参考資料３

２００３年世界保健機関国際分類ファミリー協力センター分類改正委員会における保留１４項目（原文）

No.01 **Proposed by: Australia**

Change to Volume: I & III Type of proposal: Major

Description (term/code): Morbidly adherent placenta

Proposal for update:

Background

This proposal for change was originally tabled in the URC papers for 2001 and the background to the change is reproduced below:

Placenta accreta, placenta percreta and placenta increta are all currently classified as retained placenta (O72.- with haemorrhage, O73.0 - without haemorrhage). In Australia, a significant number of these cases are now being diagnosed antenatally, and a code from the Labour and Delivery category is not appropriate. The NCCH proposes to introduce a new code in category O43 *Placental disorders*, a category within the block *Maternal care related to the fetus and amniotic cavity and possible delivery problems* which may then be assigned at all stages of the pregnancy and puerperium.

Suggested changes to the classification:

Instruction	Tabular list entries	Source	Major/Minor update	Suggested implementation date
	O43.1 Malformation of placenta	Australia (URC:0110)	Major	January 2003
Add subcategory Add inclusion terms	**O43.2 Morbidly adherent placenta** Placenta: .accreta .increta .percreta			
Add instructional note	Use additional code, if desired, to identify post partum haemorrhage (O72.0) or retained placenta without haemorrhage (O73.0)			
	O72 .0 Third stage haemorrhage			

Revise inclusion term	Haemorrhage associated with retained~~,~~ or trapped ~~or adherent~~ placenta			
Add instructional note	Use additional code, if desired, to identify morbidly adherent placenta (O43.2)			
	O73 .0 Retained placenta without haemorrhage			
Delete inclusion term	~~Placenta accreta without haemorrhage~~			
Add instructional note	Use additional code, if desired, to identify morbidly adherent placenta (O43.2)			

No.02 **Proposed by:** **United Kingdom**

Change to Volume: III Type of proposal: Major

Description (term/code): Hereditary Creutzfeldt-Jacob disease

Proposal for update:
There is a clear index trail in ICD-10 for Creutzfeldt-Jacob disease:

Creutzfeldt-Jacob disease or syndrome A81.0
- with dementia A81.0† F02.1*

There are no modifiers for hereditary. The code A81.0 is obviously the infectious form of the disease. However, there is also a hereditary form (see attached paper). Given the current prominence of this disease, and for epidemiological purposes, we feel it is important to be able to identify the hereditary form. Could the WHO Update Committee consider a different code for the hereditary form of this disease? Clinical information attached at the end of this proposal.

Suggested changes to the classification:

Instruction	Alphabetic index entries	Source	Major/Minor update	Suggested implementation date
	Creutzfeldt-Jacob disease or syndrome A81.0 - with dementia A81.0† F02.1* - hereditary ???	United Kingdon (URC:0180)	Major	January 2006

Clinical information:

Introduction

Creutzfeldt-Jakob disease (CJD) is a rare and fatal neurodegenerative disease of unknown cause. Patients are usually aged between 50 and 75 and typical clinical features include a rapidly progressive dementia associated, myoclonus and a characteristic electroencephalographic pattern. Neuropathological examination reveals cortical spongiform change, hence the term `spongiform encephalopathy'.

H.G. Creutzfeldt is credited with the first description of the disorder in 1920, although by current diagnostic criteria his case would be highly atypical. A year later another German neurologist, A. Jakob, described four cases, at least two of whom had clinical features suggestive of the entity we recognise as CJD.

Although CJD appears to occur as a predominantly sporadic disorder it can also occur as a dominantly inherited or infective condition. The latter mode of transmission was first elucidated during the study of kuru in the 1950's. This neurodegenerative condition occurs only in the people of the Fore region of Papua New Guinea and is thought to have resulted from the consumption of brains during endocannabalistic funeral rituals. The similarities between kuru and scrapie, the transmissible spongiform encephalopathy of sheep, prompted a veterinary neuropathologist, Hadlow, to suggest that transmission studies of kuru be performed. The success of those studies and the recognition that the neuropathological changes in kuru were similar to those of CJD, was followed by the transmission of CJD to the chimpanzee by intracerebral inoculation of brain tissue. In 1974 a case of iatrogenic CJD due to corneal transplantation occurred and subsequently contaminated neuro-surgical instruments, dural grafts, and brain depth electrodes have all been recognised as transmitting the disease. In 1985 the first case was reported in a recipient of contaminated human derived growth hormone and subsequently over 60 similar cases have arisen world-wide in addition to 4 cases associated with human derived gonadotrophin. The familial occurrence of CJD has been recognised for many years but was unexplained. The discovery of linkage to a region on the short arm of chromosome 20 has led recently to the elucidation of various dominantly inherited mutations.

Aetiology

The nature of the transmissible agent is the matter of some controversy. Previously considered a `slow virus' no viral agent has ever been convincingly demonstrated and no evidence of an immunological response seen. Additionally the infectious pathogen shows a remarkable resistance to treatments that would normally be expected to inactivate viruses. The viral hypothesis has been elegantly challenged by the prion (`proteinaceous infectious particle') theory which states that the infectious agent is derived from a protease-sensitive protein (designated PrPc) which is a constituent of the normal cell membrane. It is postulated that the normal protein undergoes a post-translational conformational change forming the insoluble pathogenic form of the prion protein (PrPsc). This in turn induces more of the normal PrPc to form PrPsc - hence a chain reaction is set in motion with the exponential production of the insoluble prion protein being formed. The initial abnormal prion protein needed to seed this process may occur spontaneously as a rare event (which would account for the low incidence of sporadic CJD), following inoculation (accounting for observed transmission phenomena) or when initiated by a genetic abnormality of the PrP gene. The mechanism by which PrPsc induces the pathological changes in CJD - spongiform change, gliosis, neuronal loss and (infrequently) plaques remains unclear. Although the prion hyopthesis

neatly explains many of the observed phenomena of transmissible spongiform encephalopathies (TSEs) it has one particular weakness. Scrapie is known to exhibit various `strains' characteristed by different incubation periods, clinical features and pathology when transmitted. This is much more in keeping with a virus-like agent and strain variation, independent of the host genome, is difficult to reconcile with the prion theory.

Epidemiology

The majority of cases are sporadic (85%), between 10-15% are familial and the remainder are iatrogenic.

CJD occurs worldwide with a roughly even incidence of between 0.5-1.0 cases per million per year. Higher rates (upto 100-fold) have reported in Slovakia and Libyan-born Israelis but this is explained by the high incidence of a certain mutation of the PrP gene in these groups. The geographical distribution of CJD in the United Kingdom over the past 25 years demonstrates no overall evidence of spatio-temporal aggregation of cases, despite the occurrence of local areas of relatively high incidence over short periods. There is no evidence of case to case transmission and spouses of sporadic cases do not have an increased incidence of the disease.

TSEs are known to affect various animal species including sheep, goats, mink, mule deer, cows and recently cats. Scrapie, a disorder of sheep and goats, has been known for over 300 years and is endemic in the British Isles. In 1938 experimental transfer of scrapie from one sheep to another by inoculation provided evidence of an infective aetiology. However there is no evidence of transmission of scrapie from sheep to man and there is no increased incidence of CJD in countries with scrapie compared to those without (e.g. UK and Australia).

No.03 Proposed by: MRG

Change to Volume: I III Type of proposal: Minor

Description (term/code): Subsequent myocardial infarction

Proposal for update:
The MRG has been discussing if I22 should be used for all subsequent infarctions, regardless of the time elapsed since the first one or only for subsequent infarctions occurring within four weeks from the first one. The MRG got information from WHO on what was intended for I22 and background information that the content of this code reflects findings from the WHO MONICA (MONItoring or Cardiovascular diseases) study.
The code I21 should only be used for an individual's first myocardial infarction. All subsequent acute infarctions should be coded to I22. This is illustrated below in three examples. The MRG recommends changes in Volumes 1 and 3 to clarify the instructions.

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Example 1: Ia Myocardial infarction I22.9
II Myocardial infarction 2 years ago I25.8

Example 2: Ia Reinfarction I22.9
Ib Myocardial infarction I21.9

Example 3: Ia Generalised arteriosclerosis I70.9
II Old and new myocardial infarction I25.2 I22.9

Reference for MRG Problem Set 5, Question 28: See Question 1999-10-04_03 (http://www.pubcare.uu.se/nordwho/verksam/mortforum/mortindex.htm) for related discussion in the Mortality Forum.

Suggested changes to the classification:

Instruction	Tabular list entries	Source	Major/Minor update	Suggested implementation date
	I21 Acute myocardial infarction	MRG	Minor	January 2005

Replace deleted text with underlined text	***Includes:*** myocardial infarction specified as acute or with a stated duration of 4 weeks (28 completed days) or less from onset ***Excludes:*** certain current complications following acute myocardial infarction (I23.-) myocardial infarction: • old (I25.2) • specified as chronic or with a stated duration of ~~more than~~ 4 weeks (~~more than 28~~ 29 completed days) or greater from onset (I25.8) • subsequent or recurrent, irrespective of the time elapsed since the first infarction (I22.-) postmyocardial infarction syndrome (I24.1)	(URC:0198)		
Replace deleted text with underlined text	**I22 Subsequent myocardial infarction** ***Includes:*** recurrent myocardial infarction, irrespective of the time elapsed since the first infarction ***Excludes:*** specified as chronic or with a stated duration of ~~more than~~ 4 weeks (~~more than 28~~ 29 completed days) or greater from onset (I25.8)	MRG (URC:0198)	Minor	January 2005

Instruction	Alphabetic index entries	Source	Major/Minor update	Suggested implementation date
Modify subterms	**Infarct, infarction** (of) - myocardium, myocardial (acute or with a stated duration of 4 weeks (28 completed days) or less) I21.9 - - subsequent (recurrent), irrespective of the time elapsed since the first infarction I22.9 - - - anterior (wall), irrespective of the time elapsed since the first infarction I22.0 - - - diaphragmatic (wall), irrespective of the time elapsed since the first infarction I22.1 - - - inferior (wall), irrespective of the time elapsed since the first infarction I22.1 - - - specified ~~NEC~~, irrespective of the time elapsed since the first infarction I22.8	MRG (URC:0198)	Minor	January 2005

No.04 Proposed by: United Kingdom

Change to Volume: I & III Type of proposal: Major

Description (term/code): Eisenmenger's syndrome

Proposal for update:

Background
This proposal for change was originally tabled in the URC papers for 2000 and the background to the change is reproduced below:

The UK believes that Eisenmenger's syndrome is an acquired condition, secondary to a cardiac septal defect, which leads to irreversible pulmonary vascular disease. Conversely, Eisenmenger's disease is a congenital condition classified to Q21.8 *Other congenital conditions of cardiac septa*. The ICD-10 index entry is the same for both conditions. We recommend that both the index and tabular list entries be amended. Our recommendation follows clinical advice obtained from Mr. Rodney Franklin who has been involved since the 1980s with the Pan-European Cardiology Coding System. This system forms a comprehensive list of paediatric congenital heart problems and procedures.

Suggested changes to the classification:

Instruction	Tabular list entries	Source	Major/Minor update	Suggested implementation date
Revise inclusion term	**Q21.8 Other congenital malformations of cardiac septa** Eisenmenger's defect or disease	UK (URC:0103)	Major	January 2003

Instruction	Alphabetic index entries	Source	Major/Minor update	Suggested implementation date
Delete term and	**Eisenmenger's ~~defect, complex or syndrome Q21.8~~**	UK	Major	January 2003

code Add subterms and codes	<u>- complex or syndrome I27.8</u> <u>- defect Q21.8</u>	(URC:0103)		

No.05 **Proposed by:** **MRG**

Change to Volume: I III Type of proposal: Major

Description (term/code): bacterial hepatitis

Proposal for update:
A question has arisen on how to code bacterial hepatitis. The MRG recommends a new code, changes in excludes note for volume 1, and changes in Volume 3.

Reference for MRG Problem Set 5, Question 69: See Question 1999-01-25 02 (http://www.pubcare.uu.se/nordwho/verksam/mortforum/mortindex.htm) for related Mortality Forum discussions.

Suggested changes to the classification:

Instruction	Tabular list entries	Source	Major/Minor update	Suggested implementation date
Add subcategory Add inclusion term	**K75 Other inflammatory liver diseases** *Excludes*: chronic hepatitis NEC (K73.-) hepatitis: • acute or subacute (K72.0) • viral (B15-B19) toxic liver disease (K71.-) **K75.3 Granulomatous hepatitis, not elsewhere classified** **K75.5 Bacterial hepatitis** Bacterial hepatitis (acute)(subacute)(chronic) **K75.8 Other specified inflammatory liver diseases**	MRG (URC: 0166)	Major	January 2006

Instruction	Alphabetic index entries	Source	Major/Minor update	Suggested implementation date
	Hepatitis K75.9 - Australia-antigen (positive) (*see also* Hepatitis, viral, type B) B16.9	MRG (URC:0166)	Major	January 2006

Add subterm and code	- bacterial (acute) (chronic) (subacute) NEC K75.5 - catarrhal (acute) B15.9			

No.06 Proposed by: MRG

Change to Volume: I III Type of proposal: Major

Description (term/code): Hypoxic ischaemic encephalopathy

Proposal for update:

Hypoxic ischaemic encephalopathy of newborn is a common statement on certificates for newborns for which a separate code would be useful. The MRG recommends creating a new code and modifying the index to reflect the new code.

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Reference for MRG Problem Set 5, Question 67:
See Question 1999-09-20 Q1 at (http://www.pubcare.uu.se/nordwho/verksam/mortforum/mortindex.htm) for related Mortality Forum discussions.

Suggested changes to the classification:

Instruction	Tabular list entries	Source	Major/Minor update	Suggested implementation date
Add subcategory	**P91 Other disturbances of cerebral status of newborn** **P91.0 Neonatal cerebral ischaemia** **P91.1 Acquired periventricular cysts of newborn** **P91.2 Neonatal cerebral leukomalacia** **P91.3 Neonatal cerebral irritability** **P91.4 Neonatal cerebral depression** **P91.5 Neonatal coma** **P91.6 Hypoxic ischaemic encephalopathy of newborn** **P91.8 Other specified disturbances of cerebral status of newborn** **P91.9 Disturbance of cerebral status of newborn, unspecified**	MRG (URC:0172)	Major	January 2006

Instruction	Alphabetic index entries	Source	Major/Minor update	Suggested implementation date

Add subterm and code	**Encephalopathy (acute)** G93.4 - hypoxic – *see* Damage, brain, anoxic - - ischaemic of newborn P91.6 - in (due to)	MRG (URC:0172)	Major	January 2006