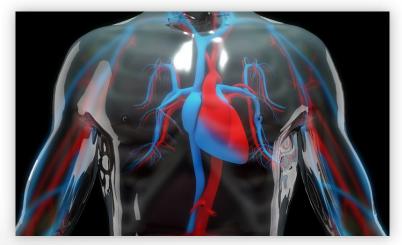
National Heart Foundation of Australia & Cardiac Society of Australia and New Zealand

Australian Clinical Guidelines for the Management of Acute Coronary Syndromes 2016 [1]







Prevalence

- Chest pain and acute coronary syndrome (ACS) symptoms are common presenting complaints in emergency departments (EDs).
- There were 68,200 ACS events recorded in 2012 [1].
- >500,000 patients present with chest pain in Australia each year, but ≥80% of all patients investigated for ACS do not have a diagnosis confirmed [1,2].
- There are significant health burdens and health sector costs associated with ACS diagnosis and assessment.







^{1.} Australian Institute of Health and Welfare. 2014. Cardiovascular disease, diabetes and chronic kidney disease—Australian facts: Prevalence and incidence. 2. Cullen L, et al. Med J Aust 2015;202 (8):427–32.

Background

- Aim is to provide a clinical guideline to assist the management of patients presenting with chest pain, due to suspected or confirmed ACS.
- Intended to replace the NHFA/CSANZ ACS guidelines of 2006 [1], addenda 2007 [2] and 2011 [3].
- These guidelines should be read in conjunction with:
 - ACS Clinical Care Standards developed by the Australian Commission for Safety and Quality in Health Care (ACSQHC) [4].
 - Australian Acute Coronary Syndromes Capability Framework developed by the Heart Foundation [5].





Working Group

- An ACS Guideline Development Working Group was facilitated by the National Heart Foundation of Australia (NHFA) in partnership with Cardiac Society of Australia and New Zealand (CSANZ).
- The Working Group included a broad mix of health professionals, including a general practitioner, general physician, cardiac surgeon, pathologist, ambulance representative, cardiologists, emergency physicians, exercise physiologists, cardiac nurses and a consumer representative.







The process for developing the guidelines

- Literature review:
 - informed by stakeholder consultation, the working group developed clinical questions on which the literature review was based
 - conducted by an external literature reviewer, who was appointed though an open tender process (KP Health)
 - included published studies from 2010 to 2015.





The process for developing the guidelines

- Governance
 - Processes in place to ensure transparency, minimise bias, manage conflict of interest (COI) and limit other influences during development.
- Recommendations developed using
 - NHMRC (level of evidence)
 - GRADE methodology (strong or weak).





The process for developing the guidelines

- Public consultation period of 30 days in April 2016 on the final draft.
- NHFA and CSANZ clinical committee and National Board approvals followed.
- Endorsed by key stakeholder organisations.
- Publication in peer review journals August 2016.





What is new from previous guidelines?

- Recommendations are graded on the strength of the evidence and the expected value of the intervention.
- Recommendations focus on the interventions and therapies most likely associated with improved outcomes.
- Use of **practice points** to highlight aspects of care that are supported by limited evidence or modest benefits.
- Focus on pathways for the assessment of patients with suspected ACS.





What is new from previous guidelines?

- Guidance on:
 - troponin testing integrated into chest pain assessment pathways
 - patient groups not requiring further testing
 - duration of cardiac monitoring
 - prompt transfer of patients receiving fibrinolysis in STEMI
 - provision and timing of early invasive management in NSTEACS
 - reduced indication for glycoprotein llb/llla inhibition
 - combination antiplatelet and anti-thrombin therapy
 - duration of P2Y₁₂ inhibition
 - reduced indication for beta-blocker therapy.





Recommendations







Initial assessment of chest pain

Rec	ommendation	Grade
1.	It is recommended that a patient with acute chest pain or other symptoms suggestive of an ACS receives a 12-lead ECG and this ECG is assessed for signs of myocardial ischaemia by an ECG-experienced clinician within 10 minutes of first acute clinical contact.	Strong IIIC
2.	A patient presenting with acute chest pain or other symptoms suggestive of ACS should receive care guided by an evidence-based Suspected ACS Assessment Protocol (Suspected ACS-AP) that includes formal risk stratification.	Strong IA
3.	Using serial sampling, cardiac-specific troponin levels should be measured at hospital presentation and at clearly defined periods after presentation using a validated Suspected ACS-AP in patients with symptoms of possible ACS.	Strong IA





Oxygen supplementation

- Routine use of oxygen therapy among patients with a blood oxygen saturation (SaO₂) level > 93% is not recommended, but its use when the SaO₂ is below this level is advocated, despite the absence of clinical data [1,2].
- Target SaO₂ level for patients with chronic obstructive pulmonary disease is 88–92%.

Initial aspirin therapy

- In all patients with possible ACS and without contraindications, aspirin (300 mg orally) should be given as soon as possible after presentation.
- Additional antiplatelet and anticoagulation therapy, or other therapies such as beta blockers, should not be given to patients without a confirmed or probable diagnosis of ACS.





Initial assessment of chest pain

Rec	commendation	Grade
4.	Non-invasive objective testing is recommended in intermediate-risk patients, as defined by a validated Suspected ACS-AP, with normal serial troponin and ECG testing and who remain symptom-free.	Weak IA
5.	Patients in whom no further objective testing for coronary artery disease (CAD) is recommended are those at low risk, as defined by a validated Suspected ACS-AP: age <40 years, symptoms atypical for angina, in the absence of known CAD, with normal troponin and ECG testing, and who remain symptom-free.	Weak III-3C
6.	The routine use of validated risk stratification tools for ischaemic and bleeding events (e.g. GRACE score for ischaemic risk or CRUSADE score for bleeding risk) may assist in patient-centric clinical decision-making in regards to ACS care.	Weak IIIB





Assessment protocols for suspected ACS

IMPORTANT NOTICE: Management protocols never replace clinical judgement. The care outlined in this protocol must be altered if it is not clinically appropriate for the individual patient.

Troponin and ECG testing on presentation (0h) High risk features for possible cardiac cause of chest pain (including ACS and other cardiac diagnoses) · Ongoing or repetitive chest pain despite initial ED treatment · Elevated level of cardiac troponin* • Persistent or dynamic electrocardiographic changes of ST-segment depression ≥0.5 mm or new T-wave inversion ≥2 mm in more than two contiguous leads Transient ST-segment elevation (≥0.5 mm) in more than two contiguous leads • Haemodynamic compromise — systolic blood pressure < 90 mmHg, cool peripheries, diaphoresis, Killip Class > I, and/ or new-onset mitral regurgitation · Sustained ventricular tachycardia • Known left ventricular systolic dysfunction (left ventricular ejection fraction <40%) · Prior AMI, percutaneous coronary intervention or prior coronary artery bypass surgery within 6 months No to all High risk for cardiac condition including ACS Refer for admission and further inpatient investigation Repeat troponin and ECG testing at 6-8h after presentation Repeat troponin and ECG at 6-8h Further symptoms, new ECG abnormalities, elevated troponin level Very low risk for ACS No further objective Age <40yrs testing recommended Normal ECG Normal serial troponin values No ongoing symptoms Further Low risk for AMI objective testing Symptoms atypical for Assess for risk of CAD and need for objective testing recommended angina ACS-AP. *Elevated troponin defined as >99th percentile of a normal reference population.

- Point of care assays
- Sensitive lab-based assays
- Highly sensitive lab-based assays





Differential diagnosis of causes of chest pain

Ischaemic cardiovascular causes	 ACS (e.g. acute myocardial infarction, unstable angina) Stable angina Severe aortic stenosis Tachyarrhythmia (atrial or ventricular)
Non-ischaemic cardiovascular causes of chest pain	 Aortic dissection (tear between the layers of the wall of the aorta) and expanding aortic aneurysm Pulmonary embolism Pericarditis and myocarditis Gastrointestinal causes (e.g. gastro-oesophageal reflux, oesophageal spasm, peptic ulcer, pancreatitis, biliary disease)
Non-cardiovascular causes	 Musculoskeletal causes (e.g. costochondritis, cervical radiculopathy, fibrositis) Pulmonary (e.g. pneumonia, pleuritis, pneumothorax) Other aetiologies (e.g. sickle cell crisis, herpes zoster)





Causes of troponin elevation*

- Cardiac contusion, or other trauma including surgery, ablation, pacing, frequent defibrillator shocks
- Congestive heart failure acute and chronic
- Coronary vasculitis, e.g. SLE, Kawasaki syndrome
- Aortic dissection
- Aortic valve disease
- Hypertrophic cardiomyopathy
- Tachy- or bradyarrhythmias, or heart block
- Stress cardiomyopathy (Takotsubo cardiomyopathy)
- Rhabdomyolysis with cardiac injury
- **Pulmonary embolism**, severe pulmonary hypertension
- Renal failure
- Acute neurological disease, including stroke or subarachnoid haemorrhage
- Infiltrative diseases, e.g. amyloidosis, haemochromatosis, sarcoidosis, and scleroderma
- Inflammatory diseases, e.g. myocarditis or myocardial extension of endo-/pericarditis
- Drug toxicity or toxins e.g. anthracyclines, CO poisoning
- Critically ill patients, especially with respiratory failure or sepsis
- Hypoxia
- Burns, especially if affecting > 30% of body surface area
- Extreme exertion
- False positives: Cross reacting heterophile antibodies





^{*}Life-threatening, non-coronary conditions highlighted in bold

Markers of increased risk with confirmed ACS

Risk classification	Clinical characteristic
Very high	 Haemodynamic instability, heart failure, cardiogenic shock or mechanical complications of MI Life-threatening arrhythmias or cardiac arrest Recurrent or ongoing ischaemia i.e. chest pain refractory to medical treatment, or recurrent dynamic ST-segment and/or T-wave changes, particularly with intermittent ST-segment elevation, de Winter T-wave changes, or Wellens'
	syndrome, or widespread ST-segment elevation in two coronary territories
High	Rise and/or fall in troponin level consistent with MI
	Dynamic ST-segment and/or T-wave changes with or without symptoms
	GRACE Score >140
Intermediate	Diabetes mellitus
	Renal insufficiency (GFR<60mL/min/1.73m²)
	• LVEF <40%
	Prior revascularisation: PCI or CABG
	GRACE score >109 and <140

GRACE, Global Registry of Acute Coronary Events; GFR, glomerular filtration rate; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; MI, myocardial infarction





Acute reperfusion and invasive management

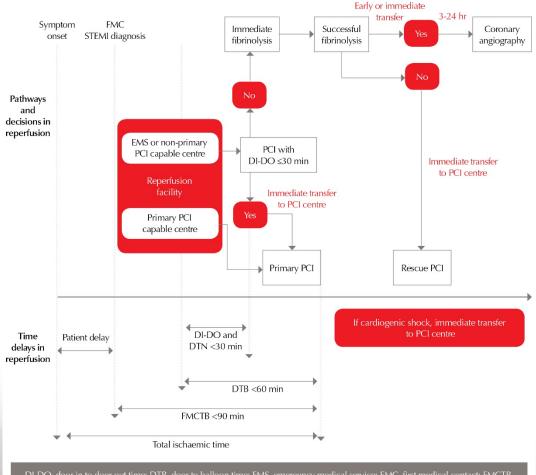
Rec	ommendation	Grade
1.	For patients with ST elevation myocardial infarction (STEMI) presenting within 12 hours of symptom onset, and in the absence of comorbidities that influence the individual's overall survival, emergency reperfusion therapy with either primary percutaneous coronary intervention (PCI) or fibrinolytic therapy is recommended.	Strong IA
2.	Primary PCI is preferred for reperfusion therapy in patients with STEMI if it can be performed within 90 minutes of first medical contact; otherwise fibrinolytic therapy is preferred for those without contra-indications.	Strong IA
3.	Among patients treated with fibrinolytic therapy who are not in a PCI-capable hospital, early or immediate transfer to a PCI-capable hospital for angiography, and PCI if indicated, within 24 hours is recommended.	Weak IIA
4.	Among patients treated with fibrinolytic therapy, for those with ≤50% ST recovery at 60–90 minutes, and/or with haemodynamic instability, immediate transfer for angiography with a view to rescue angioplasty is recommended.	Strong IB





Decision-making and timing considerations in reperfusion for STEMI

Decision-making and organisation of reperfusion strategies within 12 hours of first medical contact (FMC) with ideal time interval for interventions



Adapted from ESC. Eur Heart J 2012;33:2569-619.

DI-DO, door in to door out time; DTB, door to balloon time; EMS, emergency medical service; FMC, first medical contact to balloon time; PCI, percutaneous coronary intervention; STEMI, ST elevation myocardial infarction





Invasive management in NSTEACS

Rec	ommendation	Grade
1.	Among high- and very high-risk patients with non ST elevation acute coronary syndromes (NSTEACS) (except Type 2 MI), angiography with coronary revascularisation (PCI or coronary artery bypass grafts) where appropriate is recommended.	Strong IA
2.	Patients with NSTEACS who have no recurrent symptoms and no risk criteria are considered at low risk of ischaemic events, and can be managed with a selective invasive strategy guided by provocative testing for inducible ischaemia.	Strong IA
3.	Among patients with NSTEACS with very high-risk criteria (ongoing ischaemia, haemodynamic compromise, arrhythmias, mechanical complications of MI, acute heart failure, recurrent dynamic or widespread ST-segment and/or T-wave changes on ECG), an immediate invasive strategy is recommended (within 2 hours of admission).	Strong IIC
4.	In the absence of very high-risk criteria, for patients with NSTEACS with high-risk criteria (GRACE score >140, dynamic ST-segment and/or T-wave changes on ECG, or rise and/or fall in troponin compatible with MI) an early invasive strategy is recommended (within 24 hours of admission).	Weak IC
5.	In the absence of high-risk criteria, for patients with NSTEACS with intermediate-risk criteria (such as recurrent symptoms or substantial inducible ischaemia on provocative testing), an invasive strategy is recommended (within 72 hours of admission).	Weak IIC





Pharmacology for ACS

Rec	ommendation	Grade
1.	Aspirin 300 mg orally initially (dissolved or chewed) followed by 100–150 mg/day is recommended for all patients with ACS in the absence of hypersensitivity.	Strong IA
2.	Among patients with confirmed ACS at intermediate to very high-risk of recurrent ischaemic events, use of a P2Y ₁₂ inhibitor (ticagrelor; or prasugrel; or clopidogrel) is recommended in addition to aspirin. (ticagrelor or prasugrel preferred).	Strong IA
3.	Intravenous glycoprotein IIb/IIIa inhibition in combination with heparin is recommended at the time of PCI among patients with high-risk clinical and angiographic characteristics, or for treating thrombotic complications among patients with ACS.	Strong IB
4.	Either unfractionated heparin or enoxaparin is recommended in patients with ACS at intermediate to high risk of ischaemic events.	Strong IA
5.	Bivalirudin (0.75 mg/kg IV with 1.75 mg/kg/hr infusion) may be considered as an alternative to glycoprotein IIb/IIIa inhibition and heparin among patients with ACS undergoing PCI with clinical features associated with an increased risk of bleeding events.	Weak IIB





- Choosing between P2Y₁₂ inhibitors
 - Given their superior efficacy, ticagrelor and prasugrel are the preferred first-line P2Y₁₂ inhibitors.
- Timing of P2Y₁₂ initiation: Based on limited data
 - Ticagrelor or clopidogrel should be commenced soon after diagnosis but due consideration should be given to ischaemic and bleeding risk, the likelihood of need for CABG and the delay to angiography.
 - Prasugrel should be commenced immediately following diagnosis among patients undergoing primary PCI for STEMI, or after the coronary anatomy is known among those undergoing urgent PCI. Initiation of prasugrel prior to coronary angiography outside the context of primary PCI is not recommended.





- In patients with a strong long-term indication for anticoagulation (i.e. mechanical heart valves, atrial fibrillation (AF) with CHA2DS2VASC score ≥2):
 - anticoagulant should be continued at reduced dose
 - and clopidogrel used, rather than ticagrelor or prasugrel.



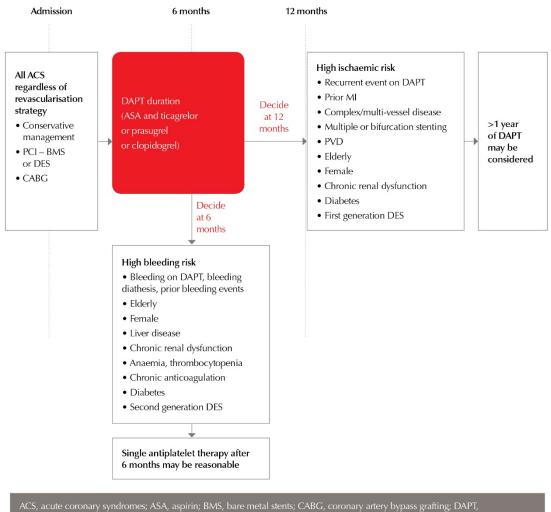


- Duration of triple therapy should be determined by bleeding risk:
 - HAS-BLED score <3, consider 3-6 months of triple therapy and then aspirin or clopidogrel with oral anticoagulation (OAC) up to 12 months
 - HAS-BLED score ≥3, consider 1 month of triple therapy and then aspirin or clopidogrel with OAC up to 12 months
 - Patients with AF at low thromboembolic risk (CHA2DS2VASC score = 1) should be managed with dual antiplatelet therapy for 12 months, beyond which OAC may be considered
 - routine concurrent use of a proton pump inhibitor should be considered for the duration of triple therapy.





Considerations for dual antiplatelet therapy (DAPT) in patients with ACS







Discharge management

Rec	ommendation	Grade
1.	Aspirin (100–150 mg/day) should be continued indefinitely unless it is not tolerated or an indication for anticoagulation becomes apparent.	Strong IA
2.	Clopidogrel should be prescribed if aspirin is contraindicated or not tolerated.	Strong IA
3.	Dual-antiplatelet therapy with aspirin and a $P2Y_{12}$ inhibitor (clopidogrel or ticagrelor) should be prescribed for up to 12 months in patients with ACS, regardless of whether coronary revascularisation was performed. The use of prasugrel for up to 12 months should be confined to patients receiving PCI.	Strong IA
4.	Consider continuation of dual-antiplatelet therapy beyond 12 months if ischaemic risks outweigh the bleeding risk of P2Y ₁₂ inhibitor therapy; conversely consider discontinuation if bleeding risk outweighs ischaemic risks.	Weak IIC





Discharge management

Rec	ommendation	Grade
5.	Initiate and continue indefinitely, the highest tolerated dose of HMG-CoA reductase inhibitors (statins) for a patient following hospitalisation with ACS unless contraindicated or there is a history of intolerance.	Strong IA
6.	Initiate treatment with vasodilatory beta-blockers in patients with reduced left ventricular (LV) systolic function (LV ejection fraction [EF] ≤40%) unless contraindicated.	Strong IIA
7.	Initiate and continue angiotensin converting enzyme (ACE) inhibitors (or angiotensin receptor blockers) in patients with evidence of heart failure, LV systolic dysfunction, diabetes, anterior myocardial infarction or co-existent hypertension.	Strong IA
8.	Attendance at cardiac rehabilitation or undertaking a structured secondary prevention service is recommended for all patients hospitalised with ACS.	Strong IA





Individualisation of cardiac rehabilitation/secondary prevention service referral:

- A wide variety of prevention programs improve health outcomes in patients with coronary disease.
- Following discharge from hospital, patients with ACS and their companion(s) should be referred to an individualised preventive intervention according to personal preference, values and the available resources.
- Services can be hospital-based, in primary care, the local community or in the home.





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Endorsement

- Australasian College for Emergency Medicine
- Australian Cardiovascular Health and Rehabilitation Association
- Royal College of Pathologists of Australasia
- Internal Medicine Society of Australia and New Zealand
- The Australasian Cardiovascular Nursing College
- Council of Remote Area Nurses of Australia
- Australian and New Zealand Society of Cardiac and Thoracic Surgeons
- Australian Commission on Safety and Quality in Health Care





Publications

Executive summary in MJA



Full document in HLC

1443-950s/04/458-36 http://dx.dei.org/10.3094-556-2094-06-299	2000 1000 1000 1000 1000 1000 1000 1000
National Heart Foundation Cardiac Society of Austra New Zealand: Australian Guidelines for the Manag	lia and Clinical
Coronary Syndromes 201	6
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Resources on NHFA website

- PowerPoint presentations for health professionals
 - Short and longer versions
- COI register and governance document (for the working group during development of the guidelines)
- Treatment algorithm for STEMI (print version)
- Assessment protocols for suspected ACS (print versions)
 - Using point of care, sensitive and highly sensitive assays
- Resources available at: http://heartfoundation.org.au/for-professionals/clinical-information/acute-coronary-syndromes.





Questions







