**Generic Name**

Dicyclomine

**IUPAC Name**

2-diethylaminoethyl 1-cyclohexylcyclohexane-1-carboxylate

**Chemical Structure**

![Chemical Structure of Dicyclomine]

**Molecular Weight**

309.48

**Molecular formula**

C₁₉H₃₅NO₂

**Melting Point**

172-174 °C

**State**

Solid

**Drug Type**

Approved

**Pharmacology**

Dicyclomine is an anticholinergic drug, a medication that reduces the effect of acetylcholine, a chemical released from nerves that stimulates muscles, by blocking the receptors for acetylcholine on smooth muscle. It also has a direct relaxing effect on smooth muscle. Dicyclomine is used to treat or prevent spasm in the muscles of the gastrointestinal tract in the irritable bowel syndrome. In addition, dicyclomine inhibits gastrointestinal propulsive motility and decreases gastric acid secretion and controls excessive pharyngeal, tracheal and bronchial secretions.

**Mechanism of Action**

Action is achieved via a dual mechanism: (1) a specific anticholinergic effect (antimuscarinic) at the acetylcholine-receptor sites and (2) a direct effect upon smooth muscle (musculotropic).
Pharmacokinetic Profile

Dicyclomine is absorbed rapidly from the GI tract, achieving peak plasma concentrations within 1–1.5 hours after oral administration. The apparent volume of distribution of dicyclomine is reportedly 3.65 L/kg. It is metabolized by hepatic or enzymatic hydrolysis and eliminated by Urine.

Adverse Reactions

- **Cardiovascular**: Syncope, tachycardia, palpitation.
- **Central nervous system**: Dizziness, lightheadedness, tingling, headache.
- **Dermatologic**: Rash, urticaria, itching, and other dermal manifestations.
- **Respiratory**: Dyspnea, apnea, asphyxia, nasal stuffiness or congestion.

Indications

For the treatment of functional bowel/irritable bowel syndrome including abdominal pain; diverticulitis.

Method for Estimation

Several analytical techniques including titrimetric, HPLC and spectrophotometric (Bebawy et al., 2003) have been reported for analysis of dicyclomine in combination with other drugs. Spectrophotometric method was used in present studies because it is simple, rapid, and economical methods for the estimation of dicyclomine.
Generic Name: Paracetamol [INN]

IUPAC Name: Acetamide, N-(4-hydroxyphenyl)-, N-Acetyl-p-aminophenol; p-Acetamidophenol

Chemical Structure:

![Chemical Structure Diagram]

Molecular Weight: 151.16

Molecular formula: C₈H₉NO₂

Melting Point: 174-176 °C

State: Solid

Drug Type: Approved

Pharmacology:

Paracetamol (INN) is a popular analgesic and antipyretic drug that is used for the relief of fever, headaches, other minor aches and pains. It is a major ingredient in numerous cold and flu medications and many prescription analgesics. It is extremely safe in standard doses, but because of its wide availability, deliberate or accidental overdoses are not uncommon. Acetaminophen, unlike other common analgesics such as aspirin and ibuprofen, has no anti-inflammatory properties or effects on platelet function, and so it is not a member of the class of drugs known as non-steroidal anti-inflammatory drugs or NSAIDs. In normal doses acetaminophen does not irritate the lining of the stomach nor affect blood coagulation, the kidneys, or the fetal ductus arteriosus (as NSAIDs can). Like NSAIDs and unlike opioid analgesics, acetaminophen does not cause euphoria or alter mood in any way. Acetaminophen and NSAIDs have the benefit of being completely free of problems with addiction, dependence, tolerance and withdrawal. Acetaminophen is used on its own or in...
combination with pseudoephedrine, dextromethorphan, chlorpheniramine, diphenhydramine, doxylamine, codeine, hydrocodone.

**Mechanism of Action**

Paracetamol is thought to act primarily in the CNS, increasing the pain threshold by inhibiting both isoforms of cyclooxygenase, COX-1 and COX-2, enzymes involved in prostaglandin (PG) synthesis. Unlike NSAIDs, acetaminophen does not inhibit cyclooxygenase in peripheral tissues and, thus, has no peripheral anti-inflammatory affects. While aspirin acts as an irreversible inhibitor of COX and directly blocks the enzyme's active site, studies have found that acetaminophen indirectly blocks COX, and that this blockade is ineffective in the presence of peroxides. This might explain why acetaminophen is effective in the central nervous system and in endothelial cells but not in platelets and immune cells which have high levels of peroxides. Studies also report data suggesting that acetaminophen selectively blocks a variant of the COX enzyme that is different from the known variants COX-1 and COX-2. This enzyme is now referred to as COX-3. Its exact mechanism of action is still poorly understood.

**Pharmacokinetics and Metabolism**

Paracetamol has excellent oral bioavailability. Peak plasma concentrations occur within 30–60 minutes and the t ½ in plasma is 2 hours. Binding of the drug to plasma proteins is less than with other NSAIDs. 90–100% of the drug may be recovered in the urine. A small proportion of acetaminophen undergoes CYP-mediated N-hydroxylation to form N-acetyl-p-benzoquinoneimine (NAPQI), a highly reactive intermediate.

**Adverse Effects**

Erythematous or urticarial rash, drug fever, mucosal lesions, hepatic necrosis, renal tubular necrosis, hypoglycemic coma.

**Indications**

For the treatment of arthritic and rheumatic conditions, headache, pain, dysmenorrheal, myalgias and neuralgias.
Method for Estimation

Several analytical techniques including titrimetric (Maheshwari et al., 2006), colourimetric, spectrofluorimetric (Kousy 1999), densitometric, HPLC (Raja et al., 2005), spectrophotometric (Nikam et al., 2008) and stripping voltametric (Posac et al., 1995) have been reported for analysis of paracetamol alone or in combination with other drugs. Spectrophotometric method was used in present studies because it is simple, rapid, and economical methods for the estimation of paracetamol.