Neutropenia, thrombocytopenia, or hemolytic anemia may rarely be encountered.

While the drug was not found to concentrate in the brain at levels higher than in plasma, it is not known if PA crosses the placenta. It has a half-life of approximately five minutes. While PA has been shown to cross the blood-brain barrier in the dog, it is not known if this occurs in humans. Its effects are primarily on the cardiovascular system, with minor effects on the central nervous system. PA is eliminated by active tubular secretion as well as glomerular filtration.

Acetylators" to 24 to 33 percent in “fast-acetylators”. Since NAPA also has significant antiarrhythmic activity and may be significant in the presence of myocardial damage. Therapeutic levels of PA may exert vagolytic slowing action and a weaker vagolytic effect which may speed A-V conduction slightly. Myocardial excitability of His-Purkinje system and ventricles of the heart. It reduces impulse conduction velocity in the atria, His-CLINICAL PHARMACOLOGY

Procainamide hydrochloride, a Group 1A cardiac antiarrhythmic drug, is p-amino-N-[2-(diethylamino) ethyl]propionic acid (NAPA). It is a white crystalline powder with a bitter taste. It is remarkably stable in solution and may be stored in the dark for several months. Procainamide hydrochloride is rapidly converted to NAPA, a metabolite that does not have antiarrhythmic activity.

Procainamide hydrochloride injection, USP Rx only

PHARMACOTHERAPEUTIC AND CLINICAL PHARMACOLOGY

Procainamide hydrochloride injection is indicated for the treatment of documented ventricular arrhythmia, such as sustained ventricular tachycardia, that is, the group of the physiologic, or life-threatening. Because the pharmacologic effect of PA is prominent, it should not be used in the absence of a documented arrhythmia. PA is effective for the treatment of atrial and ventricular tachycardia and may be used prophylactically to prevent SVT. PA is also effective in atrial fibrillation and flutter, and for ventricular fibrillation and tachycardia.

The patient should be encouraged to disclose any past history of drug sensitivity, especially to procaine or benzocaine. Deaths due to angioneurotic edema, urticaria, pruritus, flushing, and maculopapular rash have also occurred.

Drug Interactions

If other antiarrhythmic drugs are being used, additive effects on the heart may occur with PA administration. Pharmacokinetic interaction studies have not been performed. There are no data available on the use of PA and lidocaine, procainamide, quinidine, or disopyramide to treat supraventricular tachycardia.

Non-steroidal anti-inflammatory drugs (NSAIDs) may decrease the antiarrhythmic and antitubular secretion activity of PA. PA has been shown to have lidocaine-like properties in vitro and in vivo. The mechanism of drug interaction is unknown. However, the effect of PA on the tubular secretion of lidocaine-like compounds has been demonstrated in vitro. The interaction of PA with PA does not appear to be a simple competitive inhibition. The effect of PA on the tubular secretion of lidocaine-like compounds is not affected by PA. The effect of PA on the tubular secretion of lidocaine-like compounds is not affected by PA.

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PRECAUTIONS and OVERDOSAGE)

contact Lambda Therapeutics Limited (Toll Free Number: 1-855-642-2594 or by email: safety@lambda-cro.com) or the FDA (Toll Free Number: 1-800-FDA-1088 or by email: safety@lambda-cro.com) for relevant data.

Intravenous Procainamide Hydrochloride Injection should be done cautiously to avoid a possible hypotensive response (see PRECAUTIONS and OVERDOSAGE). Initial antidyssrhythmic therapy, and the patient's general condition, may usually be accomplished safely within a half-hour by either of the two methods.

Concentration of Procainamide Hydrochloride prior to intravenous injection to facilitate control of dosage rate. Doses of 0.5 mg/kg may be administered every 5 minutes at this rate and the antidyssrhythmic effect is evident within 10 minutes or longer in the patient's basic cardiac rhythm appears to be stabilized, oral antiarrhythmic maintenance therapy is possible.

In addition, a loading solution of 30 mg of Procainamide Hydrochloride per kg of body weight in 5% Dextrose Injection, USP, is used in patients with acute coronary insufficiency, congestive heart failure, and acute myocardial infarction. This amount should be divided into fractional doses of one-eighth to one-quarter to be injected intramuscularly every three to six hours until oral therapy is possible. If more than three injections are given, the physician may prefer to exceed 50 mg per minute. It is advisable to dilute either the 100 mg/mL or the 500 mg/mL concentrations under ECG monitoring, may usually be accomplished safely within a half-hour by either of the two methods.

To maintain therapeutic levels, a more dilute intravenous infusion at a concentration of 2 mg/mL is convenient for patients who may have malabsorptive problems. An initial daily dose of 50 mg per kg body weight may be estimated.

Holter monitoring in patients with ventricular arrhythmias, including those following myocardial infarction; it should be carried out in circumstances where close observation and monitoring of the patient are possible, such as in hospital or emergency facilities. Holter monitoring is found to be useful in circumstances where close observation and monitoring of the patient are possible, such as in hospital or emergency facilities.

The maintenance infusion rates are calculated to deliver 2 to 6 mg per minute, depending on body weight and elimination rate, and steady-state plasma level needed to establish control of the arrhythmia. The 4 mg/mL maintenance concentration may be preferred if total infused volume must be limited.

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