2nd Ed CLINICAL PRACTICE GUIDELINES
on
STABLE CAD,
2018
This CPG provides guidance on:

- diagnosis of CAD in individuals presenting with stable chest symptoms.
- risk stratification of individuals who are diagnosed with CAD. This helps to determine the need for revascularization.
- optimal medical therapy in all individuals with CAD.
- revascularization strategies
Acute Coronary Syndrome

Versus

Stable CAD
<table>
<thead>
<tr>
<th>Acute Coronary Syndrome</th>
<th>STABLE CAD</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Unstable Angina</td>
<td>Includes individuals:</td>
</tr>
<tr>
<td>➢ NonSTEMI</td>
<td>➢ with stable chest pain or other symptoms (e.g. dyspnea) which are known or suspected to be due to CAD.</td>
</tr>
<tr>
<td>➢ STEMI</td>
<td>➢ who had a previous episode of ACS but who are now stable and need regular follow up and monitoring.</td>
</tr>
</tbody>
</table>

|                      | ➢ post revascularization (by CABG or PCI) who are at present asymptomatic or have stable symptoms due to CAD. |
|                      | ➢ who are asymptomatic but are suspected or known to have CAD on non-invasive testing. This may occur in the absence or presence of ischemia and/or Left Ventricular dysfunction. |
ACS Versus Stable CAD

DIFFERENCES

➢ Pathophysiology

➢ Natural history
Pathophysiology

➢ **Angina due to Stable CAD** is due to myocardial ischemia resulting from a transient and reversible imbalance (mismatch) between myocardial oxygen demand and supply.

➢ In contrast, in an **ACS**, the thrombotic component of the ruptured plaque dominates the overall pathophysiological process and clinical picture.
Adventitia  
Media  
Intima  
Lumen  

Asymptomatic atherosclerotic plaque  
Stable fixed atherosclerotic plaque  
Stable angina  
Plaque disruption and platelet aggregation  
Thrombus  
Unstable plaque  
Unstable angina  
Non-ST-segment elevation MI  
ST-segment elevation MI  
Acute coronary syndromes
Pathophysiology

➢ The **natural history** of Stable CAD is marked by episodes of sudden deterioration due to plaque fissuring, ulceration or erosion with superimposed thrombosis resulting in ACS.
Phase 1 (I-III) and Phase 2 (IV-Va) lead to Acute Syndromes: Myocardial infarction, Unstable angina, Ischemic sudden death. Phase 3 (VI) is No Symptoms. Phase 4 (Vb-Vc) leads to Acute silent occlusive process. Phase 5 (Vb-Vc) is associated with Angina pectoris.
Angina may occur in the presence of:

- **Atherosclerotic obstructive CAD** – (coronary lesions >50% luminal narrowing)

- **Non-obstructive CAD** (≥20% and <50% luminal narrowing). The prognosis of these patients is not benign. It is worse if myocardial ischemia is documented.

- **Normal coronary arteries** (Cardiac Syndrome X) – (<20% luminal narrowing)
ACS Versus Stable CAD

Differences

➢ Prognosis
PROGNOSIS AFTER ACS
Cardiovascular risk in post-myocardial infarction patients: nationwide real world data
(SWEDISH National Registry)

Composite endpoint risk was 18.3% during the first 365 days post-index MI.

Kaplan–Meier estimate of the risk of the combined endpoint (MI, ischaemic stroke, or cardiovascular death) during the first 365 days after the index MI, stratified by age.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Hazard Ratio (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aged 60–69 vs. &lt;60 years</td>
<td>1.37 (1.30–1.45)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Aged 70–79 vs. &lt;60 years</td>
<td>2.13 (2.03–2.24)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Aged ≥80 vs. &lt;60 years</td>
<td>3.96 (3.78–4.15)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

N= 108 315 patients between 1.7.2006 and 30.6.2011

PROGNOSIS IN STABLE CAD
CORONOR (Suivi d’une cohorte de patients COROnariens stables en region NORd-Pas-de-Calais) Registry (inclusion period 2010 to 2011)

Registry of N=4094, 1 year Post ACS,FU: 5 years

Medications:
antiplatelet drugs 96.4%,
statins 92.2%,
ACEi/ARB 81.8%
beta-blockers 79.2%

- In stable CAD outpatients, incident MI (after the 1st year) occurs at a stable rate of 0.8% annually.

- In conclusion, the mortality rate of patients with stable CAD in modern clinical practice is similar to that of the general population and is mostly due to noncardiovascular causes.

In patients with Stable CAD on Optimal Medical therapy, the incidence of MI (after the 1st year), occurs at a rate of 0.8% per year.

Predictors of MI are:
- CV risk factors such as:
  - active smoking,
  - poorly controlled diabetes and/or lipids,
- persistent angina and/or
- multivessel disease.

The most important predictors of adverse CV outcomes are:

- LV function and
- the extent of myocardial ischemia (total ischemic burden).
CPG ON STABLE CAD

- Diagnosis
- Risk Stratification
- Management
Diagnosis

- History
- Physical Examination
- Clinical Investigations
- Resting ECG
- Echocardiography
- Non invasive tests
- Coronary Anatomy
A detailed *history and physical examination* are of paramount importance in making the diagnosis of Stable CAD.
Duration and Nature of Chest pain

- Stable CAD --- chest pain/symptoms of more than 2 months duration

- May present as:
  - Chest pain
  - Dyspnoea
  - Palpitations, near syncope and syncope.
Chest pain may be categorized into:

- **Stable angina (typical/definite angina)** – This is a clinical syndrome of retrosternal chest discomfort with the following characteristics and fulfilling these 3 criteria:
  1. predictable and with possible radiation to jaw, shoulders, arms and/or back
  2. provoked by physical exertion and/or emotional stress
  3. relieved by rest and/or with glyceryl trinitrate (GTN)

- **Atypical angina (probable)** – chest pain or discomfort which meets 2 out of the above 3 criteria.

- **Non-anginal chest pain or discomfort** – this meets 1 or none of the typical angina criteria.
This involves:

➢ inspection of the general habitus of the patient, looking for signs of anaemia, polycythaemia and stigmata of hyperlipidaemia.

➢ examination of the peripheral pulses.

➢ measurement of the blood pressure.

➢ auscultation of the precordium for additional heart sounds and murmurs and the carotid and renal arteries for bruit.

➢ excluding non-coronary causes of angina such as severe aortic stenosis, hypertrophic obstructive cardiomyopathy, and hyperthyroidism.
Clinical investigations are necessary for the:

- confirmation of the diagnosis and
- detection of myocardial ischemia and
- for prognostication.
Biochemical-

- Full Blood Count
- Fasting glucose and/or A1c
- Lipid profile - fasting or non fasting
- Renal profile - serum electrolytes and/or creatinine clearance or estimated GFR
- Liver Profile

Resting ECG – preferably during an episode of chest pain

Chest Radiography - Where indicated, it may be helpful in assessing cardiac size, pulmonary vasculature and excluding certain non-cardiac causes of chest pain.
Echocardiography is indicated:

- Presence of abnormal auscultatory findings and/or
- Presence of abnormal resting ECG and/or
- Assessment of LV function/regional wall motion abnormalities in patients with shortness of breath and/or known CAD.

It is a useful test to assess LV function in individuals with:

- Hypertension and/or diabetes.
- Chest pain suspected to be due to CAD.
Other non-invasive tests may be:

- Functional - for myocardial ischemia or
- Anatomical - for visualization of the coronary arteries
OTHER NON INVASIVE TESTS

May be performed for:

- Diagnosis of the chest pain/chest pain equivalents
- Prognosis
CPG ON STABLE CAD

FUNCTIONAL TESTS FOR MYOCARDIAL ISCHEMIA

▪ Exercise ECG

▪ Stress Echocardiogram
  ➢ Treadmill
  ➢ Dobutamine

▪ Cardiac MRI
  ➢ Vasodilators

▪ Nuclear perfusion studies (SPECT)
  ➢ Exercise
  ➢ Vasodilators
The choice of non-invasive test(s) will depend on the:

- Pre-Test Probability (PTP) of CAD in that individual
- Sensitivity and specificity of the different diagnostic modalities
## Pre Test Probability (PTP) of CAD in patients with stable Chest Pain*

*This is based on the EURO model – unfortunately no studies on the applicability of this model in the local population*

<table>
<thead>
<tr>
<th>Age</th>
<th>Typical angina</th>
<th>Atypical angina</th>
<th>Non-anginal pain</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
</tr>
<tr>
<td>30-39</td>
<td>59</td>
<td>28</td>
<td>29</td>
</tr>
<tr>
<td>40-49</td>
<td>69</td>
<td>37</td>
<td>38</td>
</tr>
<tr>
<td>50-59</td>
<td>77</td>
<td>47</td>
<td>49</td>
</tr>
<tr>
<td>60-69</td>
<td>84</td>
<td>58</td>
<td>59</td>
</tr>
<tr>
<td>70-79</td>
<td>89</td>
<td>68</td>
<td>69</td>
</tr>
<tr>
<td>&gt;80</td>
<td>93</td>
<td>76</td>
<td>78</td>
</tr>
</tbody>
</table>

Red boxes: High PTP >85%;
Yellow boxes: Intermediate PTP >15-%85%;
Green box: Low PTP <15%

Patients with a:

- **low PTP of <15%** can be assumed to have *no obstructive CAD*. In these individuals, CV risk factors should be treated to target. Other causes of chest pain should be looked for.

- **high PTP >85%** can be assumed to *have obstructive CAD* and invasive coronary angiography maybe a more appropriate initial investigation.

- **intermediate PTP (≥15-≤85%)** require further non-invasive evaluation.
Patients with intermediate PTP (≥15-≤85%) require further non-invasive evaluation.

The choice of non-invasive tests will depend:
✓ on the patient’s ability to exercise,
✓ ECG interpretability,
✓ obesity and the presence of good echo windows
✓ availability of local services and expertise
The choice of Functional non invasive test(s) will depend on the:

- Pre-Test Probability (PTP) of CAD in that individual
- Sensitivity and specificity of the different diagnostic modalities
## Sensitivity and Specificity of the Various Non-invasive Diagnostic tests for the Detection of CAD*

<table>
<thead>
<tr>
<th>Diagnosis of CAD</th>
<th>Sensitivity(%)</th>
<th>Specificity(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise ECG</td>
<td>45-50</td>
<td>85-90</td>
</tr>
<tr>
<td>Exercise stress echocardiography</td>
<td>80-85</td>
<td>80-88</td>
</tr>
<tr>
<td>Exercise stress SPECT</td>
<td>73-92</td>
<td>63-87</td>
</tr>
<tr>
<td>Dobutamine stress echocardiography</td>
<td>79-83</td>
<td>82-86</td>
</tr>
<tr>
<td>Dobutamine stress MRI</td>
<td>79-88</td>
<td>81-91</td>
</tr>
<tr>
<td>Vasodilator stress echocardiography</td>
<td>72-79</td>
<td>92-95</td>
</tr>
<tr>
<td>Vasodilator stress SPECT</td>
<td>90-91</td>
<td>75-84</td>
</tr>
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<td>Vasodilator stress MRI</td>
<td>67-94</td>
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<tr>
<td>Coronary CTA</td>
<td>95-99</td>
<td>64-83</td>
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<td>Vasodilator stress PET</td>
<td>81-97</td>
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FUNCTIONAL TESTS FOR MYOCARDIAL ISCHEMIA

- Exercise ECG

- Stress Echocardiogram
  - Treadmill
  - Dobutamine

- Cardiac MRI
  - Vasodilators

- Nuclear perfusion studies (SPECT)
  - Exercise
  - Vasodilators
Algorithm for the investigation of individuals with stable chest symptoms suspected to be due to CAD

Symptomatic individuals with intermediate pre-test likelihood of CAD (PTP >15% - < 85%)

- Normal ECG, Good exercise tolerance
  - Exercise stress test
    - Negative Test
      - Risk Factor Reduction ± Medical Therapy for CAD
    - Positive Test
      - **Invasive Coronary Angiogram

- Abnormal ECG, Limited exercise tolerance
  - *Exercise/ Dobutamine Stress Echo or
    - *Myocardial perfusion Imaging by SPECT or
    - *Cardiac Magnetic Resonance Imaging or
    - *Calcium score and/or CT coronary angiogram
  - Equivocal/Positive Test
    - **Invasive Coronary Angiogram

*The choice of non-invasive tests will depend on the patient’s ability to exercise, ECG interpretability, obesity and the presence of good echo windows and availability of local services and expertise

**In individuals with typical symptoms and a high pre-test likelihood of CAD (PTP>85%), an invasive coronary angiogram may be the initial investigation of choice (please refer to Appropriate Use Criteria for Investigations and Revascularization in CAD 2015 (1st edition): available at www.acadmed.org.my)
In the *diagnosis* of CAD: Exercise stress ECG is the non-invasive test of choice in patients who:

- can exercise and
- have interpretable ECGs.

If the exercise stress test is *negative* and:

- there is intermediate to high probability of CAD (PTP >65%), the patient should be referred for further evaluation
- there is low probability of CAD, (PTP 15-65%) appropriate risk reduction therapy and treatment of CV risk factors to target should be advised.
If the exercise stress test is \textit{positive} at:

- low workloads, the patient should be referred for an invasive coronary angiogram (ICA).

- moderate to high work loads, depending on the clinical condition, the patient may be referred for a non-invasive stress imaging test, Computerised Tomographic Coronary Angiogram (CTA) or an invasive coronary angiogram (ICA).
Stress imaging tests are useful in individuals who have intermediate PTP of CAD and who:

➢ are unable to exercise adequately and/or

➢ have uninterpretable resting ECG and/or

➢ have exercise stress ECG with equivocal results or which are abnormal at moderate to high
Other non-invasive tests may be:

- Functional - for myocardial ischemia or
- Anatomical - for visualization of the coronary arteries
EVALUATION OF CHEST PAIN & CHEST PAIN EQUIVALENTS

OTHER NON INVASIVE TESTS

- Coronary Cardiac CT
  - Coronary Calcium Score
  - CT coronary Angiogram
- Invasive coronary Angiogram
Coronary calcium score has been used to detect CAD.

Most studies demonstrated a high sensitivity but a much lower specificity, and an overall predictive accuracy of ≈70% in typical CAD patient populations.

CAC was found not to be superior to other noninvasive diagnostic modalities for the detection of CAD.

OTHER NON INVASIVE TESTS

- **Coronary Cardiac CT**
  - **Coronary Calcium Score**
  - **CT coronary Angiogram**

- **Invasive coronary Angiogram**
## Sensitivity and Specificity of the Various Non-invasive Diagnostic tests for the Detection of CAD

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</table>

EVALUATION OF CHEST PAIN & CHEST PAIN EQUIVALENTS

OTHER NON INVASIVE TESTS

- Coronary Cardiac CT
  - Coronary Calcium Score
  - CT coronary Angiogram
- Invasive coronary Angiogram
Invasive Coronary Angiography for the diagnosis of CAD

- Invasive Coronary angiography has been the “gold standard” for the diagnosis of CAD.

- It can detect obstructive lesions with negative remodelling accurately but unlike CTA, it may not be able to detect non-obstructive lesions with positive remodelling where the lumen diameter is maintained.

- ICA is rarely necessary in stable patients with suspected CAD for the sole purpose of establishing the diagnosis of CAD.

- It is indicated, following non-invasive risk stratification, to determine the most appropriate mode of revascularization.
CPG ON STABLE CAD

- Diagnosis
- Risk Stratification
- Management
CPG ON STABLE CAD

Risk Stratification

- Clinical Features
- Resting ECG
- Echocardiography
- Non invasive tests
- Coronary Anatomy and Physiological assessment
Risk may be defined as:

- high risk – annual mortality of >3%
- intermediate risk – annual mortality of 1-3%
- low risk – annual mortality of <1%
Clinical evaluation

Resting ECG

Assessment of LV function

Non-invasive assessment for myocardial ischaemia

Where indicated, evaluation of coronary anatomy and physiological assessment of the significance of the coronary lesion by Fractional Flow Reserve (FFR).
Clinical investigations are necessary for the:

- confirmation of the diagnosis and
- detection of myocardial ischemia and
- for prognostication.
<table>
<thead>
<tr>
<th>Modality</th>
<th>Definition of Risk</th>
<th>Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise Stress Test based on Duke Treadmill Score (DTS) (Table 7, pg 36)</td>
<td><strong>DTS: ≤ -11:</strong> High risk</td>
<td>Annual mortality &gt;5%</td>
</tr>
<tr>
<td></td>
<td><strong>DTS: +4 to -10:</strong> Moderate Risk</td>
<td>Annual mortality 0.25-5%</td>
</tr>
<tr>
<td></td>
<td><strong>DTS: ≥ +5:</strong> Low risk</td>
<td>Annual mortality &lt;0.25%</td>
</tr>
<tr>
<td>Stress Echocardiogram</td>
<td>Low risk: No inducible ischemia (negative test)</td>
<td>Annual rate of CV death /MI 0.54%, annual mortality &lt;1%</td>
</tr>
<tr>
<td></td>
<td>High Risk: inducible wall motion abnormalities in ≥3 segments of the standard LV model</td>
<td>Annual rate of CV death/MI: 4.5% (range: 3.8% to 5.9% /yr)</td>
</tr>
<tr>
<td>Modality</td>
<td>Definition of Risk</td>
<td>Risk</td>
</tr>
<tr>
<td>---------------------</td>
<td>-----------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Exercise MPI (nuclear)</strong></td>
<td>No inducible ischemia (Negative test)</td>
<td>Annual rate of CV death/MI: 0.45% per year</td>
</tr>
<tr>
<td></td>
<td>High risk: stress induced reversible perfusion defect (≥10% of total LV myocardium)</td>
<td>Annual rate of CV death/MI: 4.9% (interquartile range: 3.7% to 5.3%/year)</td>
</tr>
<tr>
<td><strong>Stress CMR</strong></td>
<td>No inducible ischemia (Negative test)</td>
<td>The 3-year event-free survival: 99.2%.</td>
</tr>
<tr>
<td></td>
<td>≥4 of 32 stress perfusion defects or ≥3 dysfunctional dobutamine induced segments</td>
<td>Annual risk of CAD death/ MI: ~5%</td>
</tr>
</tbody>
</table>
## Coronary Calcium Score for CV Risk Assessment

### Prognostic indicators for Adverse CV outcomes on Non-Invasive testing

<table>
<thead>
<tr>
<th>Calcium score</th>
<th>HR for incident MI and CHD mortality</th>
<th>CV Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-100</td>
<td>1.0</td>
<td>Mild</td>
</tr>
<tr>
<td>101-400</td>
<td>2.4</td>
<td>Moderate</td>
</tr>
<tr>
<td>401-1000</td>
<td>5.1</td>
<td>High</td>
</tr>
<tr>
<td>&gt;1000</td>
<td>7.6</td>
<td>Very High</td>
</tr>
<tr>
<td>Modality</td>
<td>Definition of Risk</td>
<td>Risk</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
<td>--------------------------------------------------------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>CT coronary Angiography (CTA)</td>
<td>Absence of any plaque</td>
<td>CV event rate is low - 0.24% for CV death/non-fatal MI; annual mortality: 0.28%.</td>
</tr>
<tr>
<td></td>
<td>Coronary plaque but without stenosis</td>
<td>Annual mortality rate is higher but remains &lt; 0.5%</td>
</tr>
<tr>
<td></td>
<td>Left main stenosis or proximal triple vessel disease</td>
<td>HR for all-cause mortality: 3.70</td>
</tr>
</tbody>
</table>
Clinical evaluation

Resting ECG

Assessment of LV function

Non-invasive assessment for myocardial ischaemia

Where indicated, evaluation of coronary anatomy and physiological assessment of the significance of the coronary lesion by Fractional Flow Reserve (FFR).
The prognosis is worse if:

- the greater the number of vessels involved.
- there is left main stem stenosis of >50%.
- the proximal LAD is involved.
2.2 a) Indications for Invasive coronary Angiography and Revascularization in Stable CAD

Invasive Coronary Angiography may be considered in the following clinical scenarios:

<table>
<thead>
<tr>
<th>Indications for Invasive Coronary Angiography in Stable CAD by symptoms and Non Invasive testing**</th>
<th>Appropriate Use Criteria (1-9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina Symptoms on *OMT Absent</td>
<td>Ischemia absent or present at high work- loads on non- invasive testing</td>
</tr>
<tr>
<td></td>
<td>Ischemia present at low or intermediate work- loads on non- invasive testing</td>
</tr>
<tr>
<td></td>
<td>Ischemia absent or present at high work- loads on non- invasive testing</td>
</tr>
<tr>
<td></td>
<td>Ischemia present at low or intermediate work- loads on non- invasive testing</td>
</tr>
<tr>
<td>Minimal CCSI- II</td>
<td>Ischemia absent or present at high work- loads on non- invasive testing</td>
</tr>
<tr>
<td>Moderate to severe CCS III-IV</td>
<td>Ischemia absent on non- invasive testing</td>
</tr>
<tr>
<td></td>
<td>Ischemia present on non- invasive testing</td>
</tr>
</tbody>
</table>

* OMT includes lifestyle changes, antiplatelet agents, - blockers, statins and at least 2 different classes of anti- angina medications at maximal tolerated doses for at least 2 weeks
** Appendix IV, pg 78
In general, individuals with:

- no ischemia demonstrated by non-invasive testing and/or
- have no or minimal plaque in the coronary arteries by CTA

have an excellent prognosis with a rate of CV death/non-fatal MI of <0.5% and an annual mortality of <1%.
Low risk individuals should be managed with risk factor reduction and/or anti anginal medications as necessary. Revascularization has not been shown to improve their long-term CV outcomes.

Intermediate risk individuals may be managed with risk reduction strategies +/- anti anginal therapy or considered for invasive coronary angiogram and revascularization depending on the clinical condition, ischemic burden and patient preferences.

High risk individuals, in addition to risk reduction strategies, should be considered for invasive coronary angiography with view to revascularization.
CPG ON STABLE CAD

- Diagnosis
  - History
  - Physical Examination
  - Clinical Investigations

- Risk Stratification
  - Clinical Features
  - Resting ECG
  - Echocardiography
  - Non invasive tests
  - Coronary Anatomy and Physiological assessment

- Management
The management of the patients with Stable CAD should be multifaceted.

It involves *optimal medical therapy* which includes both:

- behavioural modification therapy *and*
- pharmacological therapy.
Behavioural modification therapy (BMT) – includes patient education and lifestyle modification.

➢ patient education about the illness
➢ appropriate dietary modification
➢ regular physical activity
➢ smoking cessation
➢ weight management
Pharmacological therapy

This aims at:

➢ prevention of CV events
➢ relieving symptoms
Prevention of CV events

➢ All patients should receive:
  ▪ aspirin and a
  ▪ statin (+/- non-statin therapy) with the aim of achieving a LDL-C <1.8 mmol/l – the lower the better.

➢ All CV risk factors should be treated to target.
Patients with depressed LV function (LVEF <40%) should receive:

- ACEi/ARB,
- β-blockers and
- mineralocorticoid antagonists—spironolactone, epleronone.
- Angiotensin -receptor -neprilysin inhibitors may also be considered.
Relieving symptoms

➢ β-blockers and/or calcium channel blockers (CCBs) should be prescribed as first-line treatment to reduce angina because they are widely available.

➢ Ivabradine, trimetazidine, long-acting nitrates and ranolazine are recommended as add-on therapy in patients who remain symptomatic.
Management of Stable CAD

Symptom control

- Short-acting nitrate, e.g. GTN + β-blocker and/or CCB

If symptoms persist, consider:
- long-acting Nitrates
- trimetazidine
- ivabradine
- ranolazine
- nicorandil

Prevention of CV events

- Lifestyle modification
- Risk factor control
- Aspirin 100mg once daily (Clopidogrel in Aspirin intolerance)
- Lipid-lowering therapy to target
- Consider ACEi/ARB in the presence of:
  - Diabetes
  - Hypertension
  - LV dysfunction (EF<40%)
- Consider β-blocker for LV dysfunction (EF<40%)

If symptoms not controlled or large ischaemic burden by non-invasive testing

Consider coronary angiography with view for revascularization.
Optimal medical therapy should be instituted prior to revascularization procedures.

The decision to revascularize patients with stable CAD on OMT will depend on:

- symptoms – presence of angina affecting quality of life.
- extent of ischemia as determined by non-invasive testing – mild vs moderate to severe myocardial ischemia
- extent of coronary disease and where applicable physiological functional testing using FFR.
FFR is calculated as the ratio of distal coronary pressure to aortic pressure measured during maximal hyperaemia. A normal value for FFR is 1.0 regardless of the status of the microcirculation.

Physiological functional testing using FFR:
- FFR <0.75 – benefit from revascularization as compared to OMT.
- FFR between >0.75 but <0.8 – have intermediate benefit with revascularization and management should be based on clinical judgement.
- FFR >0.8 – no benefit from revascularization.
Wherever possible, a discussion with the patient and Heart Team should be encouraged prior to revascularization to determine the best strategy – PCI or CABG.
AUC FOR INVESTIGATIONS AND REVASCULARIZATION IN CAD
## AUC FOR CORONARY REVASCULARIZATION IN STABLE CAD

<table>
<thead>
<tr>
<th>Revascularization in Stable CAD by extent of CAD in patients already on OMT# and still having symptoms and / or ischemia (anatomical and functional)*</th>
<th>Appropriate Use Criteria (1-9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left main stenosis &gt; 50%**</td>
<td>A9</td>
</tr>
<tr>
<td>Any significant proximal LAD stenosis &gt; 70%**</td>
<td>A9</td>
</tr>
<tr>
<td>2 or 3 vessel CAD with significant stenosis &gt; 70%** and LVEF &lt; 40%***</td>
<td>A8</td>
</tr>
<tr>
<td>2 or 3 vessel CAD with significant stenosis &gt; 70%** and LVEF &gt; 40%***</td>
<td>A8</td>
</tr>
<tr>
<td>1 vessel CAD with stenosis &gt; 70%** and LVEF &lt; 40%***</td>
<td>A8</td>
</tr>
<tr>
<td>1 vessel CAD with stenosis &gt; 70%** and LVEF &gt; 40%***</td>
<td>A7</td>
</tr>
</tbody>
</table>
PCI vs CABG

BMS PCI vs SA CABG
Survival 0-9 yrs
76.3% vs. 86.9%; p < 0.001) HR 0.38

BMS PCI vs MA CABG
Survival at 5 yrs (86.3% vs. 95.6%)
Survival at 9 yrs (82.8% vs. 89.8%)
(HR: 0.45; p < 0.001)

DES PCI vs SA CABG
Survival 0 to 3 years
HR: 1.06; p = 0.615

DES-PCI vs MA CABG
Survival at 5 yrs (86.3% vs. 95.6%)
Survival at 9 yrs (82.8% vs. 89.8%)
(HR: 0.45; p < 0.001)

PCI vs CABG
## Mode of Revascularization in Stable CAD and UA/NSTEMI after initial medical stabilization

<table>
<thead>
<tr>
<th>Coronary angiographic findings&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Appropriate Use Grade&lt;sup&gt;1-8&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PCI</td>
</tr>
<tr>
<td>1  Left main stenosis and additional CAD with intermediate to high CAD burden (Syntax score &gt; 22)</td>
<td>Diabetes present</td>
</tr>
<tr>
<td></td>
<td>Diabetes absent</td>
</tr>
<tr>
<td>2  Left main stenosis and additional CAD with low CAD burden (Syntax score &lt; 22)</td>
<td>Diabetes present</td>
</tr>
<tr>
<td></td>
<td>Diabetes absent</td>
</tr>
<tr>
<td>3  Isolated Left main stenosis (ostial and/or body)</td>
<td>Diabetes present</td>
</tr>
<tr>
<td></td>
<td>Diabetes absent</td>
</tr>
<tr>
<td>4  Triple vessel disease with intermediate to high CAD burden (Syntax score &gt; 22)</td>
<td>Diabetes present</td>
</tr>
<tr>
<td></td>
<td>Diabetes absent</td>
</tr>
<tr>
<td>5  Triple vessel disease with low CAD burden (Syntax score &lt; 22)</td>
<td>Diabetes present</td>
</tr>
<tr>
<td></td>
<td>Diabetes absent</td>
</tr>
<tr>
<td>6  Two vessel disease</td>
<td>Proximal LAD involved</td>
</tr>
<tr>
<td></td>
<td>Diabetes absent</td>
</tr>
<tr>
<td></td>
<td>Proximal LAD not involved</td>
</tr>
<tr>
<td></td>
<td>Diabetes absent</td>
</tr>
<tr>
<td>7  Single vessel disease with symptoms and ischemia despite OMT</td>
<td>Proximal LAD involved</td>
</tr>
<tr>
<td></td>
<td>Proximal LAD not involved</td>
</tr>
</tbody>
</table>

<sup>a</sup> CAD burden is determined by Syntax score<sup>b</sup> should be: (see Appendix VIII, pg 82)
2\textsuperscript{nd} ED CLINICAL PRACTICE GUIDELINES

on

STABLE CAD

2018