DOWNLOAD PDF CHOLECYSTOKININ AND ITS ANTAGONISTS IN PAIN MANAGEMENT

Chapter 1 : Cholecystokinin and Pain

Cholecystokinin and Its Antagonists in Pain Management. McCleane G. Binghamton, NY: The Haworth Press, ISBN pages, $/$ This is a page text devoted to a pain medicine topic of contemporary import.

Other commonly used names are: For a listing of dosage forms and brand names by country availability, see Dosage Forms section s. It is used to stimulate gallbladder contraction and emptying prior to, or during, cholecystography with contrast media to aid in visualization of the cystic duct and gallbladder. As part of oral cholecystography or preoperative cholangiography, cholecystokinin facilitates the evaluation of the contractile patterns of the gallbladder, filling of the bile ducts, flow of contrast medium into the duodenum, and localization of gallstones in the lower common bile duct. Cholescintigraphy is the preferred method rather than cholangiography, especially in patients in whom acute cholecystitis is suspected. Cholecystokinetic and Diagnostic aid gallbladder disorders â€"Cholecystokinin, a natural polypeptide formed in the amine precursor uptake and decarboxylation APUD cells of the proximal mucosa of the small intestine, induces contraction of the gallbladder muscle, resulting in reduction of gallbladder size and evacuation of bile. Cholecystokinin inhibits contraction of the lower esophageal sphincter and the sphincter of Oddi. Contraction of the gallbladderâ€"Within 1 to 3 minutes. Contraction of gallbladderâ€"Approximately 2 hours or more. However, problems in humans have not been documented. Pediatrics Appropriate studies on the relationship of age to the effects of cholecystokinin have not been performed in the pediatric population. Geriatrics Appropriate studies performed to date have not demonstrated geriatrics-specific problems that would limit the usefulness of cholecystokinin in the elderly. Except under special circumstances, this medication should not be used when the following medical problem exists: Description of use Procedure for cholecystokinin test: For oral cholecystography, the patient should be given the contrast medium on the evening before the examination. Fluoroscopy is recommended before the x-ray examination. If the gallbladder is visible, cholecystokinin may be injected. Preoperativeâ€"Intravenous, 40 Ivy dog units IDU 4 mL administered approximately one minute prior to administration of contrast medium. Concurrent with the last injection, cholangiography may be performed. After ingestion of to mL of barium mixture, patients should lie on their right side for 10 to 15 minutes; if fluoroscopy shows that most of the contrast medium has passed into the first part of the jejunum, cholecystokinin is injected. Usual geriatric dose Strength s usually available U. However, sincalide CCK-8 , a synthetically prepared C-terminal octapeptide of cholecystokinin, is commercially available in the U.

Chapter 2 : Cholecystokinin and pain: a review.

Haworth series in clinical pain and symptom palliation QP Cholecystokinin (CCK) is the regulatory peptide hormone found primarily in the gastro-intestinal tract that works as a neurotransmitter throughout the nervous system.

Trout Originally Published at Opioids. This is directly responsible for a number of the problems associated with narcotic use and abuse since increasing tolerance requires that steadily larger doses be used to achieve the same effects or degree of pain relief. This also underlies much of the crime associated with street addiction as the cost of maintaining a habit also escalates along with the dosage, often leading addicts to turn to drug dealing, prostitution or criminal activities to enable them to afford their daily dose. Many experienced junkies, especially if heroin users, address this problem by taking regular breaks from their drug of choice, allowing their tolerance to diminish and their effective dosage to also be decreased. Due to the unpredictable quality of unregulated black-market street drugs this can actually be potentially dangerous if they then acquire material of greater potency than they were expecting. Junkies who relapse after recovery face a similar risk when they return to use. Some users employ materials like cimetidine Tagamet [R. While this has been reported by many users to be effective at maximizing per dose results this does not affect the development of tolerance. Presently many questions remain, as there is also been some conjecture made that administration of grapefruit juice might interfere with the conversion of codeine to morphine due to its lesser inhibition of some CYP subfamilies. This does not seem to be the case; Caraco et al. Although, it is certainly reasonable to assume that CYP3A is responsible for its metabolism since it is proven as such for other opioids such as codeine Caraco et al. It is important to keep in mind that grapefruit juice can also prove problematic due to the elevated levels of bioavailable drug, requiring a reduction of the dosage. Sometimes it can even be dangerous if certain other drugs are being used. The combination of grapefruit juice with some specific pharmaceuticals has produced many serious problems and even some deaths. This is said to produce a rough doubling of intensity with the addition of unwanted side effects like a dry mouth. An interesting approach is the combination of opiates with the opiate antagonists naloxone or naltrexone in miniscule amounts. The combination of less than 0. It is also said to prevent respiratory depression, tolerance and addiction. However, many people are unaware that both enhanced effectiveness of narcotic analgesics AND prevention or reversal of tolerance is readily achievable through the oral use of up to mg of Proglumide [DL Benzamido-N,N-dipropylglutaramic acid]. Rather than simply augment the action of the opiates, proglumide actually interferes with the anti-opioid activity of the neuropeptide CCK. The chronic administration of opiates, or spinal cord and other CNS injuries, elevates the level of Cholecystokinin CCK that is present. Such elevated levels exert an antagonistic effect on opioid activity resulting in significantly diminished analgesic effects. This anti-opiate effect can be prevented or even reversed through the administration of CCK inhibitors such as proglumide. Often this can provide a higher quality of analgesia for those patients who suffer from an incomplete response to pain medications. They suggested that not simply did this indicate that effective narcotic doses could be decreased but it also indicated that proglumide might be able to enhance the effects of other procedures, such as acupuncture, which involve endogenous opiates. It shows NO analgesic effects of its own. Although proglumide is now considered to be an obsolete pharmaceutical due to changes in our understandings of ulcer etiology, it has already seen extensive pharmacological and toxicological testing proving its safety and has been approved for use in humans. It has largely fallen into disuse but is still available in bulk via chemical houses or as a pharmaceutical in Europe and Africa sold under the trade name Milid and Milide. However, beyond simply having seen previous use in humans, proglumide is both inexpensive and nontoxic. Ott Proglumide is not some sort of magic bullet for completely eliminating the risk of tolerance development and addiction as its effects are only effective for a limited duration before tolerance to IT begins to develop. More work is needed to better define the precise parameters of its effective use for this purpose. Despite this, proglumide has already demonstrated itself to be of value both in pain management and as an adjunct to maintaining a narcotic

addiction within a larger program of harm reduction Anonymous ; Ott What is fascinating is how few drug educators, drug treatment facilities or even drug users are aware of this despite it being readily available information for nearly 20 years. If development of tolerance and the high price of a sustained addiction are truly as serious of a problem as we all agree that they are, one can only wonder how it is that, despite the tools existing to remove or at least reduce this problem, there seems to be no interest or research except on a limited scale related to specific small areas of chronic pain management and understanding. The current misguided approach of substituting methadone is commonly reported to actually cause MORE perceptual and thinking problems than the opiates it replaces PLUS methadone is known to cause physical damage to internal organs that are not encountered with opiate use itself. Harm reduction approaches would benefit greatly by using proglumide as a cornerstone and making it readily available to both narcotic users and abusers. Those who will most certainly object include organized crime and drug dealers who enjoy the obscene profits reaped from escalating drug tolerances, and possibly also the so-called "drug educators" that sadly often seem to be the ones most in need of some factual education. There are many problems associated with opiate use and abuse. Increased analgesic effectiveness and prevention of tolerance are two obvious areas where harm reduction is readily possible TODAY. Both sufferers of chronic pain and narcotic addicts stand to benefit from having their needs met and their health risks simultaneously decreased. As this is first and foremost a health problem, the current approach of harm maximization is both counterproductive and unacceptable. To a rationale or caring mind it might even be perceived of as unethical and amoral. Not only do sufferers of chronic pain and narcotic addicts stand to benefit from such harm reduction approaches but, by decreasing drug-associated crimes, a significant area of the true "drug problem" can be directly addressed, thereby benefiting society as a whole. Weintraub Clinical Pharmacokinetics 33 2: See patent references farther below. Lasker Drug Metab. Members of a different class of cholecystokinin receptor antagonists. Mayer Brain Research Pasanen] [Abstract from PubMed] R. Xu Regulatory Peptides David Spence] Edwards, D. Extent, probable mechanism and clinical relevance. Shen US Patent No. Shen b Trends Pharmacol. Crain Brain Research Bunney Science K Trout trout yage.

Chapter 3 : CCK Drug Information, Professional

Cholecystokinin and Its Antagonists in Pain Management extensively describes opioids in pain management, CCK and the body's uses for the chemical, CCK receptor antagonists, and the evidence that points to its being a possible breakthrough application in pain management.

Selected References These references are in PubMed. This may not be the complete list of references from this article. Immunohistochemical demonstration of a CCK-like peptide in the nervous system of a marine annelid worm, Nereis diversicolor O. The ascent of cholecystokinin CCK - from gut to brain. Three components of gastrin in human serum. Gel filtration studies on the molecular size of immunoreactive serum gastrin. Immunological and biological studies on cholecystokinin in rat brain. Excitation of CA1 pyramidal neurones of the hippocampus by the tetra- and octapeptide C-terminal fragments of cholecystokinin [proceedings]. The actions of cholecystokinin and related peptides on pyramidal neurones of the mammalian hippocampus. Immunochemical evidence of cholecystokinin tetrapeptides in hog brain. New peptide in the vertebrate CNS reacting with antigastrin antibodies. Immunochemical evidence of cholecystokinin-like peptides in brain. Immunoreactive component resembling cholecystokinin octapeptide in intestine. Isolation, structure and biological activity of two cholecystokinin octapeptides from sheep brain. Cholecystokinin and its COOH-terminal octapeptide in the pig brain. Immunochemical studies on cholecystokinin. Development of sequence-specific radioimmunoassays for porcine triacontatriapeptide cholecystokinin. Demonstration of biological activity of brain gastrin-like peptidic material in the human: Localization and molecular heterogeneity of cholecystokinin in the central and peripheral nervous system. Neural crest origin of the endocrine polypeptide APUD cells of the gastrointestinal tract and pancreas. Distribution of gastrin and CCK-like peptides in rat brain. Characterization of a nontrypsin cholecystokinin converting enzyme in mammalian brain. Cholecystokinin receptors in the brain: Antagonism of stimulation-produced analgesia by naloxone, a narcotic antagonist. Brainstem control of spinal pain-transmission neurons. Central nervous system mechanisms of analgesia. Evidence for opioid and non-opioid forms of stimulation-produced analgesia in the rat. On the central sites for the antinociceptive action of morphine and fentanyl. Regional distribution of opiate receptor binding in monkey and human brain. Pain reduction by focal electrical stimulation of the brain: Behavioral and electrophysiological evidence of pain inhibition from midbrain stimulation in the cat. Surgery in the rat during electrical analgesia induced by focal brain stimulation. Opioid and non-opioid mechanisms of footshock-induced analgesia: Involvement of spinal opioid systems in footshock-induced analgesia: Reversal of morphine and stimulus-produced analgesia by subtotal spinal cord lesions. Organization of endogenous opiate and nonopiate pain control systems. Opiate vs non-opiate footshock induced analgesia FSIA: Evidence for the neuropeptide cholecystokinin as an antagonist of opiate analgesia. Caerulein and cholecystokinin suppress beta-endorphin-induced analgesia in the rat. Potentiation of opiate analgesia and apparent reversal of morphine tolerance by proglumide. Antagonism of cholecystokinin-like peptides by opioid peptides, morphine or tetrodotoxin. Cholecystokinin octapeptide releases growth hormone from the pituitary in vitro.

Chapter 4 : Erowid Opiates Vaults : Tolerance, Addiction, and Effective Pain Management, by K. Trout

Cholecystokinin and Its Antagonists in Pain Management by Gary McCleane Examine the strong evidence of this unrealized pain management tool Cholecystokinin (CCK), the regulatory peptide hormone found primarily in the gastrointestinal tract that works as a neurotransmitter throughout the nervous system, has been researched for years.

Chapter 5 : Proglumide - Wikipedia

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