

**Second Edition** 

ICD-10-AM·



Welcome to the *CDI Pocket Guide*° *ICD-10-AM*, the second Australian edition of the best-selling CDI Pocket Guide° that was originally created for hospitals in the United States.

Dr. Richard Pinson and Cynthia Tang created the original **CDI Pocket Guide®** in **2008** because they wanted to provide this information to all hospitals, large or small. At the time, the only way to receive training in this field was with large-scale, expensive consulting projects. They thought they could bring this pocketful of information with the clinical criteria to identify important diagnoses to any individual who was interested in working in the CDI field.

The CDI Pocket Guide® quickly became a best-selling book and an industry standard, and many consider it to be their CDI "bible". Since that time, they have created other products, including the popular CDI Pocket Guide® Unbound (online) edition.

As the field of CDI has emerged in Australia, Jenny Fitzpatrick and Sam Heynemann of the Uplift Group recognised the need for a resource for the Australian context. When they found the original CDI Pocket Guide\*, they reached out to Cynthia and Richard, and together these four seasoned experts created the CDI Pocket Guide\* for ICD-10-AM and AR-DRGs that you now hold in your hands.

This guide retains all the clinical content that made the original *CDI Pocket Guide*\* a success, but it has been revised for the ICD-10-AM classification and Australian-Refined DRGs (AR-DRGs).

We trust that you, like thousands before you, will find this a useful tool in addressing the daily complexities of coding and clinical documentation. The ultimate goal is not just more accurate coding and reimbursement, but improved quality and outcomes for both clinicians and hospitals.

To your success,

Jenny Fitzpatrick, Sam Heynemann, Richard Pinson, MD, and Cynthia Tang.

# INTRODUCTION TO THE CDI POCKET GUIDE®, ICD-10-AM

We trust that you will find this a useful tool in addressing the daily complexities of coding and clinical documentation.

Each of the five sections of this guide is written with a specific purpose in mind:

**Guidelines** is a shortcut to the most important guidelines and coding rules for DRG assignment and other important topics. Refer to these guidelines frequently.

**Key References** provides detailed clinical definitions and criteria, treatment, coding and documentation challenges, and references for the most important and frequently encountered conditions.

**Complex Diagnoses** lists and summarises the most common and important additional diagnoses with DCL that affect DRG assignment in the different AR-DRG versions.

**DRG Tips** includes alternative DRG assignment for select DRGs which in our experience have a high likelihood of another principal diagnosis, additional complex diagnosis or procedure.

**AR-DRG Table** is a complete list of the v10.0 AR-DRGs and their cost weights for quick reference.

All references and citations for The International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification (ICD-10-AM), Australian Classification of Health Interventions (ACHI) and Australian Coding Standards (ACS) are sourced from the Independent Hospital Pricing Authority (IHPA) 2022, Twelfth Edition publications.

References to the ACS have been shortened for simplicity to: ACS [number][Title]. References to two national coding advice sources are cited as follows: Australian Consortium for Classification Development as ACCD [date]:[Title] and Australian Classification Exchange as ACE [date]:[Title].

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# **DEFINITION**

A Transient Ischaemic Attack (TIA) is a transient episode of focal neurological dysfunction caused by cerebral ischaemia (reduced blood flow). Typical neurological deficits include focal weakness, impaired speech, difficulty with balance or walking.

# DIAGNOSTIC CRITERIA

TIA is defined by both:

- Transient focal neurological deficit lasting < 24 hours, and
- · No acute infarction or haemorrhage on imaging

Duration is counted from onset, not presentation. Patients often present having already had symptoms for several hours or more.

Persistence of a focal neurological deficit > 24 hours from onset is a stroke (CVA), not TIA, even with negative imaging.

While the duration of symptoms required to establish a diagnosis of TIA officially remains < 24 hours, it is a subject of debate amongst neurologists with many experts suggesting it should only be one hour.

# Causes of TIA include:

- Cerebral/pre-cerebral stenosis: Any degree of stenosis may cause a TIA. "Noncritical stenosis" indicates that medical therapy is preferred to surgery; it does not exclude stenosis as the cause.
- Transient cerebral embolism due to platelet aggregates is often caused by atrial fibrillation, abnormal heart valves, or atrial septal defect (ASD).
- Vertebrobasilar syndrome is considered nothing more than a TIA symptom, but documentation of stenosis, occlusion, thrombosis, embolism of any vertebrobasilar arteries is a serious cause.

Common sources of transient cerebral embolism include:

- Atrial fibrillation—especially if PT/INR is subtherapeutic
- Valvular heart disease (aortic or mitral valves)

- ASD with "paradoxical embolus" of small clots from the right (venous) side of the heart across the ASD to the left heart and from there up to the brain
- Mural (ventricular wall) thrombus—especially following MI
- Any degree of stenosis/narrowing of a carotid, vertebral, or cerebral artery

Diagnostic testing often includes trans-oesophageal echocardiogram (TOE), carotid or transcranial Doppler, CT, MRI, and MRA—all looking for sources of emboli, stenosis, occlusion, or thrombosis.

# TREATMENT

Treatment includes antiplatelet therapy, such as aspirin, dipyridamole or clopidogrel, to prevent recurrent thrombosis or embolism, even when no significant abnormality is identified. Less often, an anticoagulant like warfarin may be prescribed.

# **CODING AND DOCUMENTATION CHALLENGES**

TIA is a symptom of a significant underlying cerebrovascular process. It is the underlying condition that really matters and is treated, not the symptom. Always seek clarification of the suspected or confirmed underlying cause of TIA symptoms. Even if nothing is found on evaluation to explain the TIA, the most likely cause of TIA is unexplained "transient cerebral embolism."

Identification of CVA when criteria are actually met represents a query opportunity, especially when imaging is negative but symptoms persist > 24 hrs from onset. For *DRG Tips*, see B69 *TIA and Precerebral Occlusion*.

# References:

- National Stroke Association Guidelines for the Management of Transient Ischemic Attacks. Ann Neurol 2006; 60: 301–313
  - Diagnosis and Management of Transient Ischemic Attack, Coutts SB

 UpToDate.com: Initial evaluation and management of transient ischemic attack and minor ischemic stroke.

# DRG H07 **Open Cholecystectomy**(A) Major, (B) Intermediate, (C) Minor

H07A	5.7499	H02A	8.2447
H07B	3.5700	H02B	4.8221
H07C	2.5206	H02C	2.1043

## PRINCIPAL DIAGNOSIS

**LOOK FOR** evidence of pancreatitis. Gallstone obstruction of the pancreatic or common bile duct (gallstone pancreatitis). See *Pancreatitis*.

### ADDITIONAL DIAGNOSIS

<u>LOOK FOR</u> division of adhesions with evidence of a history of abdominal surgery to query for postprocedural adhesions. Adhesions confirmed due to previous surgery are assigned K91.89 postprocedural disorders of digestive system + K66.0 Peritoneal adhesions + appropriate external cause codes. See *Adhesions* and *Procedural Complications*.

### **PROCEDURE**

**LOOK FOR** exploration of common bile duct or choledochotomy for DRG H02 (Major biliary tract interventions).

**LOOKFOR** evidence of adhesiolysis. Synonymous terms include 'divided', 'take down', 'released', 'dissection/dissected', 'stripped down', 'separated' or 'freed'. See *Adhesions*.

MBS item number 30724, 30393, 30378 (most frequent) are commonly documented by the surgeon without qualifying terms describing the procedure within the body of the operation report.