoffice User Profile Documents >

Post-polio syndrome

Proposal ID: 1116 - Proposal State: Accepted Proposal for Update

Implementation Date: 1/2010

Originator: Lori Moskal - Last Update made by :Lori Moskal

Creation Date: 22-Aug-2006 23:15 CET - Last Update: 09-Jan-2009 19:31 CET

Previously Discussed in the group(s): MBRG

Primary Code Affected: G10-G14 Secondary Codes Affected: None

Volumes Affected: 1,3

Proposal Type: Addition of new code

Change Reason: Need to reflect a change in clinical knowledge

Detailed Description

Add code to block level:

List of three-character categories Diseases of the nervous system

(G00-G99)

Systemic atrophies primarily affecting the central nervous system (G10-G13G14)

G10 Huntington's disease

G11 Herediary ataxia

G12 Spinal muscular atrophy and related syndromes

G13* Systemic atrophies primarily affecting central nervous sysstem in diseases classified elsewhere

G14 Postpolio syndrome

Add excludes note:

B91 Sequelae of poliomyelitis

Excludes: postpolio syndrome (G14)

Add category:

Systemic atrophies primarily affecting the central nervous system (G10-G13G14)

G14 Postpolio syndrome

Postpolio myelitic syndrome

Excludes: sequelae of poliomyelitis (B91)

Add excludes note:

M89.6 Osteopathy after poliomyelitis

Use additional code (B91), if desired, to identify previous poliomyelitis.

Excludes: postpolio syndrome (G14)

Special tabulation lists for mortality and morbidity

Tabulation list for morbidity (298 causes)

Note: These lists were adopted by the World Health Assembly in 1990 for the tabulation of data. They are described, and their use is explained, in Volume 2, the Instruction Manual.

Tabulation list for morbidity

129

Other diseases of the nervous system

G10-G13 G14, G21-G26, G31-G32, G36-G37, G46-G47, G60-G73, G90-G99

Volume 3
Add subterms and lead term:

Postpoliomyelitic - see also condition

- osteopathy M89.6
- syndrome G14

Postpolio syndrome G14

Syndrome - see also Disease

- postphlebitic 187.0
- postpolio (postpoliomyelitic) G14
- ~ postvagotomy **K91.1**

Rationale

1. Justification:

1.1 Definition

PPS is a neurological disorder characterized by a new or reoccurring muscular weakness and/or muscular fatigue abnormal in individuals who had acute poliomyelitis, many years before (DALAKAS, 1995).

Although this aggravation is a late effect of poliomyelitis, it is about a defined nosological entity, that we believe cannot be identified as a sequelae, since it characterizes itself as an evolving disease of diverse etiology and physiopathology compared to basic disease. PPS is classified as motor neurone disease due to its histological and clinical chart being intimately related with the functions of inferior motor neurons. (DALAKAS, 1995).

1.2 Risk Factors

The risk factors to the development of PPS have not, until now, been elucidated accordingly. Based on repeated observations and epidemiological reports, however, the following factors seem to be associated with a precautious beginning of PPS: (1) New symptoms appear first, or could be firstly in the limbs previously damaged and in patients with more aggravated paralysis; (2) Precautious bulbar or respiratory difficulties occur in residual patients with loss of muscular strength of bulbar intervention and respiratorymusculature; and (3) Symptoms occur precautious in patients who have acute poliomyelitis in a later age.

1.3 Physiopathology

Jubelt et al in 1999 conducted a study demonstrating the existence of 9 theories that explain the physiopathology of PPS, however the most welcome is of the "Super-training" formulated initially by Charcot, in 1875, and complimented and proven through electrophysiological studies by Dalakas et al since 1995.

In the acute infection of poliomyelitis, the virus damages the cells of the anterior medullary horn partially or totally, with denervation of some motor units through the neuronal plasticity, blossoming occurs renovating the denervated fibers although this last one depends on the number of preserved neurons after this period of recovery, we go to a period of latency, also called stability plateau. According to the theory of "super-training", thirty or forty years after the acute disease through the metabolic solicitation request from the giant motor units formed due to the neuronal recovery, there is the beginning of motor neuronal failure, mainly in the distal portions of the **axonius**, bringing it to a new denervation known as new weakness and muscular atrophy.

1.4 Clinical Aspects

The symptoms and signals of PPS include a combination of muscular skeletal symptoms of the progressive muscular atrophy, post-poliomyelitis (AMPP) (DALAKAS, 1995).

These symptoms classified as more frequent include: new muscular weakness, new muscular atrophy with or without the presence of muscular and articular pain. There is still other symptoms although less frequent in evidence: muscular weakness of bulbar innervations, intolerance to cold, myalgia, fasciculations, new breathing difficulties, and sleep apnea.

In the research developed in UNIFESP-EPM, we have verified the presence of other forms of clinical occurrences including sleep disorders, increase of corporal weight, memory disorders, dizziness, syncope, and morning headache related to sleep disorder. Studies conducted in diverse populations show the same characteristics having percentual as the only variation. Another important

factor is the duration of the interval between the acute disease and the beginning of PPS symptoms is a determining factor with the highest rate of incidence of thirty to forty years.

1.5 Incidence and Prevalence

This may vary according to the definition of PPS, from diagnosed criteria used and also of population research undertaken.

Brazil: In depictive studies made in the Neuromuscular disease ward of Escola Paulista de Medicina UNIFESP, were found respectively 68% and 77.2% of incidents among the patients that were consulted (OLIVEIRA, 2002; QUADROS, 2004).

Japan: In a study conducted in the RECUPERATION WARD of Cosmos Hospital, Usuki city, County of Ohita, found 85% of incidents and a prevalence of PPS in Kitakyushu was of 18 cases/100,000 inhabitants (TAKEMURA, 2004).

Norway: The study in 1994 was made through national medical reports, and of social situations, and in a total of 2392 cases of poliomyelitis victims, many of whom registered in "the national association of polio victims" in Norway with a prevalence of 85% of PPS (GRELH, 1996).

Germany: A study conducted found evidence of 68.07% of PPS in patients. (TIENVOLL, 1997). New Zealand: A loss of muscular strength was related in 47%, and it is estimated that there maybe a number of 3000-5000 polio

survivors in New Zealand, that may be suffering from PPS (CHETWYND, 1993).

Australia: It is estimated that at least 20,000 - 40,000 individuals developed paralyzing poliomyelitis from 1930-1988.

Scotland: A study conducted in Edinburgh found 67% of PPS (PENTLAND, 1999).

Denmark: 54% of PPS was found (LONNBERG, 1992).

Sweden: 18.6% of PPS was found in a research (AHLSTROM, 1993).

Canada: It is estimated that 66,000 survivors exist and that approximately two thirds have or will develop PPS (URIADKA, 1997).

United Kingdom: 74,280 cases were reported and the incidence of PPS found was 77% (BRUNO, 1997).

Europe: There is nearly 250,000 patients of PPS in Europe and 20,000, 000 around the world (BOSCH, 2004).

2. Final Considerations

Considering that PPS is an evolving clinical situation irreversible and incurable, related to the progressive dysfunction of the motor units, it cannot and should not be classified as a polio sequelae. Sequelae means (see thesaurus/dictionary). In ICD-10 a poliomyelitis situation is contemplated with the codes: A-80, A-80.0, A-80.1, A-80.2, A-80.3, A-80.4, A-80.9 and B-91, being in this way not possible to characterize PPS as a nosological entity defined in these categories.

In November of 2003, a group of associations of PPS in Europe, and 20 members of the European Parliament met and agreed to create the European Union of Polio having as a goal to obtain recognition and funds of the European Parliament (BOSCH, 2004).

It becomes fundamental for post-polio syndrome to be recognized as a nosological entity and it should have a specific

characterization in ICD-10 due to the relevance of the legal and social assistance.

3. References:

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Proposal held over in 2007. URC secretariat

This proposal was accepted in 2008. URC

Supporting Publications (Upoaded Files)



Proposai Summary

This item was discussed at the URC session of WHOFIC in Trieste, 2007 and it was decided to refer it to the MbRG for further work on the best fit within the classification for a new code and possible subcategories.

10.4.08: Revised proposal attached for comments.

Comments

23-May-2007 14:50 CET by Michael Schopen

Comment attached to the vote of the user for Round 1 of year 2007. Voted:Can't Decide

We are seeking expert advice.

28-Jun-2007 05:31 CET by Julie Rust

Comment attached to the vote of the user for Round 1 of year 2007. Voted:Can't Decide

Seeking clinical advice

21-Aug-2007 10:06 CET by Robert Jakob

Comment attached to the vote of the user for Round 2 of year 2007. Voted:Can't Decide

seeking advice

30-Aug-2007 11:56 CET by Olafr Steinum

Comment attached to the vote of the user for Round 2 of year 2007. Voted:Yes

We support this proposal. Our neurologist suggests, however, that subcategories of G14 might be useful.

06-Sep-2007 09:23 CET by Michael Schopen

Comment attached to the vote of the user for Round 2 of year 2007. Voted: Can't Decide

We hope to have expert opinion for Trieste.

07-Sep-2007 09:37 CET by Julie Rust

Comment attached to the vote of the user for Round 2 of year 2007. Voted: Can't Decide

Still following up clinical advice - hopefully for the meeting in Trieste.

10-Sep-2007 19:40 CET by Donna Pickett

Comment attached to the vote of the user for Round 2 of year 2007, Voted:Can't Decide

Still obtaining clinical advice

10-Sep-2007 22:02 CET by Lori Moskal

Comment attached to the vote of the user for Round 2 of year 2007. Voted: Can't Decide

Still trying to get clinical input.

18-Feb-2008 03:32 CET by Julie Rust

Comments from Australia

There are a number of options for the inclusion of this syndrome in ICD-10;

- 1. Add 'post-polio syndrome' as an inclusion term at G12.2 Motor neuron disease (attached rationale and other papers indicated that it is a motor neuron disease) and also code the B91 Sequelae of poliomyelitis
- 2. Create a new code at G14 and expand category G10 G13 (to G14) to include this also code out B91.

However, we think that this proposal raises the bigger issue of who decides when a condition is a recognised disease (and what criteria are applied?). There could be similar arguments for inclusion in ICD-10 of a number of other conditions and we're not sure that the responsibility for inclusion rests with the URC.

18-Feb-2008 03:36 CET by Julie Rust

ICD Neurological application

Just for information, ICD-NA (1997) has expanded the category B91 in the following manner:

B91 Sequelae of poliomyelitis

B91.-0 Progressive postpolio muscular atrophy

B91.-1 Postpolio pain syndrome due to joint deformity

B91.-2 Postpolio pain syndrome, idiopathic

28-Mar-2008 20:00 CET by Donna Pickett

Post Polio Syndrome

The U.S. has received a response from the Neuromuscular Disease Section of the American Academy of Neurology (AAN) regarding Post-Polio Syndrome.

They support the creation of a new code and note that post polio syndrome is probably not a late effect. Based on this information the U.S. agrees with the creation of a new category G14 which could be titled to allow for further expansion in the future, and create a new code under the new category (G14.x).

Below is a discussion thread from Medlink Neurology that accompanied the AAN response.

Etiology

It is now recognized that normal aging alone cannot explain the development of post-polio syndrome because the normal loss of anterior horn cells and motor units does not become prominent until after age 60 (Jubelt and Cashman 1987). More important than a patient's chronological age is the interval from their acute polio to the onset of post-polio syndrome, an interval that averages between 30 and 40 years (Jubelt and Cashman 1987). The presently accepted most likely etiologic possibilities are degeneration of enlarged motor units, a chronic persistent poliovirus infection or an immune-mediated disease.

Degeneration of enlarged motor units. The enlarged motor units that develop via sprouting after the acute polio may never fully stabilize (Wiechers 1985). Findings from single fiber electromyographic (SFEMG) studies reveal that the largest motor units are more likely to become unstable later in life (Cashman et al 1987a; Emeryk et al 1990), and with increasing time from the acute polio, neuromuscular transmission becomes more unstable, as increased jitter and blocking occur (Wiechers and Hubbell 1981). Spontaneous denervation activity, jitter, and blocking occur more frequently in symptomatic muscles (Ryniewicz et al 1990; Maselli et al 1992). These findings are supported by muscle biopsy studies that describe an increasing number of angulated fibers accumulating over time (Dalakas and Illa 1991). This is followed by degeneration of axonal branches as demonstrated by the appearance of small group atrophy (Drachman et al 1967; Cashman et al 1987a). This can be followed by large group atrophy suggesting neuronal degeneration (Dalakas and Illa 1991). It has been frequently hypothesized that the increased metabolic demand of an increased motor unit territory results in premature exhaustion and death of the motor neuron (Jubelt and Cashman 1987). Even though there are no definitive studies examining the cell soma to prove this theory; electrophysiologic and muscle biopsy data appear to be supportive. The overuse of weakened muscles results in excessive muscular fatigue (Sharma et al 1994; Grimby et al 1996; Agre et al 1998; Sunnerhagen et al 2000; Thomas and Zijdewind 2006), which appears to contribute to the excessive metabolic demand on motor neurons and the premature exhaustion.

Chronic persistent poliovirus infection. Poliovirus and other picornaviruses can persist in the CNS of animals and cause delayed or chronic disease (Jubelt and Cashman 1987: Destombes et al 1997). Poliovirus and other enteroviruses can also persist in the CNS and systemically in immunodeficient children (Jubelt and Cashman 1987). Studies in tissue culture have found that poliovirus mutants can persist without killing the host cell (Colbere-Garapin et al 1989; Borzakian et al 1992) and can also persist in neurons (Pavio et al 1996). Support for the persistent poliovirus hypothesis was enhanced by the findings of Sharief and colleagues (Sharief et al 1991), who demonstrated poliovirus antibodies and poliovirus-sensitized cells in the CSF of post-polio patients. Leon-Monzon and Dalakas (Leon-Monzon and Dalakas 1995) found elevated IqG poliovirus antibodies in the sera of post-polio syndrome patients as compared to controls; however, ALS patients had similar elevated levels. Other investigators have been unable to find poliovirus antibodies in the CSF of post-polio patients (Kurent et al 1979; Dalakas et al 1986; Salazar-Grueso et al 1989; Melchers et al 1992; Jubelt et al 1995). CSF specimens have also been examined for the presence of poliovirus RNA by polymerase chain reaction, and the majority of studies have been negative or inconclusive (Melchers et al 1992; Leon-Monzon and Dalakas 1995; Leparc-Goffart et al 1996; Muir et al 1996). The most positive study was that of Julien and colleagues (Julien et al 1999) who detected poliovirus genome sequences in the CSF of 11 of 20 post-polio syndrome patients but in none of the 20 control patients. These same authors had reported similar findings, post-polio syndrome 5 of 10 positive, controls 0 of 23 positive, in an earlier study (Leparc-Goffart et al 1996). Conclusive viral isolation and histochemical or hybridization studies have not as yet been reported using spinal cord tissues and will be required to resolve this possibility. An immune-mediated disease. The strongest support for an inflammatory or immune-mediated mechanism for post-polio syndrome stems from the study of Pezeshkpour and Dalakas (Pezeshkpour and Dalakas 1988) in which inflammation in the spinal cords of seven post-polio patients was found. It consisted of both perivascular and parenchymal lymphocytic infiltrates, neuronal degeneration, and active gliosis. All changes were more prominent in three patients with new weakness. Other findings that support this hypothesis are the findingoligoclonal bands in the CSF (Dalakas et al 1986) and activated T-cells in the peripheral blood(Ginsberg et al 1989). Others have not found oligoclonal bands in post-polio syndrome patients (Cashman et al 1987a; Salazar-Grueso et al 1989); however, other histologic studies suggest an immunemediated or viral-induced pathogenesis or at least an inflammatory mechanism. Miller (Miller 1995) examined the spinal cord from one post-polio patient and found perivascular intraparenchymal chronic inflammatory infiltrates primarily composed of B lymphocytes with rare macrophages and no T-cells. Kaminski and colleagues (Kaminski et al 1995) found inflammation in the spinal cords of 8 of 9 post-polio syndrome patients. More recent studies supporting an immune-mediated process is the finding of inflammatorycytokines (TNF-alpha, IFN-gamma, IL-4, IL-10) in the CSF of post-polio syndrome patients (Gonzalez et al 2002; 2004).

21-Apr-2008 23:54 CET by Olafr Steinum

Let's go!

Let us go for G14 and move the proposal to URC.
22-Apr-2008 10:05 CET by Marion Mendelsohn proposal to URC
I agree with the comment of Olafr
29-Apr-2008 15:52 CET by Ulrich Vogel Let's move it to URC
We agree with Olafr's comment.
18-Jun-2008 20:02 CET by Robert Jakob
Comment attached to the vote of the user for Round 1 of year 2008. Voted:Yes
clear evidence for this change
28-Aug-2008 20:10 CET by Lori Moskal
Comment attached to the vote of the user for Round 2 of year 2008. Voted:Yes
Should there be an excludes note added at M89.6 Osteopathy after poliomyelitis Excludes: Post-polio Syndrome (G14)
30-Aug-2008 11:14 CET by Glen Thorsen
Comment attached to the vote of the user for Round 2 of year 2008. Voted:Yes
Support Lori's suggestion of exclusion note

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ICD-10の一部改正(2015年)スケジュール

平成26年 (2014年) 改正意見提出期限(学会→事務局) 9月末 学会一事務局間 相互調整 第一次修正案提示 (事務局→学会) 11月 学会一事務局間 相互調整 平成27年 (2015年) 第二次修正案提示 (事務局→学会) 1月 日本提出意見決定 3月 (社会保障審議会統計分科会ICD専門委員会) WHO URC(分類改正改訂委員会)へ意見提出 6月 第1回投票 各国からの疑義照会、反対意見への対応のため 学会一事務局間 相互調整 9月 第2回投票 2015年改正内容決定 10月 (WHO国際統計分類ネットワーク年次会議)