Key Message:
- Four types of medication (ACE-Inhibitors, beta-blockers, statins & ASA) have been shown to reduce cardiovascular risk in post-MI patients. These benefits are in addition to risk factor management (e.g. diet, lifestyle, exercise) and occur regardless of the presence of hypertension, dyslipidemia, or LV dysfunction. Clinical judgment is essential to assess risk/benefit for individual patients.

How are we doing in Saskatchewan?
- An analysis of Saskatchewan dispensing rates by the HQC suggests that important drugs are underutilized (See Figure 1).

PRACTICAL ISSUES – ACE Inhibitors (ACEI)
Which patients will benefit?
- All post MI patients without contraindications (indefinitely)
- Shown to reduce risk of CV events in post-MI patients who are high risk (elderly, LV dysfunction); some benefit also in lower risk patients (e.g. young, no LV dysfunction)
- Beneficial when initiated soon after acute MI (~first 24hrs) AHA'04

Initiation & dosing in patients with renal dysfunction
- Ensure Scr is stable before initiating ACEI therapy
- Start with low doses, slowly titrating towards targets with close monitoring. A moderate rise in Scr (that stabilizes within 1 week) may occur after each dose increase. (Of note: ACEI beneficial if existing renal impairment but may consider nephrology consult; trials exclude patients with high Scr (e.g. >200umol/l TRACE).
- Check Scr, BUN, and lytes at baseline and 7-14 days after each dose increase. If Scr rise (above baseline) is:
  - <30% - continue titration / no concern
  - 30-50% – decrease ACEI dose by 50% and recheck Scr in 7-10 days. If Scr rise still >30%, stop the ACEI
  - >50% – stop ACEI
  - When Scr rise is >30% consider investigating for renal artery stenosis and rule out other reversible causes.
  - Common reversible causes include: heart failure, aggressive diuresis, volume depletion, NSAIDs/coxibs & dehydration.
- Potassium levels above 5.6mmol/L during ACEI therapy should prompt reassessment of ACEI.

Initiation in patients with hyperkalemia
- Potassium should be ≤5.0mmol/L before initiating
- Identify reversible causes of the baseline hyperkalemia: Concurrent NSAIDs/coxibs or potassium sparing diuretics eg. spironolactone, dietary indiscretion -dietician counseling may be helpful.

Role of angiotensin receptor blockers (ARBs)
- Acceptable alternative when ACEIs not tolerated
- Studied only in post-MI patients with LV dysfunction; valsartan at very high dosage showed equivalency to ACEI VALIANT, losartan at lower dose was less effective than an ACEI OPTIMAL.
- Combination of an ACEI + ARB no more effective but more adverse effects than either ACEI or ARB alone VALIANT

Figure 1: % of Sask. Patients on Target Therapy @90days Post-MI

PRACTICAL ISSUES – Beta-blockers (BB)
Which patients will benefit?
- Shown to reduce all-cause mortality in all post-MI patients regardless of LV function, especially those at high risk (when initiated within 4 weeks of MI and continued for up to 4 years)
- Benefit also in lower risk patients (e.g. young, no LV dysfunction)
- Beta-blockers may be especially underutilized in the elderly.

Contraindications – myths and preconceptions
- Many conditions that previously contraindicated the use of beta-blockers are not “absolute”. With cautious initiation and close monitoring, benefits may outweigh the risks in the following:
  - COPD, diabetes, peripheral artery disease, & compensated HF; mild asthma (cardioselective BBs in those well controlled with inhaled steroids)

PRACTICAL ISSUES – Statins
Which patients will benefit?
- ALL post-MI patients appear to benefit from statin therapy regardless of lipid levels (HPS ~40% patients post-MI history) AHA'04

Should “high dose” statins be used in acute MI?
- Aggressive dose (atorvastatin 80mg OD) was better than moderate dose (pravastatin 40mg OD) when initiated <10 days after acute MI and continued for 2 years. MIRACL.
- Caution: risk of adverse effects (liver, muscle) with aggressive statin doses.

Monitoring
- Guidelines recommend baseline transaminase and CK levels before starting any patient on a statin AHA 23, ATP III 24
- Frequent laboratory monitoring may be necessary in patients at high risk for adverse effects (e.g. drug interactions, elderly, renal / hepatic dysfunction, high dose statins, niacin or fibrate combinations)

PRACTICAL ISSUES – ASA
- Recommend 81mg enteric coated daily (75mg-150mg) AHA'04
- Consider H. pylori eradication and cytoprotection for patients at high-risk of a GI bleed, even for ≤100mg ASA.
- Minimize regular use of ibuprofen MOTRIN, ADVIL with ASA since antiplatelet effects may be blocked (conflicting data)
- High-dose NSAIDs/COXIBs may be associated with adverse heart outcomes (eg. rofecoxib)

For specific drug & dosage considerations, see Page 2 - Table 1.
### Table 1: Post-MI – Drug and Dosage Considerations

**POST-MI TARGET DOSES**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Target Dose</th>
<th>Controlled Trials</th>
<th>$/30d</th>
<th>Benefits</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ACEI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ramipril ALTACE</td>
<td>10mg HS</td>
<td>HOPE, 5mg BID AIRE 31</td>
<td>41</td>
<td>• all-cause mortality: 17-29% RRR when started 2-16 days after event &amp; continued for 4-5 yrs in pts with LV dysfx.</td>
<td>• Adverse effects include cough.</td>
</tr>
<tr>
<td>Trandolapril MAVIK</td>
<td>4mg OD</td>
<td>TRACE 6</td>
<td>41</td>
<td>• prevents ventricular remodeling; ↓ proteinuria</td>
<td>• AHA STEMI Guidelines suggest to use ACE inhibitors in all pts indefinitely.</td>
</tr>
<tr>
<td>Lisinopril ZESTRIL/PRINIVIL</td>
<td>10mg OD</td>
<td>GISSI-3 32</td>
<td>30</td>
<td>• 16% RRR in all cause mortality in pts started in high risk pts with remote history of MI and continued for years</td>
<td>• Most benefit if started 2-16 days after event &amp; continued for 4-5 yrs in pts with LV dysfx.</td>
</tr>
<tr>
<td>(high dose)</td>
<td>~35mg OD</td>
<td>ATLAS 33 (BP)</td>
<td>65</td>
<td>• ACEI, TRACE, SAVE.</td>
<td></td>
</tr>
<tr>
<td>Perindopril COVERSIL</td>
<td>8mg OD</td>
<td>EUROPA 9</td>
<td>45</td>
<td>• increases ventricular remodeling; ↓ proteinuria</td>
<td></td>
</tr>
<tr>
<td>Enalapril VASOTE</td>
<td>20mg OD</td>
<td>CONSENSUS II 34</td>
<td>48</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Captopril CAPOTEN</td>
<td>50mg TID</td>
<td>SAVE, BID sp n 56</td>
<td>52</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ARB</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Valsartan DIOVAN</td>
<td>160mg BID</td>
<td>VALIANT 42</td>
<td>82</td>
<td>• all-cause mortality: valsartan, captopril[153,154], or combo equally effective</td>
<td>• Alternative if ACEI not tolerated &amp; HF/LVEF&lt;0.4.</td>
</tr>
<tr>
<td>Candesartan ATACAND</td>
<td>32mg OD</td>
<td>CARE-2, -3, -4</td>
<td>87</td>
<td>• ↓ proteinuria[35] even in pts with Scr&gt;265</td>
<td></td>
</tr>
<tr>
<td><strong>β-Blocker</strong></td>
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</tr>
<tr>
<td>Metoprolol LOPRESSOR</td>
<td>100mg BID</td>
<td>HAMALCOR 43</td>
<td>16</td>
<td>• all-cause mortality: 22.9% RRR when started in any pt within 5-28 days of MI &amp; continued for up to 4yr;</td>
<td>• Adverse effects reduce hypotension, dizziness, bradycardia, fatigue, insomnia, vivid dreams, &amp; sexual dysfunction</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>&lt;200mg SR OD</td>
<td>MERIT-HF 44, 45</td>
<td>20</td>
<td>• best long-term evidence with propranolol, metoprolol &amp; timolol</td>
<td></td>
</tr>
<tr>
<td>Atenolol* TENORMIN</td>
<td>100mg OD</td>
<td>SES-4 46</td>
<td>24</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carvedilol COREG</td>
<td>25mg BID</td>
<td></td>
<td>58</td>
<td></td>
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</tr>
<tr>
<td>Propranolol INDERAL</td>
<td>60-80mg TID</td>
<td>BID QT 47</td>
<td>14</td>
<td>• ↓ sudden death, reinfarction &amp; arrhythmias</td>
<td></td>
</tr>
<tr>
<td>Timolol BLOCADREN</td>
<td>10mg BID</td>
<td>NMCG 49</td>
<td>25</td>
<td>• Less benefit: ISAs (pindolol, acebutolol)</td>
<td></td>
</tr>
<tr>
<td>Acebutolol* &amp; LADA MONICAN</td>
<td>200mg BID</td>
<td>APIS 50</td>
<td>22</td>
<td>• Cardioselective agents (↑) preferred for mild &amp; moderate hypertension</td>
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</tr>
<tr>
<td><strong>Statins</strong></td>
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<tr>
<td>Simvastatin ZOCOR</td>
<td>20-40mg OD</td>
<td>4S 19</td>
<td>16</td>
<td>• all-cause mortality: 10.3% RRR in post MI pts with ↑ left ventricular EF</td>
<td>• Higher levels in Asians; rhabdomyolysis cases at doses 2-3x the usual dose.</td>
</tr>
<tr>
<td>Atorvastatin LIPISTOR</td>
<td>10mg OD</td>
<td>4S, PRP 20</td>
<td>56</td>
<td>• in pts at high CV risk</td>
<td></td>
</tr>
<tr>
<td>(high dose in ACS)</td>
<td>80mg OD</td>
<td>PROVE IT 21</td>
<td>87</td>
<td>• ↑ in major CV events</td>
<td></td>
</tr>
<tr>
<td>Pravastatin PRAVACHOL</td>
<td>40mg OD</td>
<td>LIPID 45, CARE 50</td>
<td>44</td>
<td>• in pts at high CV risk</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Rosuvastatin CRESTO – no outcome trials yet; [91, 92] 10mg OD</td>
<td></td>
<td>96</td>
<td></td>
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<tr>
<td>Higher levels in Asians; rhabdomyolysis cases at doses (20-40mg)</td>
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<td>56</td>
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<td><strong>ASA</strong></td>
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<tr>
<td>Aspirin</td>
<td>80-160mg OD</td>
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<tr>
<td>Generally start at 81mg enteric coated OD; [ASA≤100mg]</td>
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<td><strong>Anti-Platelet</strong></td>
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<td><strong>Lipid Lowering</strong></td>
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<td>Cholesterol</td>
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<tr>
<td>Spironolactone ALDACTONE</td>
<td>12.5-25mg OD</td>
<td>for severe HF; Class IIb RALES 70</td>
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<td></td>
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<tr>
<td><strong>Other</strong></td>
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<tr>
<td>Spironolactone ALDACTONE</td>
<td>12.5-25mg OD</td>
<td>for severe HF; Class IIb RALES 70</td>
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<tr>
<td>BP: BP Canadian 2004 [73]; General &lt;140/90; Diabetes &lt;130/80;若无蛋白尿, &lt;125/75;若蛋白尿 ≥1g/d.</td>
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<tr>
<td>GLUCOSE: Canadian 2003 [77]</td>
<td>Target for most: A1C ≤7%; FPG 4-7 mmol; PPBG 2hr post 5-10 mmol. If can be done safely without hypoglycemia.</td>
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</tbody>
</table>

**Risks**

- Adverse effects include cough; hyperdipoxia/hyponatremia; hyperkalemia; renal insufficiency (in pts with renal artery stenosis); angioedema; taste changes; rash; pancreatitis & blood dyscrasias.
- AHA STEMI Guidelines suggest to use ACE inhibitors in all pts indefinitely.
- Contraindicated in pts with bilateral renal artery stenosis (or unilateral stenosis if only 1 kidney).
- AHA STEMI Guidelines suggest to use beta-blockers in all pts indefinitely.
- Contraindicated in pts with severe/poorly controlled asthma. 2nd or 3rd degree heart block; HR≤50; SBP <90; & decompensated heart failure
- Some believe carvedilol better than metoprolol for HF but equivalent doses may not have been used.
- CNS adverse effects (depresion, impotence, fatigue) overestimated; common in placebo groups & may not be solely related to beta-blockers
- May start low-dose & titrate up to high risk for side effects (ie elderly, renal/hepatic dysfu, nia or fibrate combos, drug interactions, high dose or ho of intolerance)

**Comments**

- AHA STEMI Guidelines suggest to use statins in all patients (even when baseline LDL ≤ 2.5 mmol/L).
- β-Blockers generally started at 81mg enteric coated OD; [ASA≤100mg] as effective/less bleeding than 325mg, especially with Plavix.
- Steins: If on ASA+warfarin INR 2.3 for anticoagulation then D/C Plavix after: 2-month-bare metal; ≥3-months sirolimus; ≥6-month-paclitaxel.
- Adverse effects include GI upset, muscle aches, elevated LFTs, myopathy, hypotension, rhabdomyolysis; impotence; Rare: lupus-like symptoms, perip neuropathy.
- Adverse effects due to higher levels in Asians; rhabdomyolysis cases at doses 2-3x the usual dose.
- Beta-blockers, along with optimal antplatelet and statin therapy, are key components of post-MI therapy for patients with LV dysfx.
We would like to acknowledge the following contributors and reviewers: Dr. T. Wilson (Internal Medicine/Pharmacology), Dr. R. Barlow (Cardiology), Dr. T. Lauber (Family Medicine), Dr. G. Pylypchuk (Nephrology), Dr. M. Marcinick (Respiratory), Dr. B. Semchuk (Pharmacy), & the RxFiles Advisory Committee. Committee members: D. Jorgenson, P. Pedersen, M. McNally, J. Rivers, D. Marshall, J. Emery, R. Jenkins, & D. Smith. 

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