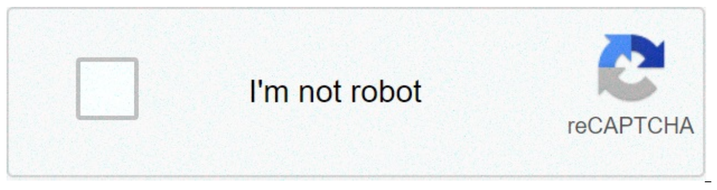

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Lidocaine hydrochloride jelly msds sheet

Allabcdefghiklmnopqrstuvxz Content Last Reviewed On November 18, 2019 lidocaine (lignocaine), prilocaine Pseudoephedrine hydrochloride lignocaine; benzydamine; dichlorobenzyl alcohol citric acid, tartaric acid, sodium bicarbonate methenamine hippurate lidocaine (lignocaine) hydrochloride lidocaine (lignocaine) hydrochloride Content Last Reviewed On June 1, 2018 SummaryLidocaine is a local anesthetic used in a wide variety of superficial and invasive procedures.Brand NamesAgoneaze, Akten, Anestacon, Anodyne Lpt, Astero, Bactine, Band-aid Hurt-free, Cathejell, Curacaine, Depo-medrol With Lidocaine, Dermacinrx Empricaine, Dermacinrx Lido V Pak, Dermacinrx Phn Pak, Dermacinrx Prikaan, Dermacinrx Prizopak, Emla, Fortacin, Glydo, Instillagel, Kenalog, Lido Bdk, Lido-prilo Caine Pack, Lidodan, Lidoderm, Lidopac, Lidopril, Lidopro, Lidothol, Lidotral, Lignospan, Marcaine, Max-freeze, Medi-derm With Lidocaine, Neo-bex, Octocaine, Octocaine With Epinephrine, Oraqix, P-care, P-care X, Pliaglis, Prilolid, Prizotral, Procomycin, Readysharp Anesthetics Plus Ketorolac, Readysharp-A, Readysharp-p40, Readysharp-p80, Relador, Synera, Venipuncture Px1, Viadur, Xylocaine, Xylocaine With Epinephrine, Xylocard, Xylonor, Zingo, ZtlidoGeneric NameLidocaineDrugBank Accession NumberDB00281BackgroundEver since its discovery and availability for sale and use in the late 1940s, lidocaine has become an exceptionally commonly used medication 6. In particular, lidocaine's principal mode of action in acting as a local anesthetic that numbs the sensations of tissues means the agent is indicated for facilitating local anesthesia for a large variety of surgical procedures 10,7,8. It ultimately elicits its numbing activity by blocking sodium channels so that the neurons of local tissues that have the medication applied on are transiently incapable of signaling the brain regarding sensations 10,7,8. In doing so, however, it can block or decrease muscle contractile, resulting in effects like vasodilation, hypotension, and irregular heart rate, among others 10,7,8. As a result, lidocaine is also considered a class Ib anti-arrhythmic agent 7,8,12. Nevertheless, lidocaine's local anesthetic action sees its use in many medical situations or circumstances that may benefit from its action, including the treatment of premature ejaculation 5. Regardless, lidocaine is currently available as a relatively non-expensive generic medication that is written for in millions of prescriptions internationally on a yearly basis. It is even included in the World Health Organization's List of Essential Medicines 9. TypeSmall MoleculeGroupsApproved, Vet approvedStructureWeightAverage: 234.3373 Monoisotopic: 234.173213336 Chemical FormulaC14H22N2OSynonyms2-(Diethylamino)-2',6'-acetoxylidide2-(Diethylamino)-N-(2,6-dimethylphenyl)acetamidealpha-diethylamino-2,6-dimethylacetanilideLidocaínaLidocainaLidocaineLidocainumLignocaineα-diethylamino-2,6-dimethylacetanilideExternal IDs ALGRX 3268 ALGRX-3268 LSM-3165 NSC-40030IndicationLidocaine is an anesthetic of the amide group indicated for production of local or regional anesthesia by infiltration techniques such as percutaneous injection and intravenous regional anesthesia by peripheral nerve block techniques such as brachial plexus and intercostal and by central neural techniques such as lumbar and caudal epidural blocks 10,7. Accelerate your drug discovery research with the industry's only fully connected ADMET dataset, ideal for:Machine LearningData ScienceDrug DiscoveryAccelerate your drug discovery research with our fully connected ADMET datasetAssociated ConditionsAssociated TherapiesContraindications & Blackbox WarningsContraindications & Blackbox WarningsWith our commercial data, access important information on dangerous risks, contraindications, and adverse effects.Our Blackbox Warnings cover Risks, Contraindications, and Adverse EffectsPharmacodynamicsExcessive blood levels of lidocaine can cause changes in cardiac output, total peripheral resistance, and mean arterial pressure 10,7. With central neural blockade these changes may be attributable to the block of autonomic fibers, a direct depressant effect of the local anesthetic agent on various components of the cardiovascular system, and/or the beta-adrenergic receptor stimulating action of epinephrine when present 10,7. The net effect is normally a modest hypotension when the recommended dosages are not exceeded 10,7. In particular, such cardiac effects are likely associated with the principal effect that lidocaine elicits when it binds and blocks sodium channels, inhibiting the ionic fluxes required for the initiation and conduction of electrical action potential impulses necessary to facilitate muscle contraction 10,7,8. Subsequently, in cardiac myocytes, lidocaine can potentially block or otherwise slow the rise of cardiac action potentials and their associated cardiac myocyte contractions, resulting in possible effects like hypotension, bradycardia, myocardial depression, cardiac arrhythmias, and perhaps cardiac arrest or circulatory collapse 10,7,8. Moreover, lidocaine possesses a dissociation constant (pKa) of 7.7 and is considered a weak base 8. As a result, about 25% of lidocaine molecules will be un-ionized and available at the physiological pH of 7.4 to translocate inside nerve cells, which means lidocaine elicits an onset of action more rapidly than other local anesthetics that have higher pKa values 8. This rapid onset of action is demonstrated in about one minute following intravenous injection and fifteen minutes following intramuscular injection 7. The administered lidocaine subsequently spreads rapidly through the surrounding tissues and the anesthetic effect lasts approximately ten to twenty minutes when given intravenously and about sixty to ninety minutes after intramuscular injection 7. Nevertheless, it appears that the efficacy of lidocaine may be minimized in the presence of inflammation 8. This effect could be due to acidosis decreasing the amount of un-ionized lidocaine molecules, a more rapid reduction in lidocaine concentration as a result of increased blood flow, or potentially also because of increased production of inflammatory mediators like peroxynitrite that elicit direct actions on sodium channels 8. Mechanism of actionLidocaine is a local anesthetic of the amide type 10,7,8. It is used to provide local anesthesia by nerve blockade at various sites in the body 10,7,8. It does so by stabilizing the neuronal membrane by inhibiting the ionic fluxes required for the initiation and conduction of impulses, thereby effecting local anesthetic action 10,7,8. In particular, the lidocaine agent acts on sodium ion channels located on the internal surface of nerve cell membranes 10,7,8. At these channels, neutral uncharged lidocaine molecules diffuse through neural sheaths into the axoplasm where they are subsequently ionized by joining with hydrogen ions 10,7,8. The resultant lidocaine cations are then capable of reversibly binding the sodium channels from the inside, keeping them locked in an open state that prevents nerve depolarization 10,7,8. As a result, with sufficient blockage, the membrane of the postsynaptic neuron will ultimately not depolarize and will thus fail to transmit an action potential 10,7,8. This facilitates an anesthetic effect by not merely preventing pain signals from propagating to the brain but by aborting their generation in the first place 10,7,8. In addition to blocking conduction in nerve axons in the peripheral nervous system, lidocaine has important effects on the central nervous system and cardiovascular system 10,7,8. After absorption, lidocaine may cause stimulation of the CNS followed by depression and in the cardiovascular system, it acts primarily on the myocardium where it may produce decreases in electrical excitability, conduction rate, and force of contraction 10,7,8. AbsorptionIn general, lidocaine is readily absorbed across mucous membranes and damaged skin but poorly through intact skin 12. The agent is quickly absorbed from the upper airway, tracheobronchial tree, and alveoli into the bloodstream 12. And although lidocaine is also well absorbed across the gastrointestinal tract the oral bioavailability is only about 35% as a result of a high degree of first-pass metabolism 12. After injection into tissues, lidocaine is also rapidly absorbed and the absorption rate is affected by both vascularity and the presence of tissue and fat capable of binding lidocaine in the particular tissues 12. The concentration of lidocaine in the blood is subsequently affected by a variety of aspects, including its rate of absorption from the site of injection, the rate of tissue distribution, and the rate of metabolism and excretion 10,7,8. Subsequently, the systemic absorption of lidocaine is determined by the site of injection, the dosage given, and its pharmacological profile 10,7,8. The maximum blood concentration occurs following intercostal nerve blockade followed in order of decreasing concentration, the lumbar epidural space, brachial plexus site, and subcutaneous tissue 10,7,8. The total dose injected regardless of the site is the primary determinant of the absorption rate and blood levels achieved 10,7,8. There is a linear relationship between the amount of lidocaine injected and the resultant peak anesthetic blood levels 10,7,8. Nevertheless, it has been observed that lidocaine hydrochloride is completely absorbed following parenteral administration, its rate of absorption depending also on lipid solubility and the presence or absence of a vasoconstrictor agent 10,7,8. Except for intravascular administration, the highest blood levels are obtained following intercostal nerve block and the lowest after subcutaneous administration 10,7,8. Additionally, lidocaine crosses the blood-brain and placental barriers, presumably by passive diffusion 10. Volume of distributionThe volume of distribution determined for lidocaine is 0.7 to 1.5 L/kg 8. In particular, lidocaine is distributed throughout the total body water 7. Its rate of disappearance from the blood can be described by a two or possibly even three-compartment model 7. There is a rapid disappearance (alpha phase) which is believed to be related to uptake by rapidly equilibrating tissues (tissues with high vascular perfusion, for example) 7. The slower phase is related to distribution to slowly equilibrating tissues (beta phase) and to its metabolism and excretion (gamma phase) 7. Lidocaine's distribution is ultimately throughout all body tissues 7. In general, the more highly perfused organs will show higher concentrations of the agent 7. The highest percentage of this drug will be found in skeletal muscle, mainly due to the mass of muscle rather than an affinity 7. Protein bindingThe protein binding recorded for lidocaine is about 60 to 80% and is dependent upon the plasma concentration of alpha-1-acid glycoprotein 10,8. Such percentage protein binding bestows lidocaine with a medium duration of action when placed in comparison to other local anesthetic agents 8. Lidocaine is metabolized predominantly and rapidly by the liver, and metabolites and unchanged drug are excreted by the kidneys 10,7. Biotransformation includes oxidative N-dealkylation, ring hydroxylation, cleavage of the amide linkage, and conjugation 10,7. N-dealkylation, a major pathway of biotransformation, yields the metabolites monoethylglycinexylidide and glycinexylidide 10,7. The pharmacological/toxicological actions of these metabolites are similar to, but less potent than, those of lidocaine HCl 10,7. Approximately 90% of lidocaine HCl administered is excreted in the form of various metabolites, and less than 10% is excreted unchanged 10,7. The primary metabolite in urine is a conjugate of 4-hydroxy-2,6-dimethylaniline 10,7. Hover over products below to view reaction partnersRoute of eliminationThe excretion of unchanged lidocaine and its metabolites occurs predominantly via the kidney with less than 5% in the unchanged form appearing in the urine 10,7. The renal clearance is inversely related to its protein binding affinity and the pH of the urine 7. This suggests by the latter that excretion of lidocaine occurs by non-ionic diffusion 7. Half-lifeThe elimination half-life of lidocaine hydrochloride following an intravenous bolus injection is typically 1.5 to 2.0 hours 10. Because of the rapid rate at which lidocaine hydrochloride is metabolized, any condition that affects liver function may alter lidocaine HCl kinetics 10. The half-life may be prolonged two-fold or more in patients with liver dysfunction 10. ClearanceThe mean systemic clearance observed for intravenously administered lidocaine in a study of 15 adults was approximately 0.64 +/- 0.18 L/min 11. Adverse EffectsReduce medical errorsand improve treatment outcomes with our comprehensive & structured data on drug adverse effects.Reduce medical errors & improve treatment outcomes with our adverse effects dataToxicitySymptoms of overdose and/or acute systemic toxicity involves central nervous system toxicity that presents with symptoms of increasing severity 7. Patients may present initially with circumoral paraesthesia, numbness of the tongue, light-headedness, hyperacusis, and tinnitus 7. Visual disturbance and muscular tremors or muscle twitching are more serious and precede the onset of generalized convulsions 7. These signs must not be mistaken for neurotic behavior 7. Unconsciousness and grand mal convulsions may follow, which may last from a few seconds to several minutes 7. Hypoxia and hypercapnia occur rapidly following convulsions due to increased muscular activity, together with the interference with normal respiration and loss of the airway 7. In severe cases, apnoea may occur. Acidosis increases the toxic effects of local anesthetics 7. Effects on the cardiovascular system may be seen in severe cases 7. Hypotension, bradycardia, arrhythmia and cardiac arrest may occur as a result of high systemic concentrations, with potentially fatal outcome 7. Pregnancy Category B has been established for the use of lidocaine in pregnancy, although there are no formal, adequate, and well-controlled studies in pregnant women 10. General consideration should be given to this fact before administering lidocaine to women of childbearing potential, especially during early pregnancy when maximum organogenesis takes place 10. Ultimately, although animal studies have revealed no evidence of harm to the fetus, lidocaine should not be administered during early pregnancy unless the benefits are considered to outweigh the risks 7. Lidocaine readily crosses the placental barrier after epidural or intravenous administration to the mother 7. The ratio of umbilical to maternal venous concentration is 0.5 to 0.6 7. The fetus appears to be capable of metabolizing lidocaine at term 7. The elimination half-life in the newborn of the drug received in utero is about three hours, compared with 100 minutes in the adult 7. Elevated lidocaine levels may persist in the newborn for at least 48 hours after delivery 7. Fetal bradycardia or tachycardia, neonatal bradycardia, hypotonia or respiratory depression may occur 7. Local anesthetics rapidly cross the placenta and when used for epidural, paracervical, pudendal or caudal block anesthesia, can cause varying degrees of maternal, fetal and neonatal toxicity 10. The potential for toxicity depends upon the procedure performed, the type and amount of drug used, and the technique of drug administration 10. Adverse reactions in the parturient, fetus and neonate involve alterations of the central nervous system, peripheral vascular tone, and cardiac function 10. Maternal hypotension has resulted from regional anesthesia 10. Local anesthetics produce vasodilation by blocking sympathetic nerves 10. Elevating the patient's legs and positioning her on her left side will help prevent decreases in blood pressure 10. The fetal heart rate also should be monitored continuously, and electronic fetal monitoring is highly advisable 10. Epidural, spinal, paracervical, or pudendal anesthesia may alter the forces of parturition through changes in uterine contractility or maternal expulsive efforts 10. In one study, paracervical block anesthesia was associated with a decrease in the mean duration of first stage labor and facilitation of cervical dilation 10. However, spinal and epidural anesthesia have also been reported to prolong the second stage of labor by removing the parturient's reflex urge to bear down or by interfering with motor function 10. The use of obstetrical anesthesia may increase the need for forceps assistance 10. The use of some local anesthetic drug products during labor and delivery may be followed by diminished muscle strength and tone for the first day or two of life 10. The long-term significance of these observations is unknown 10. Fetal bradycardia may occur in 20 to 30 percent of patients receiving paracervical nerve block anesthesia with the amide-type local anesthetics and may be associated with fetal acidosis 10. Fetal heart rate should always be monitored during paracervical anesthesia 10. The physician should weigh the possible advantages against risks when considering a paracervical block in prematurity, toxemia of pregnancy, and fetal distress 10. Careful adherence to the recommended dosage is of the utmost importance in obstetrical paracervical block 10. Failure to achieve adequate analgesia with recommended doses should arouse suspicion of intravascular or fetal intracranial injection 10. Cases compatible with unintended fetal intracranial injection of local anesthetic solution have been reported following intended paracervical or pudendal block or both. Babies so affected present with unexplained neonatal depression at birth, which correlates with high local anesthetic serum levels, and often manifest seizures within six hours 10. Prompt use of supportive measures combined with forced urinary excretion of the local anesthetic has been used successfully to manage this complication 10. It is not known whether this drug is excreted in human milk 10. Because many drugs are excreted in human milk, caution should be exercised when lidocaine is administered to a nursing woman 10. Dosages in children should be reduced, commensurate with age, body weight and physical condition 10. The oral LD 50 of lidocaine HCl in non-fasted female rats is 459 (346-773) mg/kg (as the salt) and 214 (159-324) mg/kg (as the salt) in fasted female rats 10. PathwaysPharmacogenomic Effects/ADRs Not AvailableUNII98PI200987CAS number137-58-6InChI KeyNNJVILVZKWQKPM-UHFFFAOYSA-NInChIInChI=1S/C14H22N2O/c1-5-16(6-2)10-13(17)15-14-11(3)8-7-9-12(14)4/h7-9H,5-6,10H2,1-4H3,(H,15,17)IUPAC Name2-(diethylamino)-N-(2,6-dimethylphenyl)acetamideSMILESCCN(CC)CC(=O)NC1=C(C)C=CC=C1CManufacturersAstrazeneca lp Noven pharmaceuticals inc Carlisle laboratories inc E fougera div altana inc Graham chemical co Taro pharmaceuticals usa inc Teikoku pharma usa inc Abbott laboratories pharmaceutical products div Abbott laboratories hosp products div Abraxis pharmaceutical products Akorn inc Baxter healthcare corp anesthesia and critical care Bel mar laboratories inc Dell laboratories inc Elkins sinn div ah robins co inc Gd searle llc Hospira inc International medication systems ltd International medication system Luitpold pharmaceuticals inc Miles laboratories inc Watson laboratories inc Wyeth ayerst laboratories Baxter healthcare corp B braun medical inc App pharmaceuticals llc Meridian medical technologies inc Dentsply pharmaceutical Polymedica industries inc Teva pharmaceuticals usa Hi tech pharmacal co inc Wockhardt eu operations (swiss) ag Actavis mid atlantic llc Vintage pharmaceuticals llc Roxane laboratories inc Kendall co Paco research corp Anesiva incPackagers4uOrtho LLC A. Aarons Inc. Actavis Group Aerospace Accessory Service Inc. Akorn Inc. Amend American Dental Cooperative Inc. American Regent Amphastar Pharmaceuticals APP Pharmaceuticals Aristos Pharmaceuticals A-S Medication Solutions LLC AstraZeneca Inc. Ato Zizine Sarl Auriga Pharmaceuticals LLC Avent Inc. B. Braun Melsungen AG Baxter International Inc. Benco Dental Co. Blairex Labs Bradley Pharmaceuticals Inc. Breckenridge Pharmaceuticals Brookstone Pharmaceuticals C.O. Truxton Inc. Cardent International Inc. Cardinal Health Carestream Health Inc. Carlisle Laboratories Inc. Catalent Pharma Solutions Codman and Shurtleff Inc. Covidien LP Cypress Pharmaceutical Inc. Darby Dental Supply Co. Inc. Deltex Pharmaceuticals Inc. DENTSPLY International Dispensing Solutions Diversified Healthcare Services Inc. Doak Dermatologics DSC Laboratories E. Fougera and Co. Eastman Kodak Co. Dental Products Endo Pharmaceuticals Inc. Enterprises Importfab Inc. F Hoffmann-La Roche Ltd. Fresca Gourmet Inc. 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Rising Pharmaceuticals River's Edge Pharmaceuticals Roxane Labs S&P Healthcare Safco Dental Supply Co. Sandoz Septodont Inc. Sheffield Laboratories Div Faria Limited LLC Smiths Medical ASD Inc. Sonar Products Inc. Southwood Pharmaceuticals Stat Rx Usa Taro Pharmaceuticals USA Tech Group Tempe Teikoku Seiyaku Co. Ltd. Teva Pharmaceutical Industries Ltd. Tri State Hospital Supply Corp. Veratex Corp. Vintage Pharmaceuticals Inc. Vyteris Inc. Wallach Surgical Devices Inc. Welch Allyn Inc. 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