Mechanism of Action
Chlormethiazole, possesses anti-neuropeptide, muscimol (a GABAA agonist), and glycine pound potentiates inhibitory effects of GABA (an inhibitory neuropeptide), possesses anti-inflammatory actions by inhibiting the release of p38 MAP kinase (a stress-mediator protein) and any component of the formulation; acute pulmonary insufficiency. Use with caution in patients at increased risk of developing hypoxia (eg, cardiac insufficiency, respiratory insufficiency). Avoid use in patients who develop hypoxia. The underlying condition causing hypoxia should be treated since it may manifest as confusion in some patients. Use with caution in patients with chronic pulmonary insufficiency, use is contraindicated in acute pulmonary insufficiency. Use with caution in patients with chronic renal disease. Use with caution in patients with sleep apnea.

Use with caution in geriatric patients due to potential increased drug bioavailability and delayed elimination. Some formulations may contain sorbitol; avoid use in patients with hereditary fructose intolerance.

Adverse Reactions
Frequency not defined:
- Central nervous system: Confusion (older adults; rare), hangover effect (older adults; rare), paradoxical excitation, delirium (older adults; rare), severe headache, severe sedation (older adults)
- Hepatic: Increased serum bilirubin (reversible)
- Hypersensitivity: Anaphylaxis
- Ophthalmic: Conjunctival irritation
- Respiratory: Increased bronchial secretions, nasal congestion, nasal mucosa irritation, rhinorrhea

<1%, postmarketing, and/or case reports:
- Bullous rash (rare), gastrointestinal disease, increased serum transaminases (reversible), skin rash, urticaria

Drug Interactions
Metabolism/Transport Effects Inhibits CYP2E1 (moderate)
Avoid Concomitant Use
Acetaminophen use with caution. Chlormethiazole with any of the following:
- Azelastine (Nasal; Oral)
- Oxycodeone; Oxomazine; Paraldehyde; Thalidomide

Increased Effect/Toxicity
Chloprothixene may increase the levels/effects of:
- Aze-lastine (Nasal; Oral
- Buprenorphine; CNS Depressants; CYP2E1 Substrates; Flunitrazepam; Hydroxyzine; Levodopa; Methotrimeprazine; MetyroSINE
- Ondansetron; OxyCODONE; Paraldehyde; Pribedil; Pramipexole; ROPINIRole; Rotigotine; Selective Seroto-
orphan; Sunovex; Thalidomide; Zolpidem

The levels/effects of Chlormethiazole may be increased by:
- Alcohol (Ethyl); Bromidine (Topical); Cannabis
- Chlorphenesin Carbamate; Cimetidine; Dimethindene (Topical); Dronabinol; Droperidol; Kava Kava; Lofexidine; Magnesium Sulfate; Melatonin; Nabilone; Oxomazine; Perampelan; Rufinamide; Sodium Oxbate; Tapentadol; Tetrahydrocannabinol

Decreased Effect
CarBAMazepine
Storage/Stability Store below 25°C (77°F).
Mechanism of Action Chlormethiazole, possesses anti-neuropeptide, muscimol (a GABAA agonist), and glycine pound potentiates inhibitory effects of GABA (an inhibitory neuropeptide), possesses anti-inflammatory actions by inhibiting the release of p38 MAP kinase (a stress-mediator protein) (Luo 2015; Wilby 2004).
Pharmacodynamics/Kinetics
Mechanism: Hepatic
Bioavailability: 12% (Pentikainen 1978)
Excretion: Urine (unchanged drug <0.1%) (Pentikai-
nen 1978)
Clomiphene [CLOMIPHENE] (ARM 2008; SOGC-CFAS 2011). This syndrome may begin within 24 hours of treatment, but may become most severe 7 to 10 days after therapy (SOGC-CFAS 2011). OHSS is typically self-limiting with spontaneous resolution, although it may be more severe and protracted if pregnancy occurs (ASRM 2008). Symptoms of ovarian enlargement in patients with OHSS may include abdominal distension/discomfort, diarrhea, nausea, and/or vomiting. Severe OHSS symptoms may include ovarian enlargement that is severe, acute respiratory distress syndrome, anuria/oliguria, ascites, dyspnea, hypotension, nausea/vomiting (intractable), pericardial effusions, tachycardia, or thromboembolism. Decreased estrogen levels, hemococoncentration, hypoproteinemia, elevated liver enzymes, elevated WBC, and electrolyte imbalances may also be present (ASRM 2008; Fiedler 2012; SOGC-CFAS 2011). If severe OHSS occurs, stop treatment, and consider hospitalizing the patient (ASRM 2008; SOGC-CFAS 2011). Treatment is primarily symptomatic and includes fluid and electrolyte management, bedrest, and prevention of thromboembolic complications (ASRM 2008; SOGC-CFAS 2011). The ascitic, pleural, and pericardial fluids may be removed if needed to relieve symptoms (eg, pulmonary distress or cardiac tamponade) (ASRM 2008; SOGC-CFAS 2011). Women with OHSS should avoid pelvic examination and/or intercourse (ASRM 2008; SOGC-CFAS 2011). Appropriate use: To minimize risks, use only at the lowest effective dose for the shortest duration of therapy (espe- cially for the first course of therapy). Women with polycystic ovary syndrome-galactorrhea syndrome, psychogenic amenorrhea, uncontrolled thyroid or adrenal dysfunction; or any of its components; liver disease or history of liver neoplasm, optic neuritis, ovarian cyst, ovarian hemorrhage, palpitations, phlebitis, pruritus, psychosis, pulmonary embolism, renal hemorrhage, retinal hemorrhage, retinal thrombosis, retinal vein occlusion, spasms, syncope, tachycardia, thrombophlebitis, thyroid disease, tinnitus, uterine hemorrhage, vision loss (temporary/prolonged), vitreous detachment (posterior) Drug Interactions Metabolism/Transport Effects None known. Avoid Concomitant Use Avoid concomitant use of Clomiphene with any of the following: Osimedize Increased Effect/Toxicity Clomiphene may increase the levels/effects of: Osmedize Decreased Effect Clomiphene may decrease the levels/effects of: Osmedize Hazards Drugs Handling Considerations Hazardous agent (NIOSH 2016 [group 3]). Use appropriate precautions for receiving, handling, administration, and disposal. Gloves (single) should be worn during receiving, unpacking, and placing in storage. NIOSH recommends single use or administration of intact tablets or capsules (NIOSH 2016). Storage/ Stability Store at room temperature of 15°C to 30°C (95°F to 86°F). Protect from light, heat, and excessive humidity. Mechanism of Action Clomiphene is a racemic mixture consisting of zuclophenene (~38%) and enclomiphene (~62%), each with distinct pharmacologic properties. Clomiphene production is the result of the level of the hypothalamic releasing hormone and in the pituitary, causing an increase in the hypothalamic drive to the gonadotropin-producing cells, which leads to increased levels of FSH, LH, and estrogen production. The increased estrogen levels inhibit the production of FSH and LH, leading to ovulation inhibition. This inhibition is characterized by an increase in vascular permeability which causes a fluid shift from intravascular space to third space compartments (eg, peritoneal cavity, thoracic cavity) Clomiphene and Ovulation Stimulator; Selective Estrogen Receptor Modulator (SERM)