Brand Names

HEMANGEOL, Inderal, Inderal LA, Inderal XL, InnoPran XL

Indication Specific Dosing

NOTE: Similar clinical efficacy (e.g., exercise tolerance, chest pain, blood pressure or heart rate control) are seen with equivalent daily doses of sustained-release propranolol (Inderal LA) compared to regular-release propranolol tablets (given in divided doses).

For the treatment of chronic stable angina

Oral dosage (immediate-release formulations)

Adults

Initially, 10 to 20 mg PO 2 to 4 times per day, then increase at 3 to 7 day intervals up to 160 to 320 mg/day, given in 2 to 4 divided doses. In geriatric patients, begin with conservative initial doses and titrate carefully; the elderly have unpredictable responses to beta-blockers.

Oral dosage (extended-release capsules)

Adults

80 mg PO once daily, then increase at 3 to 7 day intervals up to 160 to 320 mg PO once daily. In geriatric patients, begin with conservative initial doses and titrate carefully; the elderly have unpredictable responses to beta-blockers.

For the treatment of unstable angina†

Intravenous dosage

Adults

0.5 to 1 mg IV, followed in 1 to 2 hours by a switch to oral therapy. Per clinical practice guidelines, the intravenous dose can be reserved for high-risk patients and eliminated from the regimen in intermediate- and low-risk patients. In

geriatric patients, use conservative dose; the elderly have unpredictable responses to beta-blockers.

Oral dosage (immediate-release formulations)

Adults

40 to 80 mg PO every 6 to 8 hours; begin 1 to 2 hours after initial IV therapy. Per clinical practice guidelines, the intravenous dose can be reserved for high-risk patients and eliminated from the regimen in intermediate- and low-risk patients. In geriatric patients, begin with conservative initial doses and titrate carefully; the elderly have unpredictable responses to beta-blockers.

For the treatment of atrial fibrillation and/or atrial flutter

Intravenous dosage

Adults

1 mg IV every 2 minutes as needed for up to 3 doses. The FDA-approved dosage is 1 to 3 mg IV every 2 minutes for 2 doses with further doses given after 4 hours or more. Reserve use for atrial fibrillation or flutter that is unresponsive to standard therapy or when more prolonged control is required. Guidelines recommend IV beta-blockers to slow the ventricular response to atrial fibrillation in the acute setting in the absence of pre-excitation and to slow rapid ventricular response with acute coronary syndrome and no heart failure, hemodynamic instability, or bronchospasm.

Children† and Adolescents†

0.01 mg/kg/dose IV every 6 to 8 hours as needed. May titrate dosage gradually as needed for clinical effect. Max: 0.15 mg/kg/dose or 3 mg/dose, whichever is less.

Infants†

0.01 mg/kg/dose IV every 6 to 8 hours as needed. May titrate dosage gradually as needed for clinical effect. Max: 0.15 mg/kg/dose or 1 mg/dose, whichever is less.

Neonates†

0.01 mg/kg/dose IV every 6 to 8 hours as needed. May titrate dosage gradually as needed for clinical effect. Max: 0.15 mg/kg/dose or 1 mg/dose, whichever is less.

Oral dosage (immediate-release)

Adults

10 to 40 mg PO 3 or 4 times daily. Guidelines recommend the use of beta blockers to control the ventricular rate for patients with paroxysmal, persistent, or permanent atrial fibrillation.

Infants†, Children†, and Adolescents†

0.5 to 1 mg/kg/day PO divided every 6 to 8 hours, initially. Increase the dose by 1 mg/kg/day every 3 to 5 days as needed for clinical effect. Usual dose: 2 to 4 mg/kg/day. Max: 16 mg/kg/day or 60 mg/day, whichever is less. In older adolescents, 10 to 30 mg/dose PO every 6 to 8 hours may be given.

Neonates†

0.5 to 1 mg/kg/day PO divided every 6 to 8 hours, initially. Increase the dose by 1 mg/kg/day every 3 to 5 days as needed for clinical effect. Usual dose: 2 to 4 mg/kg/day. Max: 16 mg/kg/day or 60 mg/day, whichever is less.

For the treatment of supraventricular tachycardia (SVT)

For the treatment of paroxysmal supraventricular tachycardia due to atrioventricular nodal reentrant tachycardia as "pill-in-the-pocket" approach†

Oral dosage (immediate-release)

Adults

80 or 160 mg PO as a single dose at the onset of tachycardia episode, in combination with diltiazem.

For the treatment of supraventricular tachycardia (SVT)

Intravenous dosage

Adults

1 mg IV every 2 minutes as needed for up to 3 doses. The FDA-approved dosage is 1 to 3 mg IV every 2 minutes for 2 doses with further doses given after 4 hours or more.

Children† and Adolescents†

0.01 mg/kg/dose IV every 6 to 8 hours as needed. May titrate dosage gradually as needed for clinical effect. Max: 0.15 mg/kg/dose or 3 mg/dose, whichever is less.

Infants†

0.01 mg/kg/dose IV every 6 to 8 hours as needed. May titrate dosage gradually as needed for clinical effect. Max: 0.15 mg/kg/dose or 1 mg/dose, whichever is less.

Neonates†

0.01 mg/kg/dose IV every 6 to 8 hours as needed. May titrate dosage gradually as needed for clinical effect. Max: 0.15 mg/kg/dose or 1 mg/dose, whichever is less.

Oral dosage (immediate-release)†

Adults

10 mg PO 3 to 4 times daily, initially. Adjust dose as needed based on response. Max: 160 mg/day.

Infants, Children, and Adolescents

0.5 to 1 mg/kg/day PO divided every 6 to 8 hours, initially. Increase the dose by 1 mg/kg/day every 3 to 5 days as needed for clinical effect. Usual dose: 2 to 4 mg/kg/day. Max: 16 mg/kg/day or 60 mg/day, whichever is less. In older adolescents, 10 to 30 mg/dose PO every 6 to 8 hours may be given.

Neonates

0.5 to 1 mg/kg/day PO divided every 6 to 8 hours, initially. Increase the dose by 1 mg/kg/day every 3 to 5 days as needed for clinical effect. Usual dose: 2 to 4 mg/kg/day. Max: 16 mg/kg/day or 60 mg/day, whichever is less.

Oral dosage (extended-release)†

Adults

60 mg PO once daily, initially. Adjust dose as needed based on response. Max: 160 mg/day.

For reduction of cardiovascular mortality in stable patients who have sustained a myocardial infarction

Oral dosage (immediate-release formulations)

Adults

180 to 240 mg/day PO, given in 3 to 4 divided doses starting in the first 24 hours post-MI.

For the treatment of hypertension

Oral dosage (immediate-release)

Adults

40 mg PO twice daily, initially. May increase dose gradually if further control is needed. Usual dose: 80 to 160 mg/day in 2 divided doses. Max: 640 mg/day.

Children† and Adolescents†

0.5 to 2 mg/kg/day PO in 2 to 4 divided doses, initially. May increase dose if further control is needed. Usual dose: 1 to 6 mg/kg/day. Max: 8 mg/kg/day or 640 mg/day.

Infants†

0.25 mg/kg/dose PO every 6 to 8 hours, initially. May increase dose if further control is needed. Max: 3.5 mg/kg/dose; others recommend a maximum of 5 mg/kg/day.

Neonates†

0.25 mg/kg/dose PO every 6 to 8 hours, initially. May increase dose if further control is needed. Max: 3.5 mg/kg/dose; others recommend a maximum of 5 mg/kg/day.

Oral dosage (extended-release excluding InnoPran XL)

Adults

80 mg PO once daily, initially. May increase dose gradually if further control is needed. Usual dose: 80 to 160 mg PO once daily. Max: 640 mg/day.

Oral dosage (InnoPran XL)

Adults

80 mg PO once daily, initially. May increase dose to 120 mg PO once daily if

further control is needed. Max: 120 mg/day.

Intravenous dosage†

Neonates†

0.01 mg/kg/dose (Max: 0.15 mg/kg/dose) IV every 6 to 8 hours as needed.

For the treatment of hypertrophic obstructive cardiomyopathy

Oral dosage (immediate-release)

Adults

20 to 40 mg PO 3 or 4 times daily.

Oral dosage (extended-release)

Adults

80 to 160 mg PO once daily.

For management of pheochromocytoma, including preoperative control of tachycardia before surgery, in conjunction with an alpha-blocker

Oral dosage (immediate-release tablets or oral solution)

Adults

The usual dosage is 60 mg/day PO, given in divided doses for 3 days before surgery, in conjunction with an alpha-blocker. For the management of inoperable tumors, the usual dosage is 30 mg daily in divided doses as adjunctive therapy to alpha-adrenergic blockade. For geriatric patients, begin with low initial doses, followed by careful dosage titration; the elderly have unpredictable responses to beta-blockers.

For migraine prophylaxis

For migraine prophylaxis in pediatric patients†

Oral dosage (immediate-release tablets or oral solution)

Children and Adolescents weighing more than 35 kg

0.6 to 3 mg/kg/day PO in 2 to 3 divided doses. Max: 120 mg/day. Pediatric

patients receiving propranolol are possibly more likely than those receiving placebo to have at least a 50% reduction in headache frequency.

Children and Adolescents weighing 35 kg or less

0.6 to 3 mg/kg/day PO in 2 to 3 divided doses. Max: 60 mg/day. Pediatric patients receiving propranolol are possibly more likely than those receiving placebo to have at least a 50% reduction in headache frequency.

For migraine prophylaxis in adults

Oral dosage (immediate-release tablets or oral solution)

Adults

80 mg/day PO in divided doses, initially. May increase the dose gradually as needed. Usual dose: 160 to 240 mg/day in divided doses. Discontinue if adequate results not achieved within 4 to 6 weeks. Guidelines classify propranolol as having established efficacy for migraine prophylaxis.

Oral dosage (extended-release capsules)

Adults

80 mg PO once daily, initially. May increase the dose gradually as needed. Usual dose: 160 to 240 mg/day. Discontinue if adequate results not achieved within 4 to 6 weeks. Guidelines classify propranolol as having established efficacy for migraine prophylaxis.

For the management of tremor

For the management of essential tremor

Oral dosage (immediate-release tablets or oral solution)

Adults

40 mg PO twice daily. Increase dose as needed to 120 to 320 mg/day PO given in 2 to 3 divided doses. In geriatric patients, begin with conservative initial doses and titrate carefully; geriatric patients have unpredictable responses to beta-blockers. Clinical practice guidelines consider propranolol effective for the treatment of essential tremor.

For the management of lithium-induced tremor†

Oral dosage

Adults

Limited data suggest 30 to 80 mg/day PO may be effective; the daily dose is divided into 3 or 4 doses for administration. A common starting dose is 10 mg PO 3 times daily. In a single-blind crossover comparison of propranolol and placebo in 10 patients with lithium-induced tremor, propranolol (30 to 80 mg/day PO) and placebo were administered during two 2-week periods, 1 week on propranolol and 1 on placebo in random order. In period 1, 8 patients reported a preference for propranolol over placebo and 5 patients in period 2 reported a preference for propranolol. Treatment with propranolol resulted in a reduction in the intensity of tremor from very troublesome or somewhat troublesome to noticeable but not troublesome or not present. No adverse reactions were reported with propranolol treatment. In a case report of 5 patients with lithium-induced tremor, treatment with propranolol 30 to 40 mg/day PO, in 3 or 4 divided doses, resulted in control of the tremor. Recurrence of the tremor was reported in 3 of the cases when propranolol therapy was discontinued. In geriatric patients, begin with conservative initial doses and titrate carefully; geriatric patients have unpredictable responses to beta-blockers.

For the management of essential tremor in pediatric patients

Oral dosage (immediate-release formulations)

Adolescents†

Limited experience; dosage often not reported in the literature; efficacy rate of 50%, along with side effect profile may lead to pursuit of other treatment options. 0.5 to 1 mg/kg/day PO, given in 3 divided doses has been recommended by some experts as an initial dose. Titrate dosage gradually once weekly. Alternatively, 30 mg PO once daily, then increased to 30 mg PO twice daily has been effective in improving hand tremor. Many patients respond to a total daily dosage of 60 to 80 mg/day PO. Max: 4 mg/kg/day PO. Dosage may also be taken as needed 30 minutes prior to activities disrupted by essential tremor. Pharmacotherapy should be reserved for patients whose tremor is functionally or socially limiting. Once an optimal dosage is determined, patients may transition to an extended-release formulation of propranolol, to be given once daily. Many patients require larger doses after 1 year of therapy, due to drug tolerance and disease progression.

Children†

Limited experience; dosage often not reported in the literature; efficacy rate of 50%, along with side effect profile may lead to pursuit of other treatment options. 0.5 to 1 mg/kg/day PO, given in 3 divided doses has been recommended by some experts as an initial dose using immediate release dose forms. Titrate dosage gradually once weekly as necessary; many patients respond to a total daily dosage of 60 to 80 mg/day PO. Max: 4 mg/kg/day PO. Dosage may also be taken as needed 30 minutes prior to activities disrupted by essential tremor. Pharmacotherapy should be reserved for patients whose tremor is functionally or socially limiting; most do not require therapy until adolescence. Once an optimal dosage is determined, patients may transition to an extended-release formulation of propranolol, to be given once daily. Many patients require larger doses after 1 year of therapy, due to drug tolerance and disease progression.

For the treatment of anxiety† or panic attacks†

Oral dosage (immediate-release tablets or oral solution)

Adults

10 to 80 mg PO, given 1 hour prior to the anxiety-producing event. For geriatric patients, begin with low initial doses, followed by careful dosage titration; geriatric patients have unpredictable responses to beta-blockers.

For the treatment of thyrotoxicosis† and thyroid storm†

For the treatment of thyroid storm†

Oral dosage (immediate-release)

Adults

60 to 80 mg PO every 4 hours.

Adolescents

10 to 40 mg PO every 6 to 8 hours.

Children

2 mg/kg/day PO divided every 6 to 12 hours (Max: 40 mg/dose). Higher doses may be required. Doses of 20 to 40 mg PO every 6 to 8 hours have been used in older children.

Infants

1 to 2 mg/kg/day PO divided every 6 to 12 hours. Higher doses may be required.

Neonates

1 to 2 mg/kg/day PO divided every 6 to 12 hours. Higher doses may be required.

Intravenous dosage

Adults

1 to 2 mg IV every 15 minutes up to 10 mg.

Children and Adolescents

1 to 3 mg IV as a single dose.

For the treatment thyrotoxicosis†

Oral dosage (immediate-release)

Adults

10 to 40 mg PO every 6 to 8 hours.

Adolescents

10 to 40 mg PO every 6 to 8 hours.

Children

2 mg/kg/day PO divided every 6 to 12 hours (Max: 40 mg/dose). Higher doses may be required.

Infants

1 to 2 mg/kg/day PO divided every 6 to 12 hours. Higher doses may be required.

Neonates

1 to 2 mg/kg/day PO divided every 6 to 12 hours. Higher doses may be required.

Intravenous dosage

Children and Adolescents

1 to 3 mg IV as a single dose.

For the treatment of hypertension and the subsequent decline in renal function associated with scleroderma renal crisis (SRC)†

Oral dosage (immediate-release tablets or oral solution)

Adults

Initially, 40 mg PO twice daily, then increase at 3 to 7 day intervals up to 160 to 480 mg/day PO to attain desired blood pressure response. For geriatric patients, begin with low initial doses, followed by careful dosage titration; geriatric patients have unpredictable responses to beta-blockers.

For variceal bleeding prophylaxis† or the treatment of portal hypertension†

Oral dosage (immediate-release)

Adults

20 to 40 mg PO twice daily, initially. Increase the dose every 2 to 3 days until resting heart rate of 55 to 60 beats per minute with systolic blood pressure of 90 mmHg or more. Max: 320 mg/day in persons without ascites; 160 mg/day in persons with ascites.

For the treatment of chronic agitation† or aggressive behavior

Oral dosage (immediate-release tablets or oral solution)

Adults

Most of the literature describing positive outcomes in the treatment of chronic aggression with propranolol involved patients with co-existing organic brain disease or schizophrenia recalcitrant to other aggression modalities. For patients without preexisting cardiovascular disorders, some authors have suggested a beginning dose of 20 mg PO 3 times per day, increasing the total dose by 40 to 60 mg/day every 3 days. Mean dosages range from 160 to 320 mg/day. For geriatric patients, begin with low initial doses, followed by careful dosage titration; geriatric patients have unpredictable responses to beta-blockers.

For the prevention and treatment of hypercyanotic episodes associated with tetralogy of Fallot (i.e., tetralogy spells†)

Oral dosage (immediate-release)

Infants and Children

1 mg/kg/day PO divided every 6 hours, initially. After 1 week, may titrate dose by 1 mg/kg/day every 24 hours as necessary. Average dose: 2.3 mg/kg/day (range: 0.8 to 5 mg/kg/day). Usual Max: 5 mg/kg/day. If the patient becomes refractory after initial control, may increase dose gradually to a maximum of 10 to 15 mg/kg/day; monitor heart size, heart rate, and cardiac contractility closely. Alternatively, 4 mg/kg/day PO divided every 6 hours has been used as an initial dose.

Intravenous dosage

Infants and Children

0.15 to 0.25 mg/kg/dose (Max: 1 mg/dose) IV; may repeat once. Alternatively, 0.01 to 0.02 mg/kg/dose IV has been used, reserving higher doses for refractory spells.

For the treatment of infantile hemangioma

Oral dosage (outpatient initiation)

Infants

0.5 to 1 mg/kg/day PO in 2 to 3 divided doses, initially. May increase the dose by 0.5 mg/kg/day every 3 to 7 days based on clinical response and tolerability. Target dose: 2 to 3 mg/kg/day. Continue treatment for at least 6 months and up to 12 months of age. When discontinuing therapy, consider tapering dose over 2 weeks. The FDA-approved dosage is 0.6 mg/kg/dose PO twice daily, then 1.1 mg/kg/dose PO twice daily after 1 week, and then 1.7 mg/kg/dose PO twice daily after 1 week. Adjust dose as needed based on weight increase. Continue treatment for 6 months. If hemangiomas recur, treatment may be reinitiated.

Oral dosage (inpatient initiation)

Infants

0.33 mg/kg/dose PO every 8 hours, initially. May increase the dose to 0.66 mg/kg/dose PO every 8 hours based on clinical response and tolerability. Alternatively, 0.25 mg/kg/dose PO twice daily, initially. May increase the dose by 0.5 mg/kg/dose every 24 hours based on clinical response and tolerability. Target

dose: 2 to 3 mg/kg/day. Continue treatment for at least 6 months and up to 12 months of age. The FDA-approved dosage is 0.6 mg/kg/dose PO twice daily, then 1.1 mg/kg/dose PO twice daily after 1 week, and then 1.7 mg/kg/dose PO twice daily after 1 week. Adjust dose as needed based on weight increase. Continue treatment for 6 months. If hemangiomas recur, treatment may be reinitiated.

Neonates†

0.33 mg/kg/dose PO every 8 hours, initially. May increase the dose to 0.66 mg/kg/dose PO every 8 hours based on clinical response and tolerability. Alternatively, 0.25 mg/kg/dose PO twice daily, initially. May increase the dose by 0.5 mg/kg/dose every 24 hours based on clinical response and tolerability. Target dose: 2 to 3 mg/kg/day. Continue treatment for at least 6 months and up to 12 months of age.

For the attenuation of hypermetabolism in patients with severe burns†

For the attenuation of hypermetabolism in adult patients with severe burns

Oral dosage (immediate-release formulations)

Adults

1 mg/kg/day PO, given in divided doses every 4 hours. Adjust dose as needed to achieve a target 20% reduction in heart rate from baseline to a maximum dose of 1.98 mg/kg/day. Median dose: 80 mg/day.

For the attenuation of hypermetabolism in pediatric patients with severe burns

Oral dosage (immediate-release formulations)

Infants, Children, and Adolescents

1 to 4 mg/kg/day PO, given in divided doses every 6 hours. Adjust dose as needed to decrease heart rate by 10% to 20% of the admission value or mean age-based population value. 4 mg/kg/day PO was the mean effective dose in an interim analysis of children (n = 90; mean age 7 +/- 5 years) with more than 30% total body surface area burns. Propranolol therapy began 96 hours postburn and continued for 1 year with few adverse effects. Propranolol therapy significantly reduced heart rate and resting energy expenditure, decreased truncal fat accumulation, prevented bone loss, and improved lean body mass accretion.

Maximum dose not clearly defined; severely burned adult patients standardly receive 20 mg PO every 6 hours, with dosage titrated as needed.

For the treatment of heart failure† (ischemic origin or cardiomyopathy†) usually in conjunction with digoxin, diuretics, or ACE inhibitor therapy in children and infants

Oral dosage (immediate-release formulations)

Infants and Children

Initially, 0.5 to 1 mg/kg/day PO, given in divided doses every 6 to 8 hours has been recommended for sympathetic inhibition. Titrate dosage gradually every 3 to 14 days to a target dose of 2 mg/kg/day PO (range: 1.5 to 3 mg/kg/day). Monitor heart rate and blood pressure.

For the treatment of premature ventricular contractions (PVCs)†

Oral dosage (immediate-release)

Adults

10 to 40 mg PO every 6 hours.

Oral dosage (extended-release)

Adults

60 to 160 mg PO every 12 hours.

Intravenous dosage

Adults

1 to 3 mg IV every 5 minutes as needed up to a total of 5 mg.

For the treatment of catecholaminergic polymorphic ventricular tachycardia†

Oral dosage (immediate-release)

Adults

10 to 80 mg PO 4 times daily.

Children and Adolescents

0.5 to 1 mg/kg/day PO in 3 to 4 divided doses, initially. May increase the dose every 3 to 5 days as needed based on clinical response and tolerability. Usual dose: 2 to 4 mg/kg/day. Max: 16 mg/kg/day or 60 mg/day.

Oral dosage (extended-release)

Adults

80 to 320 mg/day PO in 1 or 2 divided doses.

Contraindications And Precaution

Drug Interactions

The coadministration of certain medications may lead to harm and require avoidance or therapy modification; review all drug interactions prior to concomitant use of other medications.

Hypersensitivity

This medication is contraindicated in patients with a history of hypersensitivity to it or any of its components.

General Information

Abrupt discontinuation of a beta-blocker in individuals with coronary artery disease may result in the exacerbation of angina, and in some cases, myocardial infarction or ventricular arrhythmia has been reported. When discontinuing chronically administered oral propranolol, gradually reduce the dose over at least a few weeks and monitor closely. If angina significantly worsens, promptly restart propranolol and take other measures appropriate for the management of unstable angina.

atopy

While taking beta-blockers, individuals with a history of sensitivity to multiple allergens (atopy) may be more reactive to repeated challenge, either accidental, diagnostic, or therapeutic. Such individuals may be unresponsive to the usual doses of epinephrine used to treat allergic reaction.

PHACE syndrome

Propranolol may increase the risk of stroke in individuals with PHACE syndrome (posterior fossa malformations, hemangioma, arterial anomalies, coarctation of the aorta/cardiac defects, and eye abnormalities) with severe cerebrovascular anomalies by

decreasing blood pressure. Investigate individuals with large facial hemangiomas for potential arteriopathy associated with PHACE syndrome prior to propranolol therapy.

acute heart failure, bradycardia, cardiac conduction disorder, cardiogenic shock, heart failure, hypotension, second or third-degree AV block, sick sinus syndrome

Propranolol is contraindicated in individuals with acute heart failure, cardiogenic shock, sinus bradycardia, sick sinus syndrome, and second or third-degree AV block. Propranolol oral solution for infantile hemangioma is also contraindicated in individuals with bradycardia or hypotension defined as a heart rate less than 80 beats per minute or blood pressure less than 50/30 mmHg. Guidelines include cardiogenic shock or heart failure, sinus bradycardia, or heart block greater than first degree as potential exclusions for the use of propranolol for infantile hemangioma that require appropriate subspecialty evaluation and clearance. Individuals with first-degree AV block, sinus node dysfunction, or a cardiac conduction disorder (including Wolff-Parkinson-White syndrome) may be at increased risk for bradycardia with propranolol. Sympathetic stimulation may be essential for support of the circulation in individuals with diminished myocardial contractility, and its inhibition by beta-adrenergic receptor blockade may precipitate more severe failure. Although beta-blockers should be avoided in acute heart failure, some have been shown to be highly beneficial when used with close follow-up in individuals with a history of heart failure who are well compensated and are receiving guideline-directed medical therapy (GDMT). In individuals without a history of heart failure, continued depression of the myocardium with beta-blocking agents over a period of time may lead to cardiac failure.

peripheral vascular disease

Beta-blockers can precipitate or aggravate symptoms of arterial insufficiency in individuals with peripheral vascular disease.

acute lower respiratory tract infection, asthma, bronchospasm, chronic obstructive pulmonary disease

Propranolol is contraindicated in individuals with asthma. Propranolol oral solution for infantile hemangioma is also contraindicated in individuals with a history of bronchospasm. Interrupt propranolol therapy in the event of an acute lower respiratory tract infection associated with dyspnea and wheezing when used for the treatment of infantile hemangioma. In general, do not use beta-blockers in individuals with bronchospastic pulmonary disease (e.g., asthma, bronchospasm, chronic obstructive pulmonary disease). Use propranolol with caution in this setting since it may provoke a

bronchial asthmatic attack by blocking bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta-receptors.

hepatic failure

Use propranolol with caution and monitor for marked bradycardia and hypotension in individuals with hepatic impairment or hepatic failure. Consider lower propranolol doses in individuals with hepatic insufficiency. Propranolol is extensively metabolized by the liver. Compared to normal subjects, individuals with chronic liver disease have decreased clearance of propranolol, increased volume of distribution, decreased protein-binding and considerable variation in half life.

renal failure, renal impairment

Use propranolol with caution and monitor for marked bradycardia and hypotension in individuals with renal impairment or renal failure. Consider lower propranolol doses in individuals with renal impairment. Propranolol plasma clearance may be reduced in individuals with chronic renal failure. Additionally, chronic renal failure has been associated with a decrease in propranolol metabolism via down-regulation of hepatic cytochrome P450 activity.

myasthenia gravis

Beta-blockers may cause exacerbation of myasthenia gravis, including ocular myasthenia gravis. Use the lowest effective propranolol dose in individuals with myasthenia gravis and monitor for exacerbation or deterioration of myasthenia gravis.

psoriasis

Beta-blockers may exacerbate psoriasis.

hyperthyroidism

Beta-blockade may mask certain clinical signs of hyperthyroidism (i.e., tachycardia). Abrupt withdrawal of beta-blockade might precipitate a thyroid storm; therefore, closely monitor individuals suspected of developing hyperthyroidism if withdrawing beta-blocker therapy.

pheochromocytoma

Propranolol oral solution for infantile hemangioma is contraindicated in individuals with pheochromocytoma. Beta-blocker use in the absence of an alpha-1 antagonist in

individuals with pheochromocytoma is not recommended because of the potential for hypertensive crisis due to unopposed stimulation of alpha adrenergic receptors and subsequent significant peripheral vasoconstriction.

major surgery, planned procedure

Do not routinely withdraw chronic beta-blocker therapy before major surgery; however, the impaired ability of the heart to respond to reflex adrenergic stimuli may increase the risk of surgical procedures and general anesthesia. Evaluate the risks vs. benefits in individuals by considering the type of surgery (e.g., cardiac vs. noncardiac), coexisting health conditions, and anesthetic strategy. Guidelines recommend continuance in individuals already on beta-blocker therapy; however, it is recommended to initiate a beta-blocker well before a planned procedure with careful perioperative titration to achieve adequate heart rate control while avoiding significant bradycardia or hypotension.

activities requiring coordination and concentration, driving or operating machinery

Advise individuals to avoid driving or operating machinery or participating in activities requiring coordination and concentration until they know how they react to beta-blocker therapy.

adolescents, children, diabetes mellitus, fasting, infants, neonates, premature neonates, vomiting

Propranolol oral solution for hemangioma is contraindicated in premature neonates, neonates, and infants with a corrected age younger than 5 weeks or a body weight less than 2 kg. Guidelines advise caution rather than exclusion in infants younger than 5 weeks and/or with a postconceptual age younger than 48 weeks. Neonates and infants are particularly sensitive to the negative inotropic and chronotropic effects of propranolol, especially when administered intravenously. Beta-blockers may inhibit catecholamine-induced glycogenolysis, gluconeogenesis, and lipolysis, predisposing to hypoglycemia. Additionally, beta-blockers may mask certain signs of hypoglycemia (e.g., tachycardia) and increase the risk for severe or prolonged hypoglycemia at any time during treatment, especially in individuals with diabetes mellitus, neonates, infants, children, adolescents, and individuals who are fasting (i.e., surgery, not eating regularly) or vomiting. Instruct individuals and their caregivers to seek immediate medical treatment if severe hypoglycemia occurs. Beta-blockers may also inhibit insulin secretion through blockade of beta-2 receptors on pancreatic islet cells, which may cause hyperglycemia or reduce insulin secretion in response to hyperglycemia; adjust

the dose of antidiabetic medications as necessary. In addition to acute blood glucose effects, beta-blockers have been shown to increase the risk of developing diabetes mellitus in hypertensive adults; evaluate the risk relative to the proven benefits of beta-blockers in reducing cardiovascular events.

geriatric

Geriatric adults have decreased clearance and a longer mean elimination half-life of propranolol; dose adjustment of propranolol may be required. In general, use cautious dose selection for propranolol in geriatric adults, usually starting at the low end of the dosing range, reflecting the greater frequency of decreased hepatic, renal, or cardiac function, and of concomitant disease or other drug therapy.

pregnancy

Prolonged experience with the use of propranolol during pregnancy, based on published interventional and observational studies, has not identified a drug-associated risk of major birth defects, miscarriage, or other adverse outcomes. Propranolol crosses the placenta. There are inconsistent reports of intrauterine growth restriction associated with beta-blocker use, including propranolol. However, hypertension also increases the fetal risk for intrauterine growth restriction.

people who can cause pregnancy in others, reproductive risk

Counsel people who can cause pregnancy in others about the reproductive risk associated with propranolol. In animal studies, propranolol has been shown to inhibit spermatogenesis; it is not known whether propranolol affects human fertility. Additionally, beta-blockers may cause impotence.

neonates and infants exposed to this medication in utero

Neonates and infants exposed to this medication in utero during the third trimester may be at increased risk for hypoglycemia, bradycardia, hypotension, and respiratory depression. Monitor blood glucose and cardiorespiratory parameters closely in exposed newborns to facilitate early recognition and prompt management of potential effects.

breast-feeding

Use propranolol with caution during breast-feeding. Propranolol is present in human milk at low concentrations. The amount ingested by the child is minimal and unlikely to cause adverse effects. Adverse effects were not reported in multiple studies in which children were exposed to propranolol through breast milk. Of the beta-blockers,

propranolol is a preferred agent during lactation. Alternatives to consider include labetalol or metoprolol.

Pregnancy And Lactation

Prolonged experience with the use of propranolol during pregnancy, based on published interventional and observational studies, has not identified a drug-associated risk of major birth defects, miscarriage, or other adverse outcomes. Propranolol crosses the placenta. There are inconsistent reports of intrauterine growth restriction associated with beta-blocker use, including propranolol. However, hypertension also increases the fetal risk for intrauterine growth restriction.

Interactions

Abiraterone: (Moderate) Monitor blood pressure and heart rate if coadministration of propranolol with abiraterone is necessary. Propranolol is a CYP2D6 substrate and abiraterone is a CYP2D6 inhibitor. Concomitant use may result in hypotension and bradycardia.
Acarbose: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Acetaminophen; Aspirin, ASA; Caffeine: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Acetaminophen; Aspirin: (Moderate) Concurrent use of beta-blockers with aspirin and

other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Acetaminophen; Aspirin; diphenhydrAMINE: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Acetaminophen; Chlorpheniramine; Dextromethorphan; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Acetaminophen; Chlorpheniramine; Dextromethorphan; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Acetaminophen; Chlorpheniramine; Phenylephrine : (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Acetaminophen; Dextromethorphan; guaiFENesin; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Acetaminophen; Dextromethorphan; guaiFENesin; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Acetaminophen; Dextromethorphan; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Acetaminophen; Dextromethorphan; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Acetaminophen; guaiFENesin; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Acetaminophen; Ibuprofen: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive

effect of beta-blockers may be diminished by NSAIDs.
Acetaminophen; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Acetaminophen; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Aclidinium; Formoterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Adagrasib: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of adagrasib as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and adagrasib is moderate CYP2D6 inhibitor.
Adenosine: (Moderate) Use adenosine with caution in the presence of beta blockers due to the potential for additive or synergistic depressant effects on the sinoatrial and atrioventricular nodes.
Albuterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Albuterol; Budesonide: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's

lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used. (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Alemtuzumab: (Moderate) Alemtuzumab may cause hypotension. Careful monitoring of blood pressure and hypotensive symptoms is recommended especially in patients with ischemic heart disease and in patients on antihypertensive agents.
ALFentanil: (Moderate) Alfentanil may cause bradycardia. The risk of significant hypotension and/or bradycardia during therapy with alfentanil is increased in patients receiving beta-blockers.
Alfuzosin: (Moderate) The manufacturer warns that the combination of alfuzosin with antihypertensive agents has the potential to cause hypotension in some patients. Alfuzosin (2.5 mg, immediate-release) potentiated the hypotensive effects of atenolol (100 mg) in eight healthy young male volunteers. The Cmax and AUC of alfuzosin was increased by 28% and 21%, respectively. Alfuzosin increased the Cmax and AUC of atenolol by 26% and 14%, respectively. Significant reductions in mean blood pressure and in mean heart rate were reported with the combination.
Allergen Immunotherapy (selected): (Moderate) Advise patients who are undergoing allergy testing or receiving maintenance allergen immunotherapy (AIT) that allergic reactions, including symptoms of anaphylaxis, may be more severe while receiving beta-blocker therapy. Risk varies by individual based on indication for therapy and history. This risk is generally considered minimal for most patients who are receiving maintenance AIT and the benefits of continuing beta-blocker therapy typically outweigh the risks in patients with cardiovascular disease.
Alogliptin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients

with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Alogliptin; metFORMIN: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Alogliptin; Pioglitazone: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Alpha-blockers: (Moderate) Orthostatic hypotension may be more likely in some patients if beta-blockers are coadministered with alpha-blockers. Monitor blood pressure and for orthostasis, especially at medication initiation.
Alpha-glucosidase Inhibitors: (Moderate) Increased frequency of blood glucose

monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.

Alprostadil: (Minor) The concomitant use of systemic alprostadil injection and antihypertensive agents, such as beta-clockers, may cause additive hypotension. Caution is advised with this combination. Systemic drug interactions with the urethral suppository (MUSE) or alprostadil intracavernous injection are unlikely in most patients because low or undetectable amounts of the drug are found in the peripheral venous circulation following administration. In those men with significant corpora cavernosa venous leakage, hypotension might be more likely. Use caution with in-clinic dosing for erectile dysfunction (ED) and monitor for the effects on blood pressure. In addition, the presence of medications in the circulation that attenuate erectile function may influence the response to alprostadil. However, in clinical trials with alprostadil intracavernous injection, anti-hypertensive agents had no apparent effect on the safety and efficacy of alprostadil.

Aluminum Hydroxide: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.

Aluminum Hydroxide; Magnesium Carbonate: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.

Aluminum Hydroxide; Magnesium Hydroxide: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.

Aluminum Hydroxide; Magnesium Hydroxide; Simethicone: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Aluminum Hydroxide; Magnesium Trisilicate: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Amifostine: (Major) Patients receiving beta-blockers should be closely monitored during amifostine infusions due to additive effects. Patients receiving amifostine at doses recommended for chemotherapy should have antihypertensive therapy interrupted 24 hours preceding administration of amifostine. If the antihypertensive cannot be stopped, patients should not receive amifostine.
Amiodarone: (Moderate) Concomitant administration of propranolol with amiodarone may cause additive electrophysiologic effects (slow sinus rate or worsen AV block), resulting in symptomatic bradycardia, sinus arrest, and atrioventricular block. This is particularly likely in patients with preexisting partial AV block or sinus node dysfunction. Because amiodarone is an inhibitor of CYP2D6, decreased clearance of propranolol, which is a CYP2D6 substrate, is also possible. Caution and close monitoring are recommended during coadministration; a dose reduction of one or both drugs may be needed based on response. It should be noted that post-hoc analysis of amiodarone therapy in patients after acute myocardial infarction in two clinical trials revealed that amiodarone in addition to a beta-blocker significantly lowered the incidence of cardiac and arrhythmic death or resuscitated cardiac arrest when compared with amiodarone or beta-blocker therapy alone.
amLODIPine: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
amLODIPine; Atorvastatin: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
amLODIPine; Benazepril: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
amLODIPine; Celecoxib: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially

in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
(Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
amLODIPine; Olmesartan: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
amLODIPine; Valsartan: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
amLODIPine; Valsartan; hydroCHLOROthiazide, HCTZ: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
Amobarbital: (Moderate) Although concurrent use of amobarbital with antihypertensive agents may lead to hypotension, barbiturates, as a class, can enhance the hepatic metabolism of beta-blockers that are significantly metabolized by the liver. Beta-blockers that may be affected include betaxolol, labetalol, metoprolol, pindolol, propranolol, and timolol. Clinicians should closely monitor patients blood pressure during times of coadministration.
Amphetamine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Amphetamine; Dextroamphetamine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Antacids: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Antithyroid agents: (Minor) Hyperthyroidism may cause increased clearance of beta blockers that possess a high extraction ratio. A dose reduction of some beta-blockers may be needed when a hyperthyroid patient treated with methimazole becomes euthyroid.
Apalutamide: (Moderate) Monitor for decreased efficacy of propranolol if coadministration with apalutamide is necessary. Propranolol is a CYP2C19 substrate and

apalutamide is a strong CYP2C19 inducer.
Apomorphine: (Moderate) Use of beta blockers and apomorphine together can increase the hypotensive effects of apomorphine. Monitor blood pressure regularly during use of this combination.
Apraclonidine: (Minor) Theoretically, additive blood pressure reductions could occur when apraclonidine is combined with antihypertensive agents.
Arformoterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
ARIPiprazole: (Minor) Aripiprazole may enhance the hypotensive effects of antihypertensive agents. It may be advisable to monitor blood pressure when these medications are coadministered.
Armodafinil: (Moderate) In vitro data indicate that armodafinil is an inhibitor of CYP2C19. In theory, dosage reductions may be required for drugs that are largely eliminated via CYP2C19 metabolism such as propranolol during coadministration with armodafinil.
Artemether; Lumefantrine: (Moderate) Lumefantrine is an inhibitor and propranolol is a substrate of the CYP2D6 isoenzyme; therefore, coadministration may lead to increased propranolol concentrations. Concomitant use warrants caution due to the potential for increased side effects.
Articaine; EPINEPHrine: (Moderate) Local anesthetics may cause additive hypotension in combination with antihypertensive agents. Thus, patients receiving antihypertensive agents may experience additive hypotensive effects. (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Ascorbic Acid, Vitamin C: (Minor) Ascorbic acid may reduce the oral bioavailability of propranolol. Advise patients against taking large doses of ascorbic acid with doses of propranolol.
Asenapine: (Moderate) Secondary to alpha-blockade, asenapine can produce vasodilation that may result in additive effects during concurrent use of propranolol. The potential reduction in blood pressure can precipitate orthostatic hypotension and associated dizziness, tachycardia, and syncope. If concurrent use is necessary, patients should be counseled on measures to prevent orthostatic hypotension, such as sitting on the edge of the bed for several minutes prior to standing in the morning and rising

slowly from a seated position. Close monitoring of blood pressure is recommended until the full effects of the combination therapy are known; the propranolol dosage may need to be adjusted.
Aspirin, ASA: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Aspirin, ASA; Butalbital; Caffeine: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Aspirin, ASA; Caffeine: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Aspirin, ASA; Caffeine; Orphenadrine: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Aspirin, ASA; Carisoprodol; Codeine: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Aspirin, ASA; Citric Acid; Sodium Bicarbonate: (Major) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals. (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Aspirin, ASA; Dipyridamole: (Major) Beta-blockers should generally be withheld before dipyridamole-stress testing. Monitor the heart rate carefully following the dipyridamole injection. (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Aspirin, ASA; Omeprazole: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Aspirin, ASA; oxyCODONE: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Atazanavir: (Moderate) Atazanavir can prolong the PR interval. Coadministration with

other agents that prolong the PR interval, like beta blockers, may result in elevated risk of conduction disturbances and atrioventricular block.
Atazanavir; Cobicistat: (Moderate) Atazanavir can prolong the PR interval. Coadministration with other agents that prolong the PR interval, like beta blockers, may result in elevated risk of conduction disturbances and atrioventricular block. (Moderate) Coadministration of cobicistat (a CYP2D6 inhibitor) with beta-blockers metabolized by CYP2D6, such as propranolol, may result in elevated beta-blocker serum concentrations. If used concurrently, close clinical monitoring with appropriate beta-blocker dose reductions are advised.
Atracurium: (Moderate) Concomitant use of neuromuscular blockers and beta-blockers may prolong neuromuscular blockade.
Azelastine; Fluticasone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Baclofen: (Moderate) Baclofen has been associated with hypotension. Concurrent use with baclofen and antihypertensive agents may result in additive hypotension. Dosage adjustments of the antihypertensive medication may be required.
Beclomethasone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Benzgalantamine: (Moderate) The increase in vagal tone induced by cholinesterase inhibitors, such as galantamine, may produce bradycardia or syncope. The vagotonic effect of galantamine may theoretically be increased when given with beta-blockers.
Benzphetamine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Berotralstat: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of berotralstat as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and berotralstat is moderate CYP2D6 inhibitor.
Beta-agonists: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly,

especially when non-cardioselective beta blockers are used.
Betamethasone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Bexagliflozin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Bismuth Subsalicylate: (Moderate) Concurrent use of beta-blockers with bismuth subsalicylate and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Bismuth Subsalicylate; metroNIDAZOLE; Tetracycline: (Moderate) Concurrent use of beta-blockers with bismuth subsalicylate and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Bretylium: (Moderate) Bretylium and beta-blockers may have an additive effect when used concomitantly; monitor for hypotension or marked bradycardia, which may produce vertigo, syncope, or postural hypotension.
Brexpiprazole: (Moderate) Due to brexpiprazole's antagonism at alpha 1-adrenergic receptors, the drug may enhance the hypotensive effects of alpha-blockers and other antihypertensive agents.
Brompheniramine; Dextromethorphan; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Brompheniramine; Phenylephrine: (Moderate) Monitor hemodynamic parameters and

for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Brompheniramine; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Brompheniramine; Pseudoephedrine; Dextromethorphan: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Budesonide: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Budesonide; Formoterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used. (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Budesonide; Glycopyrrolate; Formoterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used. (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
BUPivacaine Liposomal: (Major) Propranolol has been shown to significantly decrease the clearance of the amide local anesthetics (e.g., lidocaine, bupivacaine, and

mepivacaine). Lidocaine and bupivacaine toxicity have been reported after coadministration with propranolol. The mechanism of the interaction between propranolol and lidocaine is thought to be due to propranolol-induced decreased hepatic blood flow causing decreased elimination of lidocaine. Local anesthetics may also cause additive hypotension in combination with antihypertensive agents. Use extreme caution with the concomitant use of bupivacaine and antihypertensive agents or rapid-onset vasodilators, such as nitrates. Peripheral vasodilation may occur after use of bupivacaine. Thus, patients receiving antihypertensive agents may experience additive hypotensive effects. Blood concentrations of local anesthetics achieved after therapeutic doses are associated with minimal change in peripheral vascular resistance. Higher blood concentrations of local anesthetics may occur due to inadvertent intravascular administration or repeated doses.
BUPivacaine: (Major) Propranolol has been shown to significantly decrease the clearance of the amide local anesthetics (e.g., lidocaine, bupivacaine, and mepivacaine). Lidocaine and bupivacaine toxicity have been reported after coadministration with propranolol. The mechanism of the interaction between propranolol and lidocaine is thought to be due to propranolol-induced decreased hepatic blood flow causing decreased elimination of lidocaine. Local anesthetics may also cause additive hypotension in combination with antihypertensive agents. Use extreme caution with the concomitant use of bupivacaine and antihypertensive agents or rapid-onset vasodilators, such as nitrates. Peripheral vasodilation may occur after use of bupivacaine. Thus, patients receiving antihypertensive agents may experience additive hypotensive effects. Blood concentrations of local anesthetics achieved after therapeutic doses are associated with minimal change in peripheral vascular resistance. Higher blood concentrations of local anesthetics may occur due to inadvertent intravascular administration or repeated doses.
BUPivacaine; EPINEPHrine: (Major) Propranolol has been shown to significantly decrease the clearance of the amide local anesthetics (e.g., lidocaine, bupivacaine, and mepivacaine). Lidocaine and bupivacaine toxicity have been reported after coadministration with propranolol. The mechanism of the interaction between propranolol and lidocaine is thought to be due to propranolol-induced decreased hepatic blood flow causing decreased elimination of lidocaine. Local anesthetics may also cause additive hypotension in combination with antihypertensive agents. Use extreme caution with the concomitant use of bupivacaine and antihypertensive agents or rapid-onset vasodilators, such as nitrates. Peripheral vasodilation may occur after use of bupivacaine. Thus, patients receiving antihypertensive agents may experience additive hypotensive effects. Blood concentrations of local anesthetics achieved after therapeutic doses are associated with minimal change in peripheral vascular resistance. Higher blood concentrations of local anesthetics may occur due to inadvertent intravascular administration or repeated doses. (Moderate) Monitor hemodynamic

parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
BUPivacaine; Meloxicam: (Major) Propranolol has been shown to significantly decrease the clearance of the amide local anesthetics (e.g., lidocaine, bupivacaine, and mepivacaine). Lidocaine and bupivacaine toxicity have been reported after coadministration with propranolol. The mechanism of the interaction between propranolol and lidocaine is thought to be due to propranolol-induced decreased hepatic blood flow causing decreased elimination of lidocaine. Local anesthetics may also cause additive hypotension in combination with antihypertensive agents. Use extreme caution with the concomitant use of bupivacaine and antihypertensive agents or rapid-onset vasodilators, such as nitrates. Peripheral vasodilation may occur after use of bupivacaine. Thus, patients receiving antihypertensive agents may experience additive hypotensive effects. Blood concentrations of local anesthetics achieved after therapeutic doses are associated with minimal change in peripheral vascular resistance. Higher blood concentrations of local anesthetics may occur due to inadvertent intravascular administration or repeated doses. (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
buPROPion: (Minor) Monitor for an increased incidence of propranolol-related adverse effects if bupropion and propranolol are used concomitantly. Coadministration of bupropion and propranolol may result in increased plasma concentrations of propranolol. Bupropion and hydroxybupropion, the major active metabolite, are inhibitors of CYP2D6 in vitro. Propranolol is a CYP2D6 substrate.
buPROPion; Naltrexone: (Minor) Monitor for an increased incidence of propranolol-related adverse effects if bupropion and propranolol are used concomitantly. Coadministration of bupropion and propranolol may result in increased plasma concentrations of propranolol. Bupropion and hydroxybupropion, the major active metabolite, are inhibitors of CYP2D6 in vitro. Propranolol is a CYP2D6 substrate.
Butalbital; Aspirin; Caffeine; Codeine: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Cabergoline: (Moderate) Cabergoline should be used cautiously with antihypertensive agents, including beta-blockers. Cabergoline has been associated with hypotension. Initial doses of cabergoline higher than 1 mg may produce orthostatic hypotension. It may be advisable to monitor blood pressure.
Calcium Carbonate: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic

goals.
Calcium Carbonate; Famotidine; Magnesium Hydroxide: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Calcium Carbonate; Magnesium Hydroxide: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Calcium Carbonate; Magnesium Hydroxide; Simethicone: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Calcium Carbonate; Simethicone: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Calcium; Vitamin D: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Canagliflozin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Canagliflozin; metFORMIN: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta

blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Cannabidiol: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of cannabidiol as concurrent use may increase propranolol exposure. Propranolol is a CYP2C19 substrate and cannabidiol is a moderate CYP2C19 inhibitor.
Capivasertib: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of capivasertib as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and capivasertib is moderate CYP2D6 inhibitor.
Capmatinib: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of capmatinib as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and capmatinib is moderate CYP1A2 inhibitor.
Carbidopa; Levodopa: (Moderate) Monitor for postural hypotension if concomitant use of levodopa and an antihypertensive, such as a beta-blocker, is necessary. A beta-blocker dosage reduction may be required. Both medications lower blood pressure and this effect may be additive with concomitant use.
Carbidopa; Levodopa; Entacapone: (Moderate) Monitor for postural hypotension if concomitant use of levodopa and an antihypertensive, such as a beta-blocker, is necessary. A beta-blocker dosage reduction may be required. Both medications lower blood pressure and this effect may be additive with concomitant use.
Cariprazine: (Moderate) Orthostatic vital signs should be monitored in patients who are at risk for hypotension, such as those receiving cariprazine in combination with antihypertensive agents. Atypical antipsychotics may cause orthostatic hypotension and syncope, most commonly during treatment initiation and dosage increases. Patients should be informed about measures to prevent orthostatic hypotension, such as sitting

on the edge of the bed for several minutes prior to standing in the morning, or rising slowly from a seated position. Consider a cariprazine dose reduction if hypotension occurs.
Celecoxib: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Celecoxib; Tramadol: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Cenobamate: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of cenobamate as concurrent use may increase propranolol exposure. Propranolol is a CYP2C19 substrate and cenobamate is a moderate CYP2C19 inhibitor.
Ceritinib: (Major) Avoid concomitant use of ceritinib with propranolol if possible due to the risk of additive bradycardia. Both ceritinib and propranolol can cause bradycardia. An interruption of ceritinib therapy, dose reduction, or discontinuation of therapy may be necessary if bradycardia occurs.
Cetirizine; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Cevimeline: (Major) Cevimeline should be administered with caution to patients taking beta adrenergic antagonists, because of the possibility of conduction disturbances. Cevimeline can potentially alter cardiac conduction and/or heart rate. Patients with significant cardiovascular disease treated with beta-blockers may potentially be unable to compensate for transient changes in hemodynamics or rhythm induced by cevimeline. If use of these drugs together cannot be avoided, close monitoring of blood pressure, heart rate and cardiac function is advised.
Chlophedianol; Dexchlorpheniramine; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Chloroprocaine: (Moderate) Local anesthetics may cause additive hypotension in combination with antihypertensive agents.
Chlorpheniramine; Dextromethorphan; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Chlorpheniramine; Dextromethorphan; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic

agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Chlorpheniramine; Ibuprofen; Pseudoephedrine: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs. (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Chlorpheniramine; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Chlorpheniramine; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
chlorproMAZINE: (Moderate) Monitor for an increase in chlorpromazine and propranolol-related adverse effects during concomitant use. The concentrations of both medications may increase; concomitant use has been observed to increase propranolol concentrations by 70%,
Cholestyramine: (Moderate) Absorption of propranolol may be reduced by concurrent administration with colestipol or cholestyramine. To minimize drug interactions, administer other drugs at least 1 hour before or at least 4 to 6 hours after the administration of cholestyramine.
Choline Salicylate; Magnesium Salicylate: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Ciclesonide: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Cimetidine: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of cimetidine as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and cimetidine is weak CYP2D6 inhibitor.
Cinacalcet: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of cinacalcet as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and cinacalcet is moderate CYP2D6 inhibitor.
Cisatracurium: (Moderate) Concomitant use of neuromuscular blockers and beta-

blockers may prolong neuromuscular blockade.
Citalopram: (Minor) Citalopram mildly inhibits the hepatic CYP2D6 isoenzyme at therapeutic doses. This can result in increased concentrations of drugs metabolized via the same pathway, including propranolol. Increased serum levels of the beta-blockers could result in alterations in cardioselectivity or other clinical effects.
Clevidipine: (Moderate) Use clevidipine and propranolol with caution due to risk for additive negative effects on heart rate, AV conduction, and/or cardiac contractility.
cloBAZam: (Moderate) A dosage reduction of CYP2D6 substrates, such as propranolol, may be necessary during co-administration of clobazam. Limited in vivo data suggest that clobazam is an inhibitor of CYP2D6. If propranolol is used in combination, it is advisable to monitor the patient for adverse reactions related to beta-blockers.
cloNIDine: (Moderate) Monitor heart rate in patients receiving concomitant clonidine and agents known to affect sinus node function or AV nodal conduction (e.g., beta-blockers). Severe bradycardia resulting in hospitalization and pacemaker insertion has been reported during combination therapy with clonidine and other sympatholytic agents. Concomitant use of clonidine with beta-blockers can also cause additive hypotension. Beta-blockers should not be substituted for clonidine when modifications are made in a patient's antihypertensive regimen because beta-blocker administration during clonidine withdrawal can augment clonidine withdrawal, which may lead to a hypertensive crisis. If a beta-blocker is to be substituted for clonidine, clonidine should be gradually tapered and the beta-blocker should be gradually increased over several days to avoid the possibility of rebound hypertension; administration of beta-blockers during withdrawal of clonidine can precipitate severe increases in blood pressure as a result of unopposed alpha stimulation.
cloZAPine: (Moderate) Clozapine used concomitantly with the antihypertensive agents can increase the risk and severity of hypotension by potentiating the effect of the antihypertensive drug.
Cobicistat: (Moderate) Coadministration of cobicistat (a CYP2D6 inhibitor) with beta-blockers metabolized by CYP2D6, such as propranolol, may result in elevated beta-blocker serum concentrations. If used concurrently, close clinical monitoring with appropriate beta-blocker dose reductions are advised.
Cocaine: (Major) Although beta-blockers are indicated to reduce cocaine-induced tachycardia, myocardial ischemia, and arrhythmias, concomitant use of cocaine and non-selective beta-adrenergic blocking agents, including ophthalmic preparations, can cause unopposed alpha-adrenergic activity, resulting in heart block, excessive bradycardia, or hypertension. In theory, the use of alpha-blocker and beta-blocker combinations or selective beta-blockers in low doses may not cause unopposed alpha stimulation in this situation. Labetalol, a beta-blocker with some alpha-blocking activity, has been used successfully to treat cocaine-induced hypertension. In addition, cocaine can reduce the therapeutic effects of beta-blockers.

Codeine; guaiFENesin; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Codeine; Phenylephrine; Promethazine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Co-Enzyme Q10, Ubiquinone: (Moderate) Co-enzyme Q10, ubiquinone (CoQ10) may lower blood pressure. CoQ10 use in combination with antihypertensive agents may lead to additional reductions in blood pressure in some individuals. Patients who choose to take CoQ10 concurrently with antihypertensive medications should receive periodic blood pressure monitoring. Patients should be advised to inform their prescriber of their use of CoQ10.
Colestipol: (Moderate) Colestipol can bind with and possibly decrease the oral absorption of propranolol. To minimize drug interactions, administer other drugs at least 1 hour before or at least 4 to 6 hours after the administration of colestipol.
Corticosteroids: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Cortisone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Crizotinib: (Major) Avoid coadministration of crizotinib with agents known to cause bradycardia, such as beta-blockers, to the extent possible due to the risk of additive bradycardia. If concomitant use is unavoidable, monitor heart rate and blood pressure regularly. An interruption of crizotinib therapy or dose adjustment may be necessary if bradycardia occurs.
Dacomitinib: (Moderate) Monitor for increased toxicity of propranolol if coadministered with dacomitinib. Coadministration may increase serum concentrations of propranolol. Propranolol is a CYP2D6 substrate; dacomitinib is a strong CYP2D6 inhibitor.
Dapagliflozin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well

and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Dapagliflozin; metFORMIN: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Dapagliflozin; sAXagliptin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with

compelling indications for beta-blocker therapy when no other contraindications are present.
Darifenacin: (Moderate) Monitor for increased toxicity of propranolol if coadministered with darifenacin. Coadministration may increase serum concentrations of propranolol. Propranolol is a CYP2D6 substrate; darifenacin is a moderate CYP2D6 inhibitor.
Darunavir; Cobicistat: (Moderate) Coadministration of cobicistat (a CYP2D6 inhibitor) with beta-blockers metabolized by CYP2D6, such as propranolol, may result in elevated beta-blocker serum concentrations. If used concurrently, close clinical monitoring with appropriate beta-blocker dose reductions are advised.
Darunavir; Cobicistat; Emtricitabine; Tenofovir alafenamide: (Moderate) Coadministration of cobicistat (a CYP2D6 inhibitor) with beta-blockers metabolized by CYP2D6, such as propranolol, may result in elevated beta-blocker serum concentrations. If used concurrently, close clinical monitoring with appropriate beta-blocker dose reductions are advised.
Dasiglucagon: (Minor) A temporary increase in both blood pressure and pulse rate may occur following the administration of glucagon. Patients taking beta-blockers might be expected to have a greater increase in both pulse and blood pressure. Glucagon exerts positive inotropic and chronotropic effects and may, therefore, cause tachycardia and hypertension in some patients. The increase in blood pressure and pulse rate may require therapy in some patients with coronary artery disease.
Deflazacort: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Desflurane: (Moderate) Concurrent use of beta-blockers with desflurane may result in exaggerated cardiovascular effects (e.g., hypotension and negative inotropic effects). Beta-blockers may be continued during general anesthesia as long as the patient is monitored for cardiac depressant and hypotensive effects. Withdrawal of a beta-blocker perioperatively may be detrimental to the patient's clinical status and is not recommended. Caution is advised if these drugs are administered together.
Desloratadine; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Desogestrel: Ethinyl Estradiol; Ferrous Bisglycinate: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Desogestrel; Ethinyl Estradiol: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl

estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Desvenlafaxine: (Major) Dosage adjustments of some beta-blockers may be necessary during concurrent use of desvenlafaxine. Although clinical studies have shown that desvenlafaxine does not have a clinically relevant effect on CYP2D6 inhibition at doses of 100 mg/day, the manufacturer recommends that primary substrates of CYP2D6, such as propranolol, metoprolol, or nebivolol, be dosed at the original level when co-administered with desvenlafaxine 100 mg or lower or when desvenlafaxine is discontinued. The dose of these CYP2D6 substrates should be reduced by up to one-half if co-administered with desvenlafaxine 400 mg/day.
dexAMETHasone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Dexbrompheniramine; Dextromethorphan; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Dexbrompheniramine; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Dexchlorpheniramine; Dextromethorphan; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
dexmedeTOMIDine: (Moderate) Monitor blood pressure and heart rate during concomitant use of dexmedetomidine and beta-blockers due to the risk of additive bradycardia and hypotensive effects.
Dexmethylphenidate: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Dextroamphetamine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Dextromethorphan; buPROPion: (Minor) Monitor for an increased incidence of propranolol-related adverse effects if bupropion and propranolol are used concomitantly. Coadministration of bupropion and propranolol may result in increased plasma concentrations of propranolol. Bupropion and hydroxybupropion, the major

active metabolite, are inhibitors of CYP2D6 in vitro. Propranolol is a CYP2D6 substrate.
Dextromethorphan; diphenhydrAMINE; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Dextromethorphan; guaiFENesin; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Dextromethorphan; guaiFENesin; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Dextromethorphan; quiNIDine: (Major) Patients receiving combined therapy with quinidine and propranolol should be monitored for potential hypotension, orthostasis, bradycardia and/or AV block and heart failure, Reduce the beta-blocker dosage if necessary. Quinidine may have additive effects (e.g., reduced heart rate, hypotension) on cardiovascular parameters when used together with beta-blockers, such as propranolol. Quinidine is a known inhibitor of CYP2D6, and may additionally impair the hepatic clearance of propanolol (CYP2D6 substrate); patients should be monitored for excess beta-blockade.
Diclofenac: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Diclofenac; miSOPROStol: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Diethylpropion: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Diflunisal: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Digoxin: (Moderate) Monitor heart rate during concomitant digoxin and propranolol use due to increased risk for bradycardia. Both digoxin and propranolol slow atrioventricular conduction (AV) and decrease heart rate; additive effects on AV node conduction can result in bradycardia and advanced or complete heart block.
Dihydroergotamine: (Moderate) Monitor for an increase in the incidence and severity of vasospastic adverse reactions including cerebral and peripheral ischemia during

concomitant use of ergotamine and propranolol. Propranolol may potentiate the vasoconstrictive action of ergotamine by blocking the vasodilating properties of epinephrine.
dilTIAZem: (Major) Intravenous propranolol is contraindicated with intravenous diltiazem use in close proximity (within a few hours). Fatal cardiac arrests have occurred in patients receiving intravenous beta-blockers and intravenous calcium channel blockers. Use oral propranolol and oral diltiazem with caution due to risk for additive negative effects on heart rate, AV conduction, and/or cardiac contractility. If combination therapy is initiated or withdrawn in conjunction with propranolol, an adjustment in the propranolol dose may be warranted. Administration of diltiazem concomitantly with propranolol in 5 normal volunteers resulted in increased propranolol concentrations in all subjects, and bioavailability of propranolol was increased approximately 50%. In vitro, propranolol appears to be displaced from its binding sites by diltiazem.
Dipeptidyl Peptidase-4 Inhibitors: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
diphenhydrAMINE; Ibuprofen: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
diphenhydrAMINE; Naproxen: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
diphenhydrAMINE; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.

Dipyridamole: (Major) Beta-blockers should generally be withheld before dipyridamole-stress testing. Monitor the heart rate carefully following the dipyridamole injection.
Disopyramide: (Major) Because the pharmacologic effects of propranolol include AV nodal conduction depression and negative inotropy, additive effects are possible when used in combination with disopyramide. Propranolol has occasionally been used with disopyramide; however, the manufacturer states that the concomitant use of disopyramide with propranolol should be reserved for patients with refractory life-threatening arrhythmias. Such use may produce serious negative inotropic effects, or may excessively prolong conduction. In healthy subjects, no significant drug interaction has been observed when propranolol is coadministered with disopyramide.
DOBUTamine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Donepezil: (Moderate) The increase in vagal tone induced by some cholinesterase inhibitors may produce bradycardia, hypotension, or syncope. The vagotonic effect of these drugs may be increased when given with other medications known to cause bradycardia such as beta-blockers. These interactions are pharmacodynamic in nature rather than pharmacokinetic.
Donepezil; Memantine: (Moderate) The increase in vagal tone induced by some cholinesterase inhibitors may produce bradycardia, hypotension, or syncope. The vagotonic effect of these drugs may be increased when given with other medications known to cause bradycardia such as beta-blockers. These interactions are pharmacodynamic in nature rather than pharmacokinetic.
DOPamine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Doxapram: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Doxazosin: (Moderate) Orthostatic hypotension may be more likely in some patients if beta-blockers are coadministered with alpha-blockers. Monitor blood pressure and for orthostasis, especially at medication initiation.
Dronedarone: (Major) In dronedarone clinical trials, bradycardia was seen more frequently in patients also receiving beta blockers. If coadministration of dronedarone and a beta blocker is unavoidable, administer a low dose of the beta blocker initially and increase the dosage only after ECG verification of tolerability. Concomitant administration may decreased AV and sinus node conduction. Furthermore, dronedarone is an inhibitor of CYP2D6, and some beta blockers are substrates for

CYP2D6 (e.g., metoprolol, propranolol, nebivolol, carvedilol). Coadministration of dronedarone with a single dose of propranolol and multiple doses of metoprolol increased propranolol and metoprolol exposure by 1.3- and 1.6-fold, respectively.
Drospirenone; Ethinyl Estradiol: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Drospirenone; Ethinyl Estradiol; Levomefolate: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Dulaglutide: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
DULoxetine: (Moderate) Monitor blood pressure during concomitant duloxetine and propranolol use. Concomitant use increases the risk for hypotension, including orthostatic hypotension and syncope. Consider reducing the duloxetine dose or discontinuing duloxetine if symptomatic orthostatic hypotension, falls, or syncope occur during treatment.
Dutasteride; Tamsulosin: (Minor) Tamsulosin did not potentiate the hypotensive effects of atenolol. However, since the symptoms of orthostasis are reported more frequently in tamsulosin-treated vs. placebo patients, there is a potential risk of enhanced hypotensive effects when co-administered with antihypertensive agents.
Eletriptan: (Minor) Periodically monitor blood pressure in patients who regularly use eletriptan and are taking propranolol. Monitor for the rare patient who might

experience an increase in dose-related side effects of eletriptan, such as nausea, dizziness, and drowsiness. No dosage adjustment appears to be needed for eletriptan. The Cmax and AUC of eletriptan were increased by 10 and 33%, respectively, in the presence of propranolol. No interactive increases in blood pressure were observed. The interaction should not be significant for most patients.
Eliglustat: (Moderate) Coadministration of propranolol and eliglustat may result in increased plasma concentrations of propranolol. Consider reducing the propranolol dosage and titrating to clinical effect. Propranolol is a CYP2D6 and P-glycoprotein (P-gp) substrate; eliglustat is a CYP2D6 and P-gp inhibitor.
Elvitegravir; Cobicistat; Emtricitabine; Tenofovir Alafenamide: (Moderate) Coadministration of cobicistat (a CYP2D6 inhibitor) with beta-blockers metabolized by CYP2D6, such as propranolol, may result in elevated beta-blocker serum concentrations. If used concurrently, close clinical monitoring with appropriate beta-blocker dose reductions are advised.
Elvitegravir; Cobicistat; Emtricitabine; Tenofovir Disoproxil Fumarate: (Moderate) Coadministration of cobicistat (a CYP2D6 inhibitor) with beta-blockers metabolized by CYP2D6, such as propranolol, may result in elevated beta-blocker serum concentrations. If used concurrently, close clinical monitoring with appropriate beta-blocker dose reductions are advised.
Empagliflozin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Empagliflozin; Linagliptin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other

symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Empagliflozin; Linagliptin; metFORMIN: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Empagliflozin; metFORMIN: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A

selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Enasidenib: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of enasidenib as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 and CYP2D6 substrate and enasidenib is a strong CYP1A2 and weak CYP2D6 inhibitor.
ePHEDrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
ePHEDrine; guaiFENesin: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
EPINEPHrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Epoprostenol: (Moderate) Epoprostenol can have additive effects when administered with other antihypertensive agents, including beta-blockers. These effects can be used to therapeutic advantage, but dosage adjustments may be necessary.
Ergotamine: (Moderate) Monitor for an increase in the incidence and severity of vasospastic adverse reactions including cerebral and peripheral ischemia during concomitant use of ergotamine and propranolol. Propranolol may potentiate the vasoconstrictive action of ergotamine by blocking the vasodilating properties of epinephrine.
Ergotamine; Caffeine: (Moderate) Monitor for an increase in the incidence and severity of vasospastic adverse reactions including cerebral and peripheral ischemia during concomitant use of ergotamine and propranolol. Propranolol may potentiate the vasoconstrictive action of ergotamine by blocking the vasodilating properties of epinephrine.
Ertugliflozin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other

symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Ertugliflozin; metFORMIN: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Ertugliflozin; SITagliptin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A

selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Escitalopram: (Minor) Escitalopram modestly inhibits the hepatic CYP2D6 isoenzyme. This can result in increased concentrations of drugs metabolized via the same pathway, including propranolol. Increased serum levels of the beta-blockers could result in reductions in cardioselectivity.
Estradiol: (Minor) Estrogens can induce fluid retention and may increase blood pressure in some patients; patients who are receiving antihypertensive agents concurrently with hormonal contraceptives should be monitored for antihypertensive effectiveness.
Ethinyl Estradiol; Norelgestromin: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Ethinyl Estradiol; Norethindrone Acetate: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Ethinyl Estradiol; Norgestrel: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Ethiodized Oil: (Moderate) Use caution when administering non-ionic contrast media to patients taking beta-blockers. Beta-blockers lower the threshold for and increase the severity of contrast reactions and reduce the responsiveness of treatment of hypersensitivity reactions with epinephrine.
Ethynodiol Diacetate; Ethinyl Estradiol: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Etodolac: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Etomidate: (Major) General anesthetics can potentiate the antihypertensive effects of beta-blockers and can produce prolonged hypotension. Beta-blockers may be continued during general anesthesia as long as the patient is monitored for cardiac depressant and

hypotensive effects.
Etonogestrel; Ethinyl Estradiol: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Etrasimod: (Moderate) Monitor heart rate if concomitant use of etrasimod and a beta-blocker is necessary. Transient decreases in heart rate and AV conduction delays have been observed with etrasimod therapy which may increase the risk for bradycardia. The risk for harm may be greatest if a beta-blocker is added to a stable etrasimod regimen.
Everolimus: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of everolimus as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and everolimus is a CYP2D6 inhibitor.
Exenatide: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Ezetimibe; Simvastatin: (Minor) After administration of single doses of simvastatin and propranolol, there was a significant decrease in mean Cmax, with no change in AUC, of simvastatin. The clinical significance of this interaction is unknown. Monitor for potential reduced cholesterol-lowering efficacy when propranolol is coadministered with niacin; simvastatin.
Fedratinib: (Moderate) Monitor for increased propranolol adverse reactions including bradycardia and hypotension during coadministration of fedratinib. Propranolol exposure may increase. Fedratinib is a moderate CYP2D6 and CYP2C19 inhibitor and propranolol is a CYP2D6 and CYP2C19 substrate.
Fenofibric Acid: (Minor) At therapeutic concentrations, fenofibric acid is a weak inhibitor

of CYP2C19. Concomitant use of fenofibric acid with CYP2C19 substrates, such as propranolol, has not been formally studied. Fenofibric acid may theoretically increase plasma concentrations of CYP2C19 substrates and could lead to toxicity for drugs that have a narrow therapeutic range. Monitor the therapeutic effect of propranolol during coadministration with fenofibric acid.
Fenoldopam: (Major) Avoid concomitant use of fenoldopam with beta-blockers due to the risk of hypotension. If used together, monitor blood pressure frequently. Beta-blockers may inhibit the sympathetic reflex response to fenoldopam.
Fenoprofen: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Fexinidazole: (Major) Avoid concomitant use of fexinidazole and medications that cause bradycardia, such as beta-blockers, especially in patients with additional risk factors for torsade de pointes (TdP). Coadministration may increase the risk of QT/QTc prolongation and TdP. Fexinidazole is known to prolong the QT interval and medications that may cause bradycardia can increase the risk for TdP in patients with a prolonged QT/QTc interval.
Fexofenadine; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Fingolimod: (Major) If possible, do not start fingolimod in a patient who is taking a drug that slows the heart rate or atrioventricular conduction such as beta-blockers. Use of these drugs during fingolimod initiation may be associated with severe bradycardia or heart block. Seek advice from the prescribing physician regarding the possibility to switch to drugs that do not slow the heart rate or atrioventricular conduction before initiating fingolimod. After the first fingolimod dose, overnight monitoring with continuous ECG in a medical facility is advised for patients who cannot stop taking drugs that slow the heart rate or atrioventricular conduction. Experience with fingolimod in patients receiving concurrent therapy with drugs that slow the heart rate or atrioventricular conduction is limited.
Fish Oil, Omega-3 Fatty Acids (Dietary Supplements): (Moderate) High doses of fish oil supplements may produce a blood pressure lowering effect It is possible that additive reductions in blood pressure may be seen when fish oils are used in a patient already taking antihypertensive agents.
Flecainide: (Moderate) Monitor heart rate during concomitant flecainide and propranolol use due to risk for additive negative inotropic effects.
Fludrocortisone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.

Flunisolide: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Fluorescein: (Moderate) Patients on beta-blockers are at an increased risk of adverse reaction when administered fluorescein injection. It is thought that beta-blockers may worsen anaphylaxis severity by exacerbating bronchospasm or by increasing the release of anaphylaxis mediators; alternately, beta-blocker therapy may make the patient more pharmacodynamically resistance to epinephrine rescue treatment.
FLUoxetine: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of fluoxetine as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and fluoxetine is a moderate CYP2D6 inhibitor.
Flurbiprofen: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Fluticasone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Fluticasone; Salmeterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used. (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Fluticasone; Umeclidinium; Vilanterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used. (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk

for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Fluticasone; Vilanterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used. (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
fluvoxaMINE: (Moderate) Fluvoxamine can also inhibit hepatic cytochrome P-450 isoenzymes and has been shown to interfere with propranolol clearance however clinical symptoms of excessive beta-blocker effects were not seen.
Food: (Major) Advise patients to avoid cannabis use during propranolol treatment due to decreased exposure of propranolol which may alter its efficacy. Cannabis use induces CYP1A2 and propranolol is a CYP1A2 substrate. The induction potential of cannabis is greatest with chronic inhalation. Other routes of administration or sporadic use may have less of an effect.
Formoterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Formoterol; Mometasone: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used. (Moderate)

Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Foscarbidopa; Foslevodopa: (Moderate) Monitor for postural hypotension if concomitant use of levodopa and an antihypertensive, such as a beta-blocker, is necessary. A beta-blocker dosage reduction may be required. Both medications lower blood pressure and this effect may be additive with concomitant use.
Fosphenytoin: (Minor) Phenytoin has been shown to accelerate the hepatic metabolism of propranolol. Because fosphenytoin is metabolized to phenytoin, fosphenytoin will most likely also accelerate the hepatic metabolism of propranolol.
Gabapentin: (Minor) The combination of propranolol and gabapentin may induce dystonia via a pharmacodynamic interaction.
Galantamine: (Moderate) The increase in vagal tone induced by cholinesterase inhibitors, such as galantamine, may produce bradycardia or syncope. The vagotonic effect of galantamine may theoretically be increased when given with beta-blockers.
General anesthetics: (Major) General anesthetics can potentiate the antihypertensive effects of beta-blockers and can produce prolonged hypotension. Beta-blockers may be continued during general anesthesia as long as the patient is monitored for cardiac depressant and hypotensive effects.
Ginger, Zingiber officinale: (Minor) In vitro studies have demonstrated the positive inotropic effects of certain gingerol constituents of ginger; but it is unclear if whole ginger root exhibits these effects clinically in humans. It is theoretically possible that excessive doses of ginger could affect the action of inotropes; however, no clinical data are available.
Glimepiride: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are

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glipiZIDE: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
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Glucagon: (Minor) A temporary increase in both blood pressure and pulse rate may occur following the administration of glucagon. Patients taking beta-blockers might be expected to have a greater increase in both pulse and blood pressure. Glucagon exerts positive inotropic and chronotropic effects and may, therefore, cause tachycardia and hypertension in some patients. The increase in blood pressure and pulse rate may require therapy in some patients with coronary artery disease.
glyBURIDE: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
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Glycopyrrolate; Formoterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
guaiFENesin; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
guaiFENesin; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the

cardiovascular effects of either drug.
guanFACINE: (Moderate) Guanfacine can have additive effects when administered with other antihypertensive agents, including beta-blockers. These effects can be used to therapeutic advantage, but dosage adjustments may be necessary.
Haloperidol: (Moderate) Haloperidol should be used cautiously with propranolol due to the possibility of additive hypotension and increased concentrations of propranolol. Propranolol is significantly metabolized by CYP2D6 isoenzymes. A case report of 3 severe hypotension episodes (2 requiring cardiopulmonary resuscitation) has been reported in one 48 year old woman when propranolol and haloperidol have been coadministered. Additive hypotensive effects and haloperidol-mediated CYP2D6 inhibition may have contributed to this interaction.
Hawthorn, Crataegus laevigata: (Moderate) Hawthorn, Crataegus laevigata (also known as C. oxyacantha) may potentially interact with antihypertensive, heart failure, or arrhythmia medications such as the beta-blockers. Following hawthorn administration, the cardiac action potential duration is increased and the refractory period is prolonged. Hawthorn may also lower peripheral vascular resistance. Patients with hypertension or heart failure should be advised to only use hawthorn with their prescribed medications after discussion with their prescriber. Patients who choose to take hawthorn should receive periodic blood pressure and heart rate monitoring.
House Dust Mite Allergen Extract: (Moderate) Advise patients who are undergoing allergy testing or receiving maintenance allergen immunotherapy (AIT) that allergic reactions, including symptoms of anaphylaxis, may be more severe while receiving beta-blocker therapy. Risk varies by individual based on indication for therapy and history. This risk is generally considered minimal for most patients who are receiving maintenance AIT and the benefits of continuing beta-blocker therapy typically outweigh the risks in patients with cardiovascular disease.
hydrALAZINE; Isosorbide Dinitrate, ISDN: (Moderate) Nitroglycerin can cause hypotension. This action may be additive with other agents that can cause hypotension such as antihypertensive agents or other peripheral vasodilators. Patients should be monitored more closely for hypotension if nitroglycerin, including nitroglycerin rectal ointment, is used concurrently with any beta-blockers.
HYDROcodone; Ibuprofen: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Hydrocortisone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Ibuprofen: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.

Ibuprofen; Famotidine: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Ibuprofen; Pseudoephedrine: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs. (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Icosapent ethyl: (Moderate) Beta-blockers may exacerbate hypertriglyceridemia and should be discontinued or changed to alternate therapy, if possible, prior to initiation of icosapent ethyl.
Iloperidone: (Moderate) Secondary to alpha-blockade, iloperidone can produce vasodilation that may result in additive effects during concurrent use with antihypertensive agents. The potential reduction in blood pressure can precipitate orthostatic hypotension and associated dizziness, tachycardia, and syncope. If concurrent use of iloperidone and antihypertensive agents is necessary, patients should be counseled on measures to prevent orthostatic hypotension, such as sitting on the edge of the bed for several minutes prior to standing in the morning and rising slowly from a seated position. Close monitoring of blood pressure is recommended until the full effects of the combination therapy are known.
Iloprost: (Moderate) Additive reductions in blood pressure may occur when inhaled iloprost is administered to patients receiving other antihypertensive agents.
Imatinib: (Moderate) Propranolol is significantly metabolized by CYP2D6 isoenzymes. CYP2D6 inhibitors, such as imatinib, could theoretically impair propranolol metabolism; the clinical significance of such interactions is unknown.
Incretin Mimetics: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and

stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Indacaterol; Glycopyrrolate: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Indomethacin: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Insulin Aspart: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Insulin Aspart; Insulin Aspart Protamine: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a

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Insulin Degludec: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Insulin Degludec; Liraglutide: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and

stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Insulin Detemir: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Insulin Glargine: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Insulin Glargine; Lixisenatide: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide

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Insulin Glulisine: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Insulin Lispro: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well

and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Insulin Lispro; Insulin Lispro Protamine: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Insulin, Inhaled: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with

compelling indications for beta-blocker therapy when no other contraindications are present.
Insulins: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Intravenous Lipid Emulsions: (Moderate) High doses of fish oil supplements may produce a blood pressure lowering effect It is possible that additive reductions in blood pressure may be seen when fish oils are used in a patient already taking antihypertensive agents.
Iobenguane I 131: (Major) Discontinue propranolol for at least 5 half-lives before the administration of the dosimetry dose or a therapeutic dose of iobenguane I-131. Do not restart propranolol until at least 7 days after each iobenguane I-131 dose. Drugs that reduce catecholamine uptake or deplete catecholamine stores, such as propranolol, may interfere with iobenguane I-131 uptake into cells and interfere with dosimetry calculations resulting in altered iobenguane I-131 efficacy.
Iodixanol: (Moderate) Use caution when administering non-ionic contrast media to patients taking beta-blockers. Beta-blockers lower the threshold for and increase the severity of contrast reactions and reduce the responsiveness of treatment of hypersensitivity reactions with epinephrine.
Iohexol: (Moderate) Use caution when administering non-ionic contrast media to patients taking beta-blockers. Beta-blockers lower the threshold for and increase the severity of contrast reactions and reduce the responsiveness of treatment of hypersensitivity reactions with epinephrine.
Iomeprol: (Moderate) Use caution when administering non-ionic contrast media to patients taking beta-blockers. Beta-blockers lower the threshold for and increase the severity of contrast reactions and reduce the responsiveness of treatment of hypersensitivity reactions with epinephrine.

Iopamidol: (Moderate) Use caution when administering non-ionic contrast media to patients taking beta-blockers. Beta-blockers lower the threshold for and increase the severity of contrast reactions and reduce the responsiveness of treatment of hypersensitivity reactions with epinephrine.
Iopromide: (Moderate) Use caution when administering non-ionic contrast media to patients taking beta-blockers. Beta-blockers lower the threshold for and increase the severity of contrast reactions and reduce the responsiveness of treatment of hypersensitivity reactions with epinephrine.
Ioversol: (Moderate) Use caution when administering non-ionic contrast media to patients taking beta-blockers. Beta-blockers lower the threshold for and increase the severity of contrast reactions and reduce the responsiveness of treatment of hypersensitivity reactions with epinephrine.
Ipratropium; Albuterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Isocarboxazid: (Moderate) Monitor blood pressure and heart rate closely when beta-blockers are coadministered with catecholamine-depleting agents, such as monoamine oxidase inhibitors. An additive reduction in sympathetic tone may occur, which may increase the risk for hypotension or marked bradycardia-related vertigo, syncope, or postural hypotension.
Isoflurane: (Major) General anesthetics can potentiate the antihypertensive effects of beta-blockers and can produce prolonged hypotension. Beta-blockers may be continued during general anesthesia as long as the patient is monitored for cardiac depressant and hypotensive effects.
Isophane Insulin (NPH): (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A

selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Isoproterenol: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Isosorbide Dinitrate, ISDN: (Moderate) Nitroglycerin can cause hypotension. This action may be additive with other agents that can cause hypotension such as antihypertensive agents or other peripheral vasodilators. Patients should be monitored more closely for hypotension if nitroglycerin, including nitroglycerin rectal ointment, is used concurrently with any beta-blockers.
Isosorbide Mononitrate: (Moderate) Nitroglycerin can cause hypotension. This action may be additive with other agents that can cause hypotension such as antihypertensive agents or other peripheral vasodilators. Patients should be monitored more closely for hypotension if nitroglycerin, including nitroglycerin rectal ointment, is used concurrently with any beta-blockers.
Isosulfan Blue: (Moderate) Use caution when administering non-ionic contrast media to patients taking beta-blockers. Beta-blockers lower the threshold for and increase the severity of contrast reactions and reduce the responsiveness of treatment of hypersensitivity reactions with epinephrine.
Isradipine: (Moderate) Although concomitant therapy with beta-blockers and isradipine is generally well tolerated and can even be beneficial in some cases, coadministration of these agents can induce excessive bradycardia or hypotension. Isradipine when used in combination with beta-blockers, especially in heart failure patients, can result in additive negative inotropic effects. Finally, angina has been reported when beta-adrenergic blocking agents are withdrawn abruptly when isradipine therapy is initiated. A gradual downward titration of the beta-adrenergic blocking agent dosage during initiation of isradipine therapy can minimize or eliminate this potential interaction. Patients should be monitored carefully, however, for excessive bradycardia, cardiac conduction abnormalities, or hypotension when these drugs are given together. In general, these reactions are more likely to occur with other non-dihydropyridine calcium channel blockers than with isradipine.
Ivabradine: (Moderate) Monitor heart rate if ivabradine is coadministered with other negative chronotropes like beta-blockers. Most patients receiving ivabradine will receive concomitant beta-blocker therapy. Coadministration of drugs that slow heart rate

increases the risk for bradycardia.
Ketamine: (Major) General anesthetics can potentiate the antihypertensive effects of beta-blockers and can produce prolonged hypotension. Beta-blockers may be continued during general anesthesia as long as the patient is monitored for cardiac depressant and hypotensive effects.
Ketoprofen: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Ketorolac: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Lacosamide: (Moderate) Use lacosamide with caution in patients taking concomitant medications that affect cardiac conduction, such as beta-blockers, because of the risk of AV block, bradycardia, or ventricular tachyarrhythmia. If use together is necessary, obtain an ECG prior to lacosamide initiation and after treatment has been titrated to steady-state. In addition, monitor patients receiving lacosamide via the intravenous route closely.
Lanreotide: (Moderate) Concomitant administration of bradycardia-inducing drugs (e.g., beta-adrenergic blockers) may have an additive effect on the reduction of heart rate associated with lanreotide. Adjust the beta-blocker dose if necessary.
Lasmiditan: (Moderate) Monitor heart rate if lasmiditan is coadministered with beta-blockers as concurrent use may increase the risk for bradycardia. Lasmiditan has been associated with lowering of heart rate. In a drug interaction study, addition of a single 200 mg dose of lasmiditan to a beta-blocker (propranolol) decreased heart rate by an additional 5 beats per minute.
Levalbuterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Levamlodipine: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
Levodopa: (Moderate) Monitor for postural hypotension if concomitant use of levodopa and an antihypertensive, such as a beta-blocker, is necessary. A beta-blocker dosage

reduction may be required. Both medications lower blood pressure and this effect may be additive with concomitant use.
Levonorgestrel; Ethinyl Estradiol: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Levonorgestrel; Ethinyl Estradiol; Ferrous Bisglycinate: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Levonorgestrel; Ethinyl Estradiol; Ferrous Fumarate: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Levothyroxine: (Minor) Because thyroid hormones cause cardiac stimulation including increased heart rate and increased contractility, the effects of beta-blockers may be reduced by thyroid hormones. The reduction of effects may be especially evident when a patient goes from a hypothyroid to a euthyroid state or when excessive amounts of thyroid hormone is given to the patient.
Levothyroxine; Liothyronine (Porcine): (Minor) Because thyroid hormones cause cardiac stimulation including increased heart rate and increased contractility, the effects of beta-blockers may be reduced by thyroid hormones. The reduction of effects may be especially evident when a patient goes from a hypothyroid to a euthyroid state or when excessive amounts of thyroid hormone is given to the patient.
Lidocaine: (Major) Drugs such as beta-blockers that decrease cardiac output reduce hepatic blood flow and thereby decrease lidocaine hepatic clearance. Also, opposing effects on conduction exist between lidocaine and beta-blockers while their effects to decrease automaticity may be additive. Propranolol has been shown to decrease lidocaine clearance and symptoms of lidocaine toxicity have been seen as a result of this interaction. This interaction is possible with other beta-blocking agents since most decrease hepatic blood flow. Monitoring of lidocaine concentrations is recommended during concomitant therapy with beta-blockers.
Lidocaine; EPINEPHrine: (Major) Drugs such as beta-blockers that decrease cardiac output reduce hepatic blood flow and thereby decrease lidocaine hepatic clearance. Also, opposing effects on conduction exist between lidocaine and beta-blockers while their effects to decrease automaticity may be additive. Propranolol has been shown to decrease lidocaine clearance and symptoms of lidocaine toxicity have been seen as a result of this interaction. This interaction is possible with other beta-blocking agents

since most decrease hepatic blood flow. Monitoring of lidocaine concentrations is recommended during concomitant therapy with beta-blockers. (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Lidocaine; Prilocaine: (Major) Drugs such as beta-blockers that decrease cardiac output reduce hepatic blood flow and thereby decrease lidocaine hepatic clearance. Also, opposing effects on conduction exist between lidocaine and beta-blockers while their effects to decrease automaticity may be additive. Propranolol has been shown to decrease lidocaine clearance and symptoms of lidocaine toxicity have been seen as a result of this interaction. This interaction is possible with other beta-blocking agents since most decrease hepatic blood flow. Monitoring of lidocaine concentrations is recommended during concomitant therapy with beta-blockers. (Moderate) Local anesthetics may cause additive hypotension in combination with antihypertensive agents.
Linagliptin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Linagliptin; metFORMIN: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in

recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Linezolid: (Moderate) Linezolid is an antibiotic that is also a reversible, non-selective MAO inhibitor. Bradycardia may be worsened when MAO-inhibitors are co-administered to patients receiving beta-blockers. Use linezolid cautiously in patients receiving beta-blockers.
Liothyronine: (Minor) Because thyroid hormones cause cardiac stimulation including increased heart rate and increased contractility, the effects of beta-blockers may be reduced by thyroid hormones. The reduction of effects may be especially evident when a patient goes from a hypothyroid to a euthyroid state or when excessive amounts of thyroid hormone is given to the patient.
Liraglutide: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Lisdexamfetamine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Lixisenatide: (Moderate) Increased frequency of blood glucose monitoring may be

required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Lofexidine: (Major) Because both lofexidine and propranolol can cause hypotension and bradycardia, concurrent use should be avoided if possible. Patients being given lofexidine in an outpatient setting should be capable of and instructed on self-monitoring for hypotension, orthostasis, bradycardia, and associated symptoms. If clinically significant or symptomatic hypotension and/or bradycardia occur, the next dose of lofexidine should be reduced in amount, delayed, or skipped.
Lonafarnib: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of lonafarnib as concurrent use may increase propranolol exposure. Propranolol is a CYP2C19 substrate and lonafarnib is a moderate CYP2C19 inhibitor.
Lopinavir; Ritonavir: (Moderate) Concurrent administration of propranolol with ritonavir may result in elevated propranolol plasma concentrations. Cardiac and neurologic events have been reported when ritonavir is concurrently administered with beta-blockers. Propranolol is metabolized by the hepatic isoenzyme CYP2D6; ritonavir is an inhibitor of this enzyme. Caution and close monitoring are advised if these drugs are administered together. Decreased beta-blocker dosage may be needed.
Loratadine; Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Lumacaftor; Ivacaftor: (Minor) Concomitant use of propranolol and lumacaftor; ivacaftor may decrease the systemic exposure of propranolol; caution and monitoring of blood pressure and other therapeutic effects are advised if these drugs are used together. Propranolol is partially metabolized by CYP2C19; in vitro data suggest that lumacaftor

may induce CYP2C19.
Lumacaftor; Ivacaftor: (Minor) Concomitant use of propranolol and lumacaftor; ivacaftor may decrease the systemic exposure of propranolol; caution and monitoring of blood pressure and other therapeutic effects are advised if these drugs are used together. Propranolol is partially metabolized by CYP2C19; in vitro data suggest that lumacaftor may induce CYP2C19.
Lurasidone: (Moderate) Due to the antagonism of lurasidone at alpha-1 adrenergic receptors, the drug may enhance the hypotensive effects of alpha-blockers and other antihypertensive agents. If concurrent use of lurasidone and antihypertensive agents is necessary, patients should be counseled on measures to prevent orthostatic hypotension, such as sitting on the edge of the bed for several minutes prior to standing in the morning and rising slowly from a seated position. Close monitoring of blood pressure is recommended until the full effects of the combination therapy are known.
Magnesium Hydroxide: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Magnesium Salicylate: (Moderate) Concurrent use of beta-blockers with aspirin and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
Magnesium Salts: (Moderate) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Mavacamten: (Moderate) Expect additive negative inotropic effects during concomitant use of mavacamten and beta-blockers. If concomitant therapy with beta-blockers is initiated, or if the dose is increased, monitor left ventricular ejection fraction closely until stable doses and clinical response have been achieved. Avoid concomitant use of mavacamten and a beta-blocker plus verapamil or diltiazem due to an increased risk of left ventricular systolic dysfunction and heart failure symptoms.
Mavorixafor: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of mavorixafor as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and mavorixafor is strong CYP2D6 inhibitor.
Meclofenamate Sodium: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Mefenamic Acid: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.

Mefloquine: (Major) Concurrent use of mefloquine and beta blockers can result in ECG abnormalities or cardiac arrest.
Meglitinides: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Meloxicam: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Meloxicam; Rizatriptan: (Major) Recommendations for concomitant use of rizatriptan and propranolol vary by dosage form. Concomitant use of rizatriptan oral film and propranolol is contraindicated, and a dosage reduction may be necessary for rizatriptan oral tablets. Limit the rizatriptan dose to 5 mg/dose and 15 mg/24 hours for adults receiving rizatriptan tablets. Avoid use of rizatriptan tablets in pediatric patients weighing less than 40 kg and limit the rizatriptan dose to 5 mg/24 hours in pediatric patients weighing 40 kg or more. Rizatriptan oral film is contraindicated with propranolol because a dose adjustment is not possible. Coadministration has been observed to increase rizatriptan overall exposure by 70% without affecting concentrations of the active N-monodesmethyl metabolite. (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Mepivacaine: (Major) Propranolol has been shown to significantly decrease the clearance of the amide local anesthetics (e.g., lidocaine, bupivacaine, and mepivacaine). Lidocaine and bupivacaine toxicity have been reported after coadministration with propranolol. The mechanism of the interaction between propranolol and lidocaine is thought to be due to propranolol-induced decreased hepatic blood flow causing decreased elimination of lidocaine.

Metaproterenol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
metFORMIN: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
metFORMIN; sAXagliptin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and

stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
metFORMIN; SITagliptin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Methacholine: (Moderate) Beta-blockers may impair reversal of methacholine-induced bronchoconstriction with an inhaled rapid-acting beta-agonist.
Methamphetamine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
methIMAzole: (Minor) Hyperthyroidism may cause increased clearance of beta blockers that possess a high extraction ratio. A dose reduction of some beta-blockers may be needed when a hyperthyroid patient treated with methimazole becomes euthyroid.
Methohexital: (Major) General anesthetics can potentiate the antihypertensive effects of beta-blockers and can produce prolonged hypotension.
Methylergonovine: (Moderate) Monitor for an increase in the incidence and severity of vasospastic adverse reactions, including cerebral and peripheral ischemia, during concomitant use of methylergonovine and beta-blockers. Beta-blockers may potentiate the vasoconstrictive actions of methylergonovine.
Methylphenidate: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
methylPREDNISolone: (Moderate) Monitor blood sugar during concomitant

corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Mexiletine: (Moderate) Mexiletine has been found to increase propranolol concentrations in patients receiving concomitant therapy. The significance of the elevated propranolol concentration is not known as beta-blockers have a wide therapeutic range. It may be prudent to monitor patients for adverse effects when mexiletine are combined with propranolol.
Midodrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Miglitol: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Milrinone: (Moderate) Concurrent administration of antihypertensive agents could lead to additive hypotension when administered with milrinone. Titrate milrinone dosage according to hemodynamic response.
Mirabegron: (Moderate) Mirabegron is a moderate CYP2D6 inhibitor. Exposure of drugs metabolized by CYP2D6 such as propranolol may be increased when co-administered with mirabegron. Propranolol is primarily metabolized by CYP2D6. Therefore, appropriate monitoring and dose adjustment may be necessary.
Mixed Grass Pollens Allergen Extract: (Moderate) Advise patients who are undergoing allergy testing or receiving maintenance allergen immunotherapy (AIT) that allergic reactions, including symptoms of anaphylaxis, may be more severe while receiving beta-blocker therapy. Risk varies by individual based on indication for therapy and history. This risk is generally considered minimal for most patients who are receiving maintenance AIT and the benefits of continuing beta-blocker therapy typically outweigh

the risks in patients with cardiovascular disease.
Modafinil: (Moderate) In vitro data indicate that modafinil is an inhibitor of CYP2C19. In theory, dosage reductions may be required for drugs that are largely eliminated via CYP2C19 metabolism such as propranolol during coadministration with modafinil.
Mometasone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Monoamine oxidase inhibitors: (Moderate) Monitor blood pressure and heart rate closely when beta-blockers are coadministered with catecholamine-depleting agents, such as monoamine oxidase inhibitors. An additive reduction in sympathetic tone may occur, which may increase the risk for hypotension or marked bradycardia-related vertigo, syncope, or postural hypotension.
Nabumetone: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Naproxen: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Naproxen; Esomeprazole: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Naproxen; Pseudoephedrine: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs. (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Nateglinide: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have

negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Nefazodone: (Minor) Although relatively infrequent, nefazodone may cause orthostatic hypotension in some patients; this effect may be additive with antihypertensive agents. Blood pressure monitoring and dosage adjustments of either drug may be necessary.
Neuromuscular blockers: (Moderate) Concomitant use of neuromuscular blockers and beta-blockers may prolong neuromuscular blockade.
Niacin, Niacinamide: (Moderate) Cutaneous vasodilation induced by niacin may become problematic if high-dose niacin is used concomitantly with other antihypertensive agents. This effect is of particular concern in the setting of acute myocardial infarction, unstable angina, or other acute hemodynamic compromise.
NiCARdipine: (Moderate) Use propranolol and nicardipine with caution due to risk for additive negative effects on heart rate, AV conduction, and/or cardiac contractility. The mean Cmax and AUC of propranolol are increased by 80% and 47%, respectively, by coadministration of nicardipine.
NIFEdipine: (Moderate) In general, concomitant therapy of nifedipine with beta-blockers is well tolerated and can even be beneficial in some cases (i.e., inhibition of nifedipine-induced reflex tachycardia by beta-blockade). Negative inotropic and/or chronotropic effects can be additive when these drugs are used in combination. Finally, angina has been reported when beta-adrenergic blocking agents are withdrawn abruptly and nifedipine therapy is initiated. A gradual downward titration of the beta-adrenergic blocking agent dosage during initiation of nifedipine therapy may minimize or eliminate this potential interaction. Hypotension and impaired cardiac performance can occur during coadministration of nifedipine with beta-blockers, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis. Monitor clinical response during coadministration; adjustment of nifedipine dosage may be needed during concurrent beta-blocker therapy.
niMODipine: (Moderate) Nimodipine, a selective calcium-channel blocker, can enhance the antihypertensive effects of beta-blockers. Although often used together, concurrent use of calcium-channel blockers and beta-blockers may result in additive hypotensive, negative inotropic, and/or bradycardic effects in some patients.
Niraparib; Abiraterone: (Moderate) Monitor blood pressure and heart rate if coadministration of propranolol with abiraterone is necessary. Propranolol is a CYP2D6 substrate and abiraterone is a CYP2D6 inhibitor. Concomitant use may result in hypotension and bradycardia.
Nirmatrelvir; Ritonavir: (Moderate) Concurrent administration of propranolol with ritonavir may result in elevated propranolol plasma concentrations. Cardiac and neurologic events have been reported when ritonavir is concurrently administered with

beta-blockers. Propranolol is metabolized by the hepatic isoenzyme CYP2D6; ritonavir is an inhibitor of this enzyme. Caution and close monitoring are advised if these drugs are administered together. Decreased beta-blocker dosage may be needed.
Nisoldipine: (Moderate) Concurrent use of nisoldipine with propranolol can be beneficial (i.e., inhibition of vasodilation-induced reflex tachycardia by beta-blockade); however, the additive negative inotropic and/or chronotropic effects can cause adverse effects, especially in patients with compromised ventricular function or conduction defects (e.g., sinus bradycardia or AV block). Pharmacokinetic interactions between nisoldipine and propranolol are variable and not significant. Propranolol attenuated the heart rate increase following the administration of immediate release nisoldipine.
Nitrates: (Moderate) Nitroglycerin can cause hypotension. This action may be additive with other agents that can cause hypotension such as antihypertensive agents or other peripheral vasodilators. Patients should be monitored more closely for hypotension if nitroglycerin, including nitroglycerin rectal ointment, is used concurrently with any beta-blockers.
Nitroglycerin: (Moderate) Nitroglycerin can cause hypotension. This action may be additive with other agents that can cause hypotension such as antihypertensive agents or other peripheral vasodilators. Patients should be monitored more closely for hypotension if nitroglycerin, including nitroglycerin rectal ointment, is used concurrently with any beta-blockers.
Nitroprusside: (Moderate) Additive hypotensive effects may occur when nitroprusside is used concomitantly with other antihypertensive agents. Dosages should be adjusted carefully, according to blood pressure.
Non-Ionic Contrast Media: (Moderate) Use caution when administering non-ionic contrast media to patients taking beta-blockers. Beta-blockers lower the threshold for and increase the severity of contrast reactions and reduce the responsiveness of treatment of hypersensitivity reactions with epinephrine.
Nonsteroidal antiinflammatory drugs: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Norepinephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Norethindrone Acetate; Ethinyl Estradiol; Ferrous fumarate: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Norethindrone; Ethinyl Estradiol: (Moderate) Monitor for increased propranolol adverse

reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Norethindrone; Ethinyl Estradiol; Ferrous fumarate: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Norgestimate; Ethinyl Estradiol: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Octreotide: (Moderate) Monitor for bradycardia during concomitant use of beta-blockers and octreotide and adjust drug dosage based on response as appropriate. Both medications may cause bradycardia and concomitant use may increase bradycardia risk.
OLANZapine: (Moderate) Olanzapine may induce orthostatic hypotension and thus enhance the effects of antihypertensive agents.
OLANZapine; FLUoxetine: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of fluoxetine as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and fluoxetine is a moderate CYP2D6 inhibitor. (Moderate) Olanzapine may induce orthostatic hypotension and thus enhance the effects of antihypertensive agents.
OLANZapine; Samidorphan: (Moderate) Olanzapine may induce orthostatic hypotension and thus enhance the effects of antihypertensive agents.
Olmesartan; amLODIPine; hydroCHLOROthiazide, HCTZ: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
Olodaterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Olopatadine; Mometasone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may

increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Omeprazole; Amoxicillin; Rifabutin: (Moderate) Rifamycins are inducers of hepatic enzymes, and may alter the pharmacokinetics of beta-blockers including propranolol. Patients should be monitored for loss of propranolol effects if rifamycins are added.
Omeprazole; Sodium Bicarbonate: (Major) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Oritavancin: (Moderate) Propranolol is metabolized by CYP2C19 and CYP2D6; oritavancin is a weak inhibitor of CYP2C19 and a weak CYP2D6 inducer. Coadministration may result in altered propranolol plasma concentrations. If these drugs are administered concurrently, blood pressure should be monitored closely.
Osilodrostat: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of osilodrostat as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and osilodrostat is a moderate CYP1A2 inhibitor.
Oxaprozin: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Oxymetazoline: (Major) The vasoconstricting actions of oxymetazoline, an alpha adrenergic agonist, may reduce the antihypertensive effects produced by beta-blockers. If these drugs are used together, closely monitor for changes in blood pressure.
Ozanimod: (Moderate) Ozanimod may cause bradycardia and AV-conduction delays, which may be enhanced with the concomitant use of beta-blockers. If a calcium channel blocker that slows heart rate/cardiac conduction is also prescribed with ozanimod and a beta-blocker, a cardiologist should be consulted due to the likelyhood of additive effects.
Paliperidone: (Moderate) Paliperidone may cause orthostatic hypotension, thereby enhancing the hypotensive effects of antihypertensive agents. Orthostatic vital signs should be monitored in patients receiving paliperidone and beta-adrenergic blockers who are susceptible to hypotension.
Paltusotine: (Moderate) Monitor for bradycardia and other cardiac conduction abnormalities during the concomitant use of paltusotine and beta-blocker; a beta-blocker dosage reduction may be required based on response. Both medications have been associated with bradycardia and concomitant use may increase risk.
PARoxetine: (Minor) Paroxetine impairs metabolism of the hepatic CYP2D6 isoenzyme pathway at therapeutic doses, resulting in substantial increases in concentrations of other drugs metabolized via the same pathway, including propranolol. Clinicians should use paroxetine cautiously with propranolol; downward dose adjustments of the beta-blocker may be required if paroxetine is initiated; alternatively an upward dose

adjustment of the beta blocker may be needed if paroxetine is discontinued. Patients should be advised to report increased effects of these medications, including hypotension or increased dizziness to their health care professional.
Pasireotide: (Major) Pasireotide may cause a decrease in heart rate. Closely monitor patients who are also taking drugs associated with bradycardia such as beta-blockers. Dose adjustments of beta-blockers may be necessary.
Peanut (Arachis hypogaea) Allergen Powder: (Moderate) Advise patients who are undergoing allergy testing or receiving maintenance allergen immunotherapy (AIT) that allergic reactions, including symptoms of anaphylaxis, may be more severe while receiving beta-blocker therapy. Risk varies by individual based on indication for therapy and history. This risk is generally considered minimal for most patients who are receiving maintenance AIT and the benefits of continuing beta-blocker therapy typically outweigh the risks in patients with cardiovascular disease.
Peginterferon Alfa-2b: (Moderate) Monitor for adverse effects associated with increased exposure to propranolol if peginterferon alfa-2b is coadministered. Peginterferon alfa-2b is a CYP2D6 inhibitor, while propranolol is a CYP2D6 substrate.
Pentoxifylline: (Moderate) Pentoxifylline has been used concurrently with antihypertensive drugs (beta blockers, diuretics) without observed problems. Small decreases in blood pressure have been observed in some patients treated with pentoxifylline; periodic systemic blood pressure monitoring is recommended for patients receiving concomitant antihypertensives. If indicated, dosage of the antihypertensive agents should be reduced.
Perindopril; amLODIPine: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
Perphenazine: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of perphenazine as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and perphenazine is weak CYP2D6 inhibitor.
Perphenazine; Amitriptyline: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of perphenazine as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and perphenazine is weak CYP2D6 inhibitor.
Phendimetrazine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Phenelzine: (Moderate) Monitor blood pressure and heart rate closely when beta-blockers are coadministered with catecholamine-depleting agents, such as monoamine

oxidase inhibitors. An additive reduction in sympathetic tone may occur, which may increase the risk for hypotension or marked bradycardia-related vertigo, syncope, or postural hypotension.
Phenoxybenzamine: (Moderate) Orthostatic hypotension may be more likely in some patients if beta-blockers are coadministered with alpha-blockers. Monitor blood pressure and for orthostasis, especially at medication initiation.
Phentermine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Phentermine; Topiramate: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Phentolamine: (Moderate) Orthostatic hypotension may be more likely in some patients if beta-blockers are coadministered with alpha-blockers. Monitor blood pressure and for orthostasis, especially at medication initiation.
Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Phenytoin: (Minor) Phenytoin is an inducer of hepatic enzymes, and has been shown to accelerate the hepatic metabolism of propranolol.
Pilocarpine: (Moderate) Systemically administered pilocarpine (e.g., when used for the treatment of xerostomia or xerophthalmia) should be administered with caution in patients taking beta-blockers because of the possibility of cardiac conduction disturbances. The risk of conduction disturbances with beta-blockers and ophthalmically administered pilocarpine is low.
Pioglitazone: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do

not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Pioglitazone; Glimepiride: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present. (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Pioglitazone; metFORMIN: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of

hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Piroxicam: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Ponesimod: (Moderate) Monitor for decreases in heart rate if concomitant use of ponesimod and beta-blockers is necessary. Consider a temporary interruption in beta-blocker therapy before initiating ponesimod in patients with a resting heart rate less than or equal to 55 bpm. Beta-blocker treatment can be initiated in patients receiving stable doses of ponesimod. Concomitant use of another beta-blocker with ponesimod resulted in a mean decrease in heart rate of 12.4 bpm after the first dose of ponesimod and 7.4 bpm after beginning maintenance ponesimod.
Pramlintide: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are

present.
Prazosin: (Moderate) Orthostatic hypotension may be more likely in some patients if beta-blockers are coadministered with alpha-blockers. Monitor blood pressure and for orthostasis, especially at medication initiation.
prednisoLONE: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
predniSONE: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Prilocaine: (Moderate) Local anesthetics may cause additive hypotension in combination with antihypertensive agents.
Prilocaine; EPINEPHrine: (Moderate) Local anesthetics may cause additive hypotension in combination with antihypertensive agents. (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Primidone: (Moderate) Barbiturates can enhance the hepatic metabolism of beta-blockers that are significantly metabolized by the liver, such as propranolol. Clinicians should monitor patients for loss of beta-blockade.
Procainamide: (Major) High or toxic concentrations of procainamide may prolong AV nodal conduction time or induce AV block; these effects could be additive with the pharmacologic actions of beta-blockers, like propranolol. In general, patients receiving combined therapy with procainamide and beta-blockers should be monitored for potential bradycardia, AV block, and/or hypotension. Procainamide's elimination half-life was not significantly changed when administered concomitantly with propranolol.
Promethazine; Phenylephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Propafenone: (Major) Pharmacologically, beta-blockers, like propranolol, cause AV nodal conduction depression and additive effects are possible when used in combination with propafenone. When used together, AV block can occur. Additionally, propafenone, a CYP2D6 inhibitor, appears to inhibit the metabolism of propranolol. Coadministration of propafenone with propranolol increases the plasma concentrations and prolongs the elimination half-life of propranolol; these affects were associated with a 15% decrease in diastolic blood pressure. Patients should be monitored closely and a reduction in the dosage of propranolol may be indicated.
Propofol: (Major) General anesthetics can potentiate the antihypertensive effects of beta-blockers and can produce prolonged hypotension. Beta-blockers may be continued

during general anesthesia as long as the patient is monitored for cardiac depressant and hypotensive effects.
Propylthiouracil, PTU: (Minor) Hyperthyroidism may cause increased clearance of beta blockers that possess a high extraction ratio. A dose reduction of some beta-blockers may be needed when a hyperthyroid patient treated with methimazole becomes euthyroid.
Pseudoephedrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Pseudoephedrine; Triprolidine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
pyRIDostigmine: (Minor) Concomitant use of beta-blockers with pyridostigmine may exacerbate pyridostigmine-induced bradycardia. Monitor patients closely for adverse reactions during concurrent therapy.
quiNIDine: (Major) Patients receiving combined therapy with quinidine and propranolol should be monitored for potential hypotension, orthostasis, bradycardia and/or AV block and heart failure, Reduce the beta-blocker dosage if necessary. Quinidine may have additive effects (e.g., reduced heart rate, hypotension) on cardiovascular parameters when used together with beta-blockers, such as propranolol. Quinidine is a known inhibitor of CYP2D6, and may additionally impair the hepatic clearance of propanolol (CYP2D6 substrate); patients should be monitored for excess beta-blockade.
quiNINE: (Minor) Propranolol is significantly metabolized by CYP2D6 isoenzymes. CYP2D6 inhibitors, such as quinine, could theoretically impair propranolol metabolism; the clinical significance of such interactions is unknown.
Racepinephrine: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Ranolazine: (Moderate) Propranolol is metabolized by CYP2D6 isoenzymes. CYP2D6 inhibitors, such as ranolazine, could theoretically impair propranolol metabolism. Lower doses of some CYP2D6 substrates than are usually prescribed may be needed during therapy with ranolazine; monitor therapeutic response during coadministration.
Rasagiline: (Moderate) Additive hypotensive effects may be seen when monoamine oxidase inhibitors (MAOIs) are combined with antihypertensives. Careful monitoring of blood pressure is suggested during concurrent therapy of MAOIs with beta-blockers. Limited data suggest that bradycardia is worsened when MAOIs are administered to patients receiving beta-blockers. Although the sinus bradycardia observed was not

severe, until more data are available, clinicians should use MAOIs cautiously in patients receiving beta-blockers. Patients should be instructed to rise slowly from a sitting position, and to report syncope or changes in blood pressure or heart rate to their health care provider.
Regular Insulin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Regular Insulin; Isophane Insulin (NPH): (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Remifentanil: (Moderate) The risk of significant hypotension and/or bradycardia during therapy with remifentanil may be increased in patients receiving beta-blockers or

calcium-channel blockers due to additive hypotensive effects.
Repaglinide: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Rifabutin: (Moderate) Rifamycins are inducers of hepatic enzymes, and may alter the pharmacokinetics of beta-blockers including propranolol. Patients should be monitored for loss of propranolol effects if rifamycins are added.
rifAMPin: (Moderate) Rifamycins are inducers of hepatic enzymes, and may alter the pharmacokinetics of beta-blockers including propranolol. Patients should be monitored for loss of propranolol effects if rifamycins are added.
Rifamycins: (Moderate) Rifamycins are inducers of hepatic enzymes, and may alter the pharmacokinetics of beta-blockers including propranolol. Patients should be monitored for loss of propranolol effects if rifamycins are added.
Rifapentine: (Moderate) Rifamycins are inducers of hepatic enzymes, and may alter the pharmacokinetics of beta-blockers including propranolol. Patients should be monitored for loss of propranolol effects if rifamycins are added.
risperiDONE: (Moderate) Risperidone may induce orthostatic hypotension and thus enhance the hypotensive effects of propranolol. Lower initial doses or slower dose titration of risperidone may be necessary in patients receiving propranolol concomitantly.
Ritlecitinib: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ritlecitinib as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ritlecitinib is moderate CYP1A2 inhibitor.
Ritonavir: (Moderate) Concurrent administration of propranolol with ritonavir may result in elevated propranolol plasma concentrations. Cardiac and neurologic events have

been reported when ritonavir is concurrently administered with beta-blockers. Propranolol is metabolized by the hepatic isoenzyme CYP2D6; ritonavir is an inhibitor of this enzyme. Caution and close monitoring are advised if these drugs are administered together. Decreased beta-blocker dosage may be needed.
Rivastigmine: (Moderate) The increase in vagal tone induced by some cholinesterase inhibitors may produce bradycardia, hypotension, or syncope. The vagotonic effect of these drugs may theoretically be increased when given with other medications known to cause bradycardia such as beta-blockers.
Rizatriptan: (Major) Recommendations for concomitant use of rizatriptan and propranolol vary by dosage form. Concomitant use of rizatriptan oral film and propranolol is contraindicated, and a dosage reduction may be necessary for rizatriptan oral tablets. Limit the rizatriptan dose to 5 mg/dose and 15 mg/24 hours for adults receiving rizatriptan tablets. Avoid use of rizatriptan tablets in pediatric patients weighing less than 40 kg and limit the rizatriptan dose to 5 mg/24 hours in pediatric patients weighing 40 kg or more. Rizatriptan oral film is contraindicated with propranolol because a dose adjustment is not possible. Coadministration has been observed to increase rizatriptan overall exposure by 70% without affecting concentrations of the active N-monodesmethyl metabolite.
Rocuronium: (Moderate) Concomitant use of neuromuscular blockers and beta-blockers may prolong neuromuscular blockade.
Rolapitant: (Major) Use caution if propranolol and rolapitant are used concurrently, and monitor for propranolol-related adverse effects, including bradycardia. Propranolol is a CYP2D6 substrate that is individually dose-titrated, and rolapitant is a moderate CYP2D6 inhibitor; the inhibitory effect of rolapitant is expected to persist beyond 28 days for an unknown duration. Exposure to another CYP2D6 substrate, following a single dose of rolapitant increased about 3-fold on Days 8 and Day 22. The inhibition of CYP2D6 persisted on Day 28 with a 2.3-fold increase in the CYP2D6 substrate concentrations, the last time point measured.
ROPivacaine: (Moderate) Local anesthetics may cause additive hypotension in combination with antihypertensive agents.
Rosiglitazone: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A

selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Rucaparib: (Moderate) Monitor patients for hypotension and bradycardia if coadministration of propranolol with rucaparib is necessary. Propranolol is a CYP1A2 substrate and rucaparib is a moderate CYP1A2 inhibitor. Concomitant use of CYP1A2 inhibitors may increase exposure to propranolol.
Salmeterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Salsalate: (Moderate) Concurrent use of beta-blockers with salsalate and other salicylates may result in loss of antihypertensive activity due to inhibition of renal prostaglandins and thus, salt and water retention and decreased renal blood flow.
sAXagliptin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.

Segesterone Acetate; Ethinyl Estradiol: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of ethinyl estradiol as concurrent use may increase propranolol exposure. Propranolol is a CYP1A2 substrate and ethinyl estradiol is moderate CYP1A2 inhibitor.
Semaglutide: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Serdexmethylphenidate; Dexmethylphenidate: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Sevoflurane: (Major) General anesthetics can potentiate the antihypertensive effects of beta-blockers and can produce prolonged hypotension. Beta-blockers may be continued during general anesthesia as long as the patient is monitored for cardiac depressant and hypotensive effects.
SGLT2 Inhibitors: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate

for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Short Ragweed Pollen Allergen Extract: (Moderate) Advise patients who are undergoing allergy testing or receiving maintenance allergen immunotherapy (AIT) that allergic reactions, including symptoms of anaphylaxis, may be more severe while receiving beta-blocker therapy. Risk varies by individual based on indication for therapy and history. This risk is generally considered minimal for most patients who are receiving maintenance AIT and the benefits of continuing beta-blocker therapy typically outweigh the risks in patients with cardiovascular disease.
Silodosin: (Moderate) During clinical trials with silodosin, the incidence of dizziness and orthostatic hypotension was higher in patients receiving concomitant antihypertensive treatment. Thus, caution is advisable when silodosin is administered with antihypertensive agents. In addition, increased concentrations of silodosin may occur if it is coadministered with carvedilol; exercise caution. Carvedilol is a P-glycoprotein (P-gp) inhibitor and silodosin is a P-gp substrate.
Simvastatin: (Minor) After administration of single doses of simvastatin and propranolol, there was a significant decrease in mean Cmax, with no change in AUC, of simvastatin. The clinical significance of this interaction is unknown. Monitor for potential reduced cholesterol-lowering efficacy when propranolol is coadministered with niacin; simvastatin.
Siponimod: (Moderate) Monitor for significant bradycardia with coadministration of siponimod and beta-blockers, as additive lowering effects on heart rate may occur; temporary interruption of beta-blocker treatment may be necessary prior to siponimod initiation. Beta-blocker treatment can be initiated in patients receiving stable doses of siponimod.
SITagliptin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate

for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Sodium Bicarbonate: (Major) Antacids may reduce the absorption of propranolol. The need to stagger doses of propranolol has not been established, but may be prudent. Monitor clinical response, and adjust propranolol dosage if needed to attain therapeutic goals.
Sodium Nitrite; Sodium Thiosulfate: (Moderate) Sodium nitrite causes vasodilation resulting in hypotension. Antihypertensive agents and other hypotension-producing agents such as vasodilators and tricyclic antidepressants can cause additive hypotensive or orthostatic effects.
Sotagliflozin: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Succinylcholine: (Moderate) Concomitant use of neuromuscular blockers and beta-blockers may prolong neuromuscular blockade.
SUFentanil: (Moderate) The incidence and degree of bradycardia and hypotension during induction with sufentanil may be increased in patients receiving beta-blockers.
Sulfacetamide; Sulfur: (Moderate) Cutaneous vasodilation induced by niacin may become problematic if high-dose niacin is used concomitantly with other antihypertensive agents.
Sulfonylureas: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers

inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Sulindac: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
SUMAtriptan; Naproxen: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Sympathomimetics: (Moderate) Monitor hemodynamic parameters and for loss of efficacy during concomitant sympathomimetic agent and beta-blocker use; dosage adjustments may be necessary. Concomitant use may antagonize the cardiovascular effects of either drug.
Tamsulosin: (Minor) Tamsulosin did not potentiate the hypotensive effects of atenolol. However, since the symptoms of orthostasis are reported more frequently in tamsulosin-treated vs. placebo patients, there is a potential risk of enhanced hypotensive effects when co-administered with antihypertensive agents.
Tasimelteon: (Moderate) Advise patients to administer the beta-blocker in the morning if tasimelteon is used concomitantly. Nighttime administration of a beta-blocker may reduce the efficacy of tasimelteon by decreasing the production of melatonin via inhibition of beta1 receptors.
Telmisartan; amLODIPine: (Moderate) Coadministration of amlodipine and beta-blockers can reduce angina and improve exercise tolerance. When these drugs are given together, however, hypotension and impaired cardiac performance can occur, especially in patients with left ventricular dysfunction, cardiac arrhythmias, or aortic stenosis.
Terazosin: (Moderate) Orthostatic hypotension may be more likely in some patients if beta-blockers are coadministered with alpha-blockers. Monitor blood pressure and for orthostasis, especially at medication initiation.

Terbinafine: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of terbinafine as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 substrate and terbinafine is strong CYP2D6 inhibitor.
Terbutaline: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Tetrabenazine: (Moderate) Tetrabenazine may induce orthostatic hypotension and thus enhance the hypotensive effects of antihypertensive agents. Lower initial doses or slower dose titration of tetrabenazine may be necessary in patients receiving antihypertensive agents concomitantly.
Tetracaine: (Moderate) Local anesthetics may cause additive hypotension in combination with antihypertensive agents. Use caution with the concomitant use of tetracaine and antihypertensive agents.
Thalidomide: (Moderate) Thalidomide and other agents that slow cardiac conduction such as beta-blockers should be used cautiously due to the potential for additive bradycardia.
Theophylline, Aminophylline: (Major) Propranolol may significantly decrease aminophylline clearance by inhibiting CYP1A2. In some patients, theophylline levels can increase up to 100%. On average, co-administration of theophylline with propranolol decreases theophylline oral clearance by 30% to 52%. If aminophylline is being initiated in a patient who is already taking a drug that inhibits its clearance, the dose required to achieve a therapeutic serum theophylline concentration will be smaller. Patients should be closely monitored for toxicity. Serum theophylline concentrations should be monitored. Because propranolol is non-selective, the beta-2 blocking activity may reduce the effectiveness of aminophylline and other treatments for asthma or COPD. Discontinuation of a concomitant drug that inhibits aminophylline clearance will result in decreased serum theophylline concentrations, unless the aminophylline dose is appropriately increased. (Major) Propranolol may significantly decrease theophylline clearance by inhibiting CYP1A2. In some patients, theophylline levels can increase up to 100%. On average, co-administration of theophylline with propranolol decreases theophylline oral clearance by 30% to 52%. If theophylline is being initiated in a patient who is already taking a drug that inhibits its clearance, the dose of theophylline required to achieve a therapeutic theophylline concentration will be smaller. Patients should be

closely monitored for toxicity. Serum theophylline concentrations should be monitored. Because propranolol is non-selective, the beta-2 blocking activity may reduce the effectiveness of theophylline and other treatments for asthma or COPD. Discontinuation of a concomitant drug that inhibits theophylline clearance will result in decreased theophylline concentrations, unless the theophylline dose is appropriately increased.
Thiazolidinediones: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
Thioridazine: (Contraindicated) Coadministration of thioridazine and propranolol is contraindicated due to the potential for increased thioridazine exposure. Increased plasma concentrations of thioridazine are expected to increase the prolongation of the QTc interval associated with thioridazine and may increase the risk of serious, potentially fatal, cardiac arrhythmias, such as torsade de pointes type arrhythmias. Propranolol has been observed to increase thioridazine exposure by 370%.
Thiothixene: (Moderate) Thiothixene should be used cautiously in patients receiving antihypertensive agents. Additive hypotensive effects are possible.
Thyroid hormones: (Minor) Because thyroid hormones cause cardiac stimulation including increased heart rate and increased contractility, the effects of beta-blockers may be reduced by thyroid hormones. The reduction of effects may be especially evident when a patient goes from a hypothyroid to a euthyroid state or when excessive amounts of thyroid hormone is given to the patient.
Timothy Grass Pollen Allergen Extract: (Moderate) Advise patients who are undergoing allergy testing or receiving maintenance allergen immunotherapy (AIT) that allergic reactions, including symptoms of anaphylaxis, may be more severe while receiving beta-blocker therapy. Risk varies by individual based on indication for therapy and history. This risk is generally considered minimal for most patients who are receiving

maintenance AIT and the benefits of continuing beta-blocker therapy typically outweigh the risks in patients with cardiovascular disease.
Tiotropium; Olodaterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Tirzepatide: (Moderate) Increased frequency of blood glucose monitoring may be required when a beta blocker is given with antidiabetic agents. Since beta blockers inhibit the release of catecholamines, these medications may hide symptoms of hypoglycemia such as tremor, tachycardia, and blood pressure changes. Other symptoms, like headache, dizziness, nervousness, mood changes, or hunger are not blunted. Beta-blockers also exert complex actions on the body's ability to regulate blood glucose. Some beta-blockers, particularly non-selective beta-blockers such as propranolol, have been noted to potentiate insulin-induced hypoglycemia and a delay in recovery of blood glucose to normal levels. Hyperglycemia has been reported as well and is possibly due to beta-2 receptor blockade in the beta cells of the pancreas. A selective beta-blocker may be preferred in patients with diabetes mellitus, if appropriate for the patient's condition. Selective beta-blockers, such as atenolol or metoprolol, do not appear to potentiate insulin-induced hypoglycemia. While beta-blockers may have negative effects on glycemic control, they reduce the risk of cardiovascular disease and stroke in patients with diabetes and their use should not be avoided in patients with compelling indications for beta-blocker therapy when no other contraindications are present.
tiZANidine: (Moderate) Concurrent use of tizanidine with antihypertensive agents can result in significant hypotension. Caution is advised when tizanidine is to be used in patients receiving concurrent antihypertensive therapy.
Tobacco: (Major) Advise patients to avoid smoking tobacco while taking propranolol. Smoking tobacco has been observed to decrease propranolol exposure by approximately 50% and may decrease efficacy.
Tolmetin: (Moderate) Monitor blood pressure during concomitant beta-blocker and nonsteroidal anti-inflammatory drug (NSAID) use. The antihypertensive effect of beta-blockers may be diminished by NSAIDs.
Trandolapril; Verapamil: (Major) Intravenous propranolol is contraindicated with intravenous verapamil use in close proximity (within a few hours). Fatal cardiac arrests have occurred in patients receiving intravenous beta-blockers and intravenous calcium

channel blockers. Use oral propranolol and oral verapamil with caution and close monitoring due to risk for additive negative effects on heart rate, AV conduction, and/or cardiac contractility. There have been reports of excess bradycardia and AV block, including complete heart block, when beta-blockers and verapamil have been used for the treatment of hypertension. A decrease in propranolol clearance has been observed when administered concomitantly with verapamil.
Tranylcypromine: (Moderate) Monitor blood pressure and heart rate closely when beta-blockers are coadministered with catecholamine-depleting agents, such as monoamine oxidase inhibitors. An additive reduction in sympathetic tone may occur, which may increase the risk for hypotension or marked bradycardia-related vertigo, syncope, or postural hypotension.
traZODone: (Minor) Due to additive hypotensive effects, patients receiving antihypertensive agents concurrently with trazodone may have excessive hypotension. Decreased dosage of the antihypertensive agent may be required when given with trazodone.
Triamcinolone: (Moderate) Monitor blood sugar during concomitant corticosteroid and propranolol use due to risk for hypoglycemia. Concurrent use may increase risk of hypoglycemia because of loss of the counter-regulatory cortisol response.
Trifluoperazine: (Moderate) Monitor for an increase in trifluoperazine and propranolol-related adverse effects during concomitant use. The concentrations of both medications may increase.
Umeclidinium; Vilanterol: (Moderate) Beta-blockers will block the pulmonary effects of inhaled beta-agonists, and in some cases may exacerbate bronchospasm in patients with reactive airways. Beta-agonists can sometimes increase heart rate or have other cardiovascular effects, particularly when used in high doses or if hypokalemia is present. Use of a beta-1-selective (cardioselective) beta blocker is recommended whenever possible when this combination of drugs must be used together. Monitor the patient's lung and cardiovascular status closely. Beta-agonists and beta-blockers are pharmacologic opposites and will counteract each other to some extent when given concomitantly, especially when non-cardioselective beta blockers are used.
Vecuronium: (Moderate) Concomitant use of neuromuscular blockers and beta-blockers may prolong neuromuscular blockade.
Vemurafenib: (Moderate) Propranolol is significantly metabolized by CYP2D6 and secondarily by the CYP1A2 isoenzymes. CYP2D6 and CYP1A2 inhibitors, such as vemurafenib, could theoretically impair propranolol metabolism. The clinical significance of such interactions is unknown.
Verapamil: (Major) Intravenous propranolol is contraindicated with intravenous verapamil use in close proximity (within a few hours). Fatal cardiac arrests have occurred in patients receiving intravenous beta-blockers and intravenous calcium channel blockers. Use oral propranolol and oral verapamil with caution and close monitoring due

to risk for additive negative effects on heart rate, AV conduction, and/or cardiac contractility. There have been reports of excess bradycardia and AV block, including complete heart block, when beta-blockers and verapamil have been used for the treatment of hypertension. A decrease in propranolol clearance has been observed when administered concomitantly with verapamil.
Viloxazine: (Moderate) Monitor for increased propranolol adverse reactions, including bradycardia and hypotension, during coadministration of viloxazine as concurrent use may increase propranolol exposure. Propranolol is a CYP2D6 and CYP1A2 substrate and viloxazine is a strong 1A2 inhibitor and a weak CYP2D6 inhibitor.
Vitamin B Complex Supplements: (Moderate) Cutaneous vasodilation induced by niacin may become problematic if high-dose niacin is used concomitantly with other antihypertensive agents. This effect is of particular concern in the setting of acute myocardial infarction, unstable angina, or other acute hemodynamic compromise.
Voriconazole: (Moderate) Voriconazole is metabolized by CYP3A4 and, theoretically, inhibitors of CYP3A4, such as propranolol, could lead to increased serum levels of voriconazole.
Warfarin: (Moderate) Propranolol has been shown to increase warfarin AUC, and concurrent increases in INR values have been reported. Patients should be monitored for changes in anticoagulation parameters during concurrent therapy with propranolol and warfarin.
Zileuton: (Moderate) Concomitant administration of zileuton and propranolol results in a significant increase in propranolol serum concentrations, AUC, and elimination half-life. Bradycardia is also potentiated by the drug combination. Clinicians should monitor vital signs carefully if zileuton is added to a regimen containing propranolol and adjust dosages as needed.
Ziprasidone: (Minor) Ziprasidone is a moderate antagonist of alpha-1 receptors and may cause orthostatic hypotension with or without tachycardia, dizziness, or syncope. Additive hypotensive effects are possible if ziprasidone is used concurrently with antihypertensive agents.
ZOLMitriptan: (Minor) Periodically monitor blood pressure and for zolmitriptan-related side effects in patients who regularly use zolmitriptan and are taking propranolol. Rarely, a patient might experience an increase in dose-related common side effects of zolmitriptan, such as dizziness, nausea or drowsiness. No dosage adjustment of zolmitriptan appears to be needed. During pharmacokinetic studies, the Cmax and AUC of zolmitriptan increased 1.5-fold after 1 week of dosing with propranolol. Cmax and AUC of the active N-desmethyl metabolite of zolmitriptan were reduced by 30% and 15%, respectively. However, in clinical trials, the efficacy of zolmitriptan was not affected by the concurrent use of common migraine prophylactic drugs (e.g., propranolol). There were no interactive effects on blood pressure or pulse rate. The interaction should not be significant for most patients.

Adverse Reaction

General Information

The adverse effects of propranolol are generally mild and temporary; they usually occur at the onset of therapy and diminish over time.

AV block, bradycardia, hypotension, paresthesias

Most adverse effects of propranolol are manifestations of its therapeutic effect. Sinus bradycardia and hypotension are rarely serious and can be reversed with IV atropine if necessary. AV block, secondary to depressed conduction at the AV node, may necessitate sympathomimetic and/or pressor therapy or use of a temporary pacemaker. Paresthesias of the hands and arterial insufficiency, usually of the Raynaud type, also have been reported. Peripheral coldness was reported in 7% to 8% of infants during infantile hemangioma clinical trials.

heart failure

Heart failure has been reported with the use of propranolol. Congestive heart failure is more likely to occur in patients with preexisting left ventricular dysfunction and usually will respond to discontinuation of propranolol therapy. Elevations of blood urea nitrogen also have been reported in patients with severe heart failure who are taking propranolol.

agitation, confusion, depression, dizziness, drowsiness, emotional lability, fatigue, hallucinations, insomnia, irritability, lethargy, memory impairment, nightmares, visual impairment, weakness

Dizziness was reported in 4% to 7% and fatigue in 5% to 7% of patients with hypertension who received propranolol extended-release capsules in clinical trials. Additional adverse CNS effects reported with propranolol therapy include lethargy, weakness, catatonia, an acute reversible syndrome characterized by disorientation to time and place, visual impairment (reported as visual disturbances), hallucinations, short-term memory impairment, emotional lability, slightly clouded sensorium (e.g., confusion), vivid dreams (e.g., nightmares), decreased performance on neuropsychometrics, and depression manifested by insomnia. With immediate-release formulations, fatigue, lethargy, and vivid dreams appear to be dose-related. During clinical trials of propranolol oral solution for infantile hemangioma, unspecified sleep disorders (16% to 17.5%), nightmares, (2% to 6%), agitation (4.5% to 8.5%), irritability (1% to 5.5%), and drowsiness (0.9% to 5%) were among the most common adverse events.

abdominal pain, anorexia, colitis, constipation, diarrhea, nausea, thrombosis, vomiting

Gastrointestinal adverse effects reported with propranolol use include nausea, vomiting, diarrhea, constipation, abdominal pain (cramping), epigastric distress, mesenteric arterial thrombosis, and ischemic colitis. During clinical trials of propranolol oral solution for infantile hemangioma, diarrhea (4.5% to 6%), anorexia (2.5% to 3.5%), abdominal pain (0.5% to 3.5%), and vomiting were commonly reported.

bronchospasm, cough, dyspnea, infection

Patients with preexisting bronchospastic disease are at high risk for exacerbation of asthma, dyspnea, or bronchospasm when treated with propranolol. Bronchospasm has been reported coincident with propranolol therapy in pediatric patients. During infantile hemangioma clinical trials, aggravated respiratory tract infection such as bronchitis (8% to 13%) and bronchiolitis associated with cough and a febrile response were among the most frequently reported adverse events. In the event of a lower respiratory tract infection associated with dyspnea and wheezing, propranolol therapy should be interrupted if the clinical condition allows (e.g. migraine prophylaxis, treatment for hemangioma).

coma, diabetes mellitus, hyperglycemia, hypoglycemia, seizures

Beta-blockers may inhibit catecholamine-induced glycogenolysis, gluconeogenesis, and lipolysis, predisposing to hypoglycemia. Additionally, beta-blockers can also mask signs of hypoglycemia (e.g., tachycardia) and increase the risk for severe or prolonged hypoglycemia at any time during treatment especially in persons with diabetes mellitus, pediatric patients, and persons who are fasting (i.e., surgery, not eating regularly, or are vomiting). Hypoglycemia may present in the form of seizures, lethargy, or coma. Instruct caregivers and patients to seek immediate medical treatment if severe hypoglycemia occurs. To reduce the risk of hypoglycemia in pediatric patients, administer propranolol shortly before or after feeds and maintain a consistent feeding schedule. Carefully monitor vital signs and blood glucose concentration during drug initiation and dosage escalation. Advise caregivers with special instructions for dosage adjustment or discontinuation during intercurrent illness (if clinical condition allows) and alternative dietary recommendations. Beta-blockers may also inhibit insulin secretion through blockade of beta-2-receptors on pancreatic islet cells, which may cause hyperglycemia or reduce insulin secretion in response to hyperglycemia; adjust the dose of antidiabetic drugs as necessary. In addition to acute blood glucose effects, beta-blockers have been

shown to increase the risk of developing diabetes mellitus in adult hypertensive patients.

hypertriglyceridemia

Some beta-blockers have been shown to cause hypertriglyceridemia and decrease plasma HDL levels during therapy. The clinical implications of these effects, in light of other cardiovascular advantages of beta-blocker therapy, are not known. Hypertriglyceridemia is not reported as and adverse effect by the manufacturer of propranolol.

myopathy

Propranolol therapy has been associated with isolated reports of exacerbation of myopathy and myotonia. Use caution in patients with pre-existing skeletal muscle disease.

elevated hepatic enzymes, hyperkalemia

In hypertensive patients, propranolol has been associated with hyperkalemia and elevated hepatic enzymes (e.g., serum transaminases and alkaline phosphatase).

impotence (erectile dysfunction), infertility, penile fibrosis, Peyronie disease

Impotence (erectile dysfunction) has been reported with various beta-blocker therapies, including propranolol. Peyronie disease (an abnormal curvature of the penis during erection with penile fibrosis) has also been reported with propranolol, but it is considered to be very rare. In animal studies, propranolol has been shown to inhibit spermatogenesis, which may affect male fertility (infertility).

agranulocytosis, thrombotic thrombocytopenic purpura (TTP)

Rare but severe hematologic side effects, such as agranulocytosis, have been reported with propranolol therapy. Non-thrombocytopenic purpura and thrombotic thrombocytopenic purpura (TTP) also have been reported.

alopecia, anaphylactoid reactions, erythema multiforme, exfoliative dermatitis, fever, laryngospasm, pharyngitis, pruritus, psoriaform rash, psoriasis, Stevens-Johnson syndrome, toxic epidermal necrolysis, urticaria, xerophthalmia, xerosis

Hypersensitivity reactions, including anaphylactic/anaphylactoid reactions, pharyngitis

and agranulocytosis, erythematous rash, urticaria, fever combined with aching and sore throat, laryngospasm, and respiratory distress have been reported with propranolol use. Dermatologic reactions with beta-blockers are usually mild and transient. Some of these reactions include pruritus, reversible alopecia, xerosis, xerophthalmia, psoriaform rash, psoriasis, dermatitis psoriasiform, and exfoliative dermatitis. In addition, more serious dermatologic reactions have been reported including Stevens-Johnson Syndrome, toxic epidermal necrolysis, and erythema multiforme.

lupus-like symptoms

Lupus-like symptoms and systemic lupus erythematosus have been reported with the use of propranolol.

diaphoresis, headache, hypertension, palpitations, sinus tachycardia, tremor, withdrawal

Withdrawal symptoms, including headache, diaphoresis, palpitations, sinus tachycardia, tremor, and hypertension, have been associated with abrupt discontinuation of beta-blockers in hypertensive patients. Gradual tapering and/or prolonged administration of small doses of propranolol prior to complete cessation may prevent these symptoms.

diagnostic test interference, laboratory test interference

Beta-receptor blockade can cause reduction of intraocular pressure. Advise individuals that propranolol may cause diagnostic test interference with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure. Additionally, propranolol may cause laboratory test interference with thyroid function tests, increasing T4 and reverse T3 and decreasing T3.

Description

Propranolol is the prototype of the beta-adrenergic receptor antagonists. It is a competitive, nonselective beta-blocker without intrinsic sympathomimetic activity, similar to nadolol. Although propranolol has membrane-stabilizing effects on the action potential, these effects are clinically insignificant except in overdose situations. Propranolol is a racemic compound, with only its l-isomer having any adrenergic blocking activity. Propranolol was first approved by the FDA in 1967; an extended-release formulation designed for bedtime-dosing was approved in March 2003. Hemangeol, an oral solution specifically approved for treatment of proliferating infantile hemangiomas, was approved by the FDA in March 2014.

Mechanism Of Action

Mechanism of Action: Like other beta-adrenergic antagonists, propranolol competes with adrenergic neurotransmitters (e.g., catecholamines) for binding at sympathetic receptor sites. Similar to atenolol and metoprolol, propranolol blocks sympathetic stimulation mediated by beta1-adrenergic receptors in the heart and vascular smooth muscle. Pharmacodynamic consequences of beta1-receptor blockade include a decrease in both resting and exercise heart rate and cardiac output, and a decrease in both systolic and diastolic blood pressure. Propranolol may reduce reflex orthostatic hypotension. The fall in cardiac output induced by beta1 effects is often countered by a moderate reflex increase in peripheral vascular resistance that can be magnified by beta2 blockade (unmasked alpha stimulation). As a result, nonselective beta-blocking agents can produce a more modest decrease in (diastolic) blood pressure compared with selective beta1-antagonists. In addition, propranolol also can competitively block beta2-adrenergic responses in the bronchial muscles, potentially inducing bronchospasm.Actions that make propranolol useful in treating hypertension include a negative chronotropic effect that decreases heart rate at rest and after exercise; a negative inotropic effect that decreases cardiac output; reduction of sympathetic outflow from the CNS; and suppression of renin release from the kidneys. Thus, propranolol, like other beta-blockers, affects blood pressure via multiple mechanisms. In general, beta-blockers without intrinsic sympathomimetic activity (ISA) exert detrimental effects on LVH and the lipid profile, and cause sexual dysfunction.Actions that make propranolol useful in treating hypertension also apply to managing chronic stable angina. The reduction in myocardial oxygen demand induced by propranolol results in decreases in the frequency of anginal attacks and requirements of nitrate, and increases exercise tolerance. Other postulated anti-anginal actions include an increase in oxygen delivery to tissues, due to propranolol-induced lowering of hemoglobin's affinity for oxygen, and a reduction of platelet aggregation, postulated to be related to interference with calcium ion flux.Propranolol has been used to treat portal hypertension and to prevent bleeding of esophageal varices. Nonselective beta-blockers decrease portal venous pressure, decrease blood flow in the superior portosystemic collateral circulation, and decrease blood flow in the splanchnic region. Beta-blockade decreases cardiac output reducing hepatic arterial and portal venous perfusion. Activation of unopposed alpha-receptors lead to splanchnic vasoconstriction, thus decreasing portal perfusion.Propranolol is used to treat hypertension and the subsequent decline of renal function in patients with scleroderma renal crisis (SRC). SRC is associated with elevated peripheral renin concentrations. Propranolol blocks beta-receptors located on the surface of the juxtaglomerular cells which decreases the release of renin. In turn, this affects the renin-angiotensin-aldosterone system reducing blood pressure.Numerous mechanisms may contribute to the efficacy of propranolol in preventing migraine

headaches. Beta-blockade can prevent arterial dilation, inhibit renin secretion, and can interfere with catecholamine-induced lipolysis. A decrease in lipolysis decreases arachidonic acid synthesis and, subsequent, prostaglandin production. Inhibition of platelet aggregation is due to this decrease in prostaglandins and blockade of catecholamine-induced platelet adhesion. Other actions include increased oxygen delivery to tissues and prevention of coagulation during epinephrine release.Propranolol has two roles in the treatment of thyrotoxicosis; these actions are determined by the different isomers of propranolol. L-propranolol causes beta-blockade and can ameliorate the symptoms associated with thytotoxicosis such as tremor, palpitations, anxiety, and heat intolerance. D-propranolol blocks the conversion of T4 to T3, but the therapeutic effect of this action is minimal.Propranolol has been used in the management of hereditary or familial essential tremor. Beta-blockade controls the involuntary, rhythmic and oscillatory movements of essential tremor. Tremor amplitude is reduced, but not the frequency of tremor. The mechanism of action is unclear, but the antitremor effect may be mediated by blockade of peripheral beta2 receptor mechanisms.Propranolol can dampen the peripheral physiologic symptoms of anxiety. Beta-blockade can attenuate somatic symptoms of anxiety such as palpitations and tremor, but it is less effective in controlling psychologic components, such as intense fear. These effects are thought to be due to improvement in somatic symptoms secondary to beta-blockade, although the mechanism of action is unclear.

Pharmacokinetics

Propranolol is administered orally or intravenously. Propranolol is highly lipophilic and is widely distributed throughout the body. It readily crosses the blood-brain barrier and the placenta, and is distributed into breast milk. Propranolol is about 90% bound to plasma proteins, the R(+)-enantiomer primarily binds albumin while the S(-)-enantiomer is primarily bound to alpha-1 acid glycoprotein. The volume of distribution is about 4 L/kg. In normal subjects receiving oral doses of racemic propranolol, S(-)-enantiomer concentrations exceeded those of the R(+)-enantiomer by 40% to 90% as a result of stereoselective hepatic metabolism.

Propranolol is extensively metabolized upon first pass through the liver, and the extent of metabolism is dependent on liver blood flow. The drug also binds to and saturates nonspecific hepatic binding sites before the drug reaches the systemic circulation. An equipotent, pharmacologically active metabolite, 4-hydroxypropranolol, is produced with the initiation of oral therapy, but it is eliminated faster than the parent drug. With chronic or IV therapy, this metabolite is produced to a lesser degree. Overall, at least 8 metabolites of propranolol have been identified. Important differences may exist among ethnic groups in the ability to metabolize propranolol, which can affect the overall efficacy of the drug in some instances. Excretion of propranolol occurs renally, primarily

as metabolites, with only 1% to 4% of a dose excreted fecally as unchanged drug. Clearance of the pharmacologically active S(-)-propranolol is lower than R(+)-propranolol after intravenous and oral doses. The elimination half-life of propranolol ranges from 2 to 6 hours, with chronic administration yielding longer half-lives, possibly due to saturation of liver binding sites and/or systemic clearance.

Affected cytochrome P450 enzymes:
Cytochrome P450 enzymes involved in the metabolism of propranolol include 2D6, 1A2, and 2C19. Propranolol is also a substrate for the efflux transporter PGP. The aromatic hydroxylation of propranolol to form the active metabolite, 4-hydroxypropranolol, is mediated by CYP2D6. 4-hydroxypropranolol is a substrate and weak inhibitor of CYP2D6. In healthy subjects, no difference in clearance or half-life of propranolol was observed between extensive and poor CYP2D6 metabolizers. In extensive metabolizers, a significant increase in 4-hydroxypropranolol clearance and a significant decrease in the clearance of naphthyloxyactic acid, an inactive metabolite, was noted.

Route-Specific Pharmacokinetics

- **Oral Route**

After oral administration of immediate-release propranolol, the dose is almost completely absorbed, however, due to high first pass metabolism, bioavailability is only about 25%. Peak concentrations of immediate release tablets and long acting capsules are achieved in 1 to 4 hours and about 6 hours, respectively. Food can increase the bioavailability of the immediate release formulation by approximately 50% but does not affect the time to peak concentration. The effect of food on the bioavailability of the sustained-release formulation has not been investigated.

- **Intravenous Route**

The distribution half-life of intervenously administered propranolol is 5 to 10 minutes. Pharmacodynamic effects are seen immediately and maintained for 2 to 4 hours.

- **Hepatic Impairment**

Propranolol undergoes extensive hepatic metabolism and half-life appears to be prolonged in patients with hepatic impairment. In one study, 7 patients with cirrhosis were compared to 9 healthy subjects, each were given 7 doses of 80 mg propranolol every 8 hours. The half-life of propranolol was prolonged in patients with cirrhosis (11 hours) compared to healthy subjects (4 hours). On average, patients with cirrhosis had 3 times the concentration of unbound propranolol as the healthy subjects. A similar study, conducted with the long acting formulation, yielded a similar result with unbound propranolol concentrations increasing 2.5-fold in cirrhosis patients. After a single IV dose, the half-life of propranolol in cirrhosis patients and healthy subjects was 7.2 hours and 2.9 hours, respectively. Another study examined propranolol pharmacokinetics

after a single 40 mg IV dose was given to 6 healthy subjects and 20 subjects with chronic liver disease, including hepatic cirrhosis. Patients with chronic liver disease had decreased clearance, increased volume of distribution, decreased protein binding, and increased variation in half-life compared to healthy subjects.

- **Renal Impairment**

A reduced propranolol half-life has been reported in patients with renal impairment. This reduction in half-life is seen in conjunction with delayed absorption rate and peak propranolol plasma levels 3 to 4 fold higher than healthy subjects. In a single dose study comparing 5 chronic renal failure patients, 6 dialysis patients, and 5 healthy subjects, peak propranolol concentrations in renal failure patients were 2 to 3 times higher than in dialysis patients or healthy subjects. The renal failure group also displayed reduced plasma propranolol clearance. However, propranolol is not appreciably removed by hemodialysis.

- **Pediatrics**

Children and Adolescents

The extent of propranolol protein binding in children and adolescents (age 6 to 15 years) is similar to that of adults. In a pharmacokinetic study of cyanotic infants and children (n = 5), ages 9 months to 6 years old, mean half-life for propranolol was 4.9 +/- 1 hours (range: 3.9 to 6.4 hours). Mean half-life for the active metabolite 4-hydroxy propranolol was 6.3 +/- 1.1 hours (range: 5.2 to 7.5 hours). Investigators found no correlation between half-life and age.

Infants

Pharmacokinetics of propranolol were evaluated in a multiple dose 12 week study of infants with hemangioma (n = 23; age range: 35 to 150 days). Propranolol was initiated at 1.2 mg/kg/day PO and titrated at weekly intervals to a target dose of 3.4 mg/kg/day PO, divided into twice daily dosing. Plasma propranolol concentrations were dose-proportional in the range studied. At target dose steady state, peak plasma concentrations were observed within 2 hours. Clearance (2.7 L/kg/hour in infants younger than 90 days and 3.3 L/kg/hour in infants older than 90 days) was similar to that in adults when adjusted for body weight. Median elimination half-life was 3.5 hours. The plasma concentration of 4-hydroxy propranolol was approximately 5% of total plasma exposure of propranolol. In pharmacokinetic study of cyanotic children (n = 5) including 1 infant (age: 9 months), mean half-life of propranolol was 4.9 +/- 1 hours (range: 3.9 to 6.4 hours). Mean half-life for the active metabolite 4-hydroxy propranolol was 6.3 +/- 1.1 hours (range: 5.2 to 7.5 hours). Investigators found no correlation between half-life and age.

Neonates

Protein binding of propranolol is approximately 70% in neonates. In a pharmacokinetic

study of 36 neonates (mean gestational age 28 weeks; range: 23 to 42 weeks), patients were treated with high dose [HD] (n = 28; 0.5 mg/kg/dose PO every 6 hours) or low dose [LD] (n = 8; 0.25 mg/kg/dose PO every 6 hours) propranolol. All patients received propranolol administered by mouth or orogastric tube as a syrup shortly after a meal. As observed in children and adults, neonates displayed considerable interpatient variability in plasma propranolol concentrations in patients receiving the same dose; in neonates such variability may be a consequence of hepatic immaturity and a variable first pass effect. Drug plasma concentrations appeared to be directly related to the propranolol dose, suggesting good oral bioavailability. Mean peak plasma concentration (Cmax) was 71.7 +/- 29.8 ng/mL in the HD group and 33.9 +/- 19.1 ng/mL in the LD group; mean AUC was 364.7 +/- 150.2 ng/mL and 161.3 +/- 88.3 ng/mL in the HD and LD groups, respectively. Mean Tmax was 2.6 +/- 0.9 hours in the HD group and 2.3 +/- 0.8 hours in the LD group. Half-life was similar in both groups (HD = 14.9 +/- 4.3 hours; LD = 15.9 +/- 6.1 hours), but significantly prolonged compared to adults, most likely explained by hepatic immaturity in the neonate. Mean plasma clearance was 27.2 +/- 13.9 mL/kg/minute in the HD group and 31.3 mL/kg/minute in the LD group.

- **Geriatric**

Propranolol clearance appears to be reduced and half-life prolonged in the older adult population. A single dose study of 32 patients of varying ages found an inverse correlation between age and clearance of propranolol metabolites (4 hydroxypropranolol and naphthoxylactic acid). A second study comparing 12 older adults (62 to 79 years old) and 12 young (25 to 33 years old) patients reported reduced clearance of the S(-) enantiomer in the older adult group. This study also reported prolonged half-life in the older adult (11 hours) compared to the young group (5 hours).

- **Gender Differences**

Intravenous propranolol was evaluated in 5 women and 6 men. After adjusting for weight, no significant differences were found in half-life, volume of distribution, protein binding or clearance. In women, neither estradiol nor testosterone have demonstrated any change to propranolol plasma binding or clearance. Conflicting evidence exists in regard to the role of testosterone in propranolol metabolism and clearance in men.

- **Ethnic Differences**

African-American patients appear to have increased propranolol clearance and Chinese patients may have increased unbound propranolol concentrations as compared to White patients. In a study of 12 White and 13 African-American men, clearance of both enantiomers was increased in the African-American group. Reported increase in clearance was 76% for the R(+) enantiomer and 53% for the S(-) enantiomer. In another study, unbound plasma propranolol was 18% to 45% higher in Chinese subjects than White subjects.

- **Obesity**

In one study, obese subjects had higher AUC and lower total clearance of IV propranolol than non-obese subjects. No significant difference between the groups was noted for plasma protein binding.

- **Other**

Thyroid Dysfunction
No significant pharmacokinetic changes have been noted between hyperthyroid, hypothyroid and euthyroid subjects.

Administration

For storage information, see the specific product information within the How Supplied section.

Oral Administration

Oral Solid Formulations

Immediate-release tablets: Administer with food.
Sustained-release capsules (e.g., Inderal LA): Administer once daily. Do not crush or chew; swallow whole. If switching from immediate-release tablets to sustained-release capsules, assure desired therapeutic effects are maintained. Substitution should not be simply considered 1:1 as lower plasma concentrations are achieved with the long-acting product. Further titration may be necessary.
Extended-release capsules (e.g., Innopran XL): Administer once daily at bedtime. Do not crush or chew; swallow whole. Take consistently either on an empty stomach or with food.

Oral Liquid Formulations

Generic Oral Solution (4 mg/mL or 8 mg/mL)
Administer with food.

Hemangeol Oral Solution (4.28 mg/mL; for infantile hemangioma)
Record the date on the box when the bottle is first opened.
Do not shake the bottle before use.
Administer during or right after a feeding. Do not administer the dose if the individual is not eating or vomiting.
Avoid fasting; if inevitable, hold medication or support with a product such as Pedialyte or glucose-containing IV fluids.
Using an oral syringe, administer the medicine directly into the child's mouth, against the inside of the cheek. If this is not feasible, the solution may be diluted in a small quantity of milk or fruit juice and given immediately.

Keep the child in an upright position for a few minutes after giving the dose.
Monitor blood pressure and heart rate for 2 hours after the initial dose and after any significant dose increase (e.g., more than 0.5 mg/kg/day).
Storage: Store the bottle in the box at room temperature and discard 2 months after opening.

Injectable Administration

Visually inspect parenteral products for particulate matter and discoloration prior to administration whenever solution and container permit.

Intravenous Administration

IV Bolus Injection
No dilution necessary. If dilution is necessary for accurate dose delivery, the drug may be diluted in 5% Dextrose Injection, 0.9% Sodium Chloride Injection, or Lactated Ringer's Injection.
Monitor ECG and central venous pressure during IV administration.
Adults: Administer at a rate not to exceed 1 mg/minute.
Children: Administer via slow IV push over 10 minutes; do not exceed a rate of 1 mg/minute.

Continuous IV Infusion
Dilute 15 mg in 500 mL 5% Dextrose Injection; may be concentrated to 15 mg propranolol in 250 mL 5% Dextrose Injection for fluid-restricted individuals.
For adults, infuse at a rate of 2 to 3 mg/hour.

Maximum Dosage Limits

- **Adults**
  160 mg/day PO for idiopathic hypertrophic subaortic stenosis (IHSS); 240 mg/day PO for migraine prophylaxis, myocardial infarction prophylaxis, or post-myocardial infarction; 320 mg/day PO for angina, paroxysmal supraventricular tachycardia (PSVT), or tremor; 640 mg/day PO for hypertension. NOTE: Assumes equivalent maximum daily dosage for immediate-release and extended-release products.
- **Geriatric**
  160 mg/day PO for idiopathic hypertrophic subaortic stenosis (IHSS); 240 mg/day PO for migraine prophylaxis, myocardial infarction prophylaxis, or post-myocardial infarction; 320 mg/day PO for angina, paroxysmal supraventricular tachycardia (PSVT), or tremor; 640 mg/day PO for hypertension. NOTE: Assumes equivalent maximum daily dosage for immediate-release and extended-release products.

- **Adolescents**
  Safety and efficacy have not been established; the dose required is dependent on route of administration, indication, and often clinical response. For tachyarrhythmias, doses up to 60 mg/day PO (or 120 mg/day PO in older adolescents) or 0.25 mg/kg/dose IV (Max: 3 mg/dose) have been used. For hypertension, doses up to 8 mg/kg/day PO (Max: 640 mg/day) have been used. For migraine prophylaxis, doses up to 120 mg/day PO have been used. For essential tremor, doses up to 4 mg/kg/day PO have been used.

- **Children**
  Children weighing more than 35 kg: Safety and efficacy have not been established; the dose required is dependent on route of administration, indication, and often clinical response. For tachyarrhythmias, doses up to 60 mg/day PO or 0.25 mg/kg/dose IV (Max: 3 mg/dose) have been used. For hypertension, doses up to 8 mg/kg/day PO (Max: 640 mg/day) have been used. For migraine prophylaxis, doses up to 120 mg/day PO have been used. For essential tremor, doses up to 4 mg/kg/day PO have been used. For tetralogy spells, doses up to 15 mg/kg/day PO have been used (doses more than 5 mg/kg/day PO require close monitoring). Children weighing 35 kg or less: Safety and efficacy have not been established; the dose required is dependent on route of administration, indication, and often clinical response. For tachyarrhythmias, doses up to 60 mg/day PO or 0.25 mg/kg/dose IV (Max: 3 mg/dose) have been used. For hypertension, doses up to 8 mg/kg/day PO (Max: 640 mg/day) have been used. For migraine prophylaxis, doses up to 60 mg/day PO have been used. For essential tremor, doses up to 4 mg/kg/day PO have been used. For tetralogy spells, doses up to 15 mg/kg/day PO have been used (doses more than 5 mg/kg/day PO require close monitoring).

- **Infants**
  3.4 mg/kg/day PO for infantile hemangiomas. Safety and efficacy for other indications have not been established; the dose required is dependent on route of administration, indication, and often clinical response. For tachyarrhythmias, doses up to 16 mg/kg/day PO (Max: 60 mg/day) or 0.15 mg/kg/dose IV (Max: 1 mg/dose) have been used. For hypertension, doses up to 3.5 mg/kg/dose PO have been used. For tetralogy spells, doses up to 15 mg/kg/day PO have been used (doses more than 5 mg/kg/day PO require close monitoring).

- **Neonates**
  Safety and efficacy have not been established; the dose required is dependent on route of administration, indication, and often clinical response. For tachyarrhythmias, doses up to 16 mg/kg/day PO (Max: 60 mg/day) or 0.15 mg/kg/dose IV (Max: 1 mg/dose) have been used. For hypertension, doses up to 3.5 mg/kg/dose PO or 0.15 mg/kg/dose IV have been used.

Dosage Forms

- Hemangeol 4.28mg/mL Solution
- Inderal LA 120mg Extended-Release Capsule
- Inderal LA 160mg Extended-Release Capsule
- Inderal LA 60mg Extended-Release Capsule
- Inderal LA 80mg Extended-Release Capsule
- Inderal XL 120mg Extended-Release Capsule
- Inderal XL 80mg Extended-Release Capsule
- InnoPran XL 120mg Extended-Release Capsule
- InnoPran XL 80mg Extended-Release Capsule
- Propranolol Hydrochloride 10mg Oral tablet
- Propranolol Hydrochloride 120mg Oral capsule, extended release
- Propranolol Hydrochloride 160mg Oral capsule, extended release
- Propranolol Hydrochloride 1mg/1mL Solution for injection
- Propranolol Hydrochloride 20mg Oral tablet
- Propranolol Hydrochloride 20mg/5mL Oral solution [Cardiovascular Disease]
- Propranolol Hydrochloride 40mg Oral tablet
- Propranolol Hydrochloride 40mg/5mL Oral solution [Cardiovascular Disease]
- Propranolol Hydrochloride 60mg Oral capsule, extended release
- Propranolol Hydrochloride 60mg Oral tablet
- Propranolol Hydrochloride 80mg Oral capsule, extended release
- Propranolol Hydrochloride 80mg Oral tablet
- Propranolol Hydrochloride Bulk powder

Dosage Adjustment Guidelines

Hepatic Impairment

Since propranolol is primarily metabolized by the liver, initiate therapy at a reduced dosage for the specified indication; carefully titrate the dosage to attain the desired clinical goals.

Renal Impairment

No dosage adjustment needed.

Intermittent hemodialysis
No dosage adjustments are needed; propranolol is not significantly dialyzable.