Clinical Practice Guidelines:
Toxicology and toxinology/Calcium channel blocker

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<table>
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<tr>
<th>Date</th>
<th>March, 2017</th>
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<tr>
<td>Purpose</td>
<td>To ensure to consistent approach to the management of Calcium channel blocker poisoning.</td>
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<td>Scope</td>
<td>Applies to all QAS clinical staff.</td>
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<td>Information security</td>
<td>This document has been security classified using the Queensland Government Information Security Classification Framework (QGISCF) as UNCLASSIFIED and will be managed according to the requirements of the QGISF.</td>
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Calcium channel blockers (CCBs) are commonly prescribed medications used in the treatment of hypertension, angina pectoris and cardiac arrhythmia.[1]  

Examples:
- Verapamil
- Diltiazem
- Amlodipine
- Nifedipine
- Felodipine
- Lercanidipine
- Nimodipine

In overdose calcium channel blockers can have vascular and/or cardiac effects. This will result in changes in myocardial conduction, contractility and vascular vasodilation.[2-3,4] Calcium gluconate 10% is the antidote for CCB toxicity.

Clinical features

Cardiovascular effects
- bradycardia
- heart block
- hypotension
- cardiogenic shock

Systemic effects
- seizures
- coma
- hyperglycaemia
- metabolic acidosis

Risk assessment

CCB toxicity is potentially life-threatening, particularly verapamil and diltiazem which are the more cardioselective CCBs.

High risk populations:
- Underlying cardiorespiratory disease
- Elderly
- Co-ingestion with beta blockers or digoxin
**Additional information**

- The onset of clinical features may be delayed up to 16 hours following ingestion of slow release preparations.

**Note:** Officers are only to perform procedures for which they have received specific training and authorisation by the QAS.