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شبكة المعلومـــات الجامعية

التوثيق الالكتروني والميكروفيلم

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جامعة عين شمس

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نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
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15 – 25c and relative humidity 20-40 %

بعض الوثائق الأصلية تالفة

بالرسالة صفحات

لم ترد بالأصـــل

INTRAVENOUS REGIONAL ANAESTHESIA (IVRA) WITH LIDOCAINE, FENTANYL AND PANCURONIUM MIXTURE VERSUS LIDOCAINE , TENOXICAM AND PANCURONIUM

Thesis

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Introduction

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PERIPHERAL MECHANISMS OF SOMATIC PAIN

Pain is the perceptual counterpart of the body's response to stimuli that threaten the integrity of its tissues. The aversive nature of pain strongly motivates the organism to avoid these noxious stimuli. Additionally, pain may help promote healing by motivating the organism to avoid contact or motion of an injured area. However, pain may persist byond its useful purpose and result in profound behavioural disturbances and suffering. Recent research has improved our understanding of neural substrates of pain sensation. Mammals have been shown to have specialized peripheral sense organs for detecting noxious stimuli The information signalled by these receptors is transmitted rostrally by neurons forming a distinct subset of afferent systems projecting to the somatosensory cortex. In addition, the central processing of afferent information in the dorsal horn is modulated by activity descending from rostral areas of the central nervous system. Also, several candidate neurotransmitters have been identified that serve as

synaptic links in the dorsal horn of afferent fibers. Some of these neurotransmitters may be specific for signalling pain.[1]

How the nervous system differentiates between the various forms of sensory experiences has been controversial issue since the beginning of sensory physiology. The two principle theories which have been proposed are the pattern theory and the specificity theory. According to the pattern theory, the perceived sensation is determined by the pattern of inlet from the skin. Thus, a particular sensation is not associated with a specific receptor, but, rather, with the spatial and temporal pattern of nerve impulses from multiple receptors.[2] According to the specificity theory, specialized cutaneous receptors and neural pathways exist for each modality of sensation arising from cutaneous stimulation.[3]

A class of cutaneous afferent fibers with receptors, termed nociceptors, was identified. These nociceptors encode the occurance, intensity, duration and location of noxious stimuli and signal pain sensation. Nociceptors can be classified according to: their reponse to different modalities of intense stimulation, the conduction velocity of their peripheral axons,

and differences in the charactersitics of their response to stimuli.[4]

CLASSIFICATION OF NOCICEPTORS

Cutaneous nociceptors: Cutaneous nociceptive afferents may be myelinated (A-fibers) or unmyelinated (C-fibers). C-fibers with receptors that respond to noxious mechanical and heat stimuli, often referred to as C-mechano-heat (CMHs) receptors, are found in the skin of most species. The corresponding A-fiber nociceptive afferents are termed AMHs.[5] Some myelinated and unmyelinated afferents have receptors that respond only to intense noxious mechanical stimuli, and have, therefore, been called high threshold mechanoceptors (HTMs). A small percentage of myelinated and unmyelinated afferents respond to noxious mechnaical and cold stimuli, but not to heat.[6] Other nociceptors that respond only to intense cold or intense heat have also been reported.[7] The presence in the human skin of specific chemosenstive nociceptors has also been suggested.[8]

Muscle nociceptors: Candidate nociceptive afferents in muscle include small myelinated (group III) and unmyelinated (group IV) afferent fibers.[9] Presumably, the receptors for the muscle

nociceptive afferents are the many free nerve endings found in the connective tissue of the muscle, between muscle fibers, in blood vessel walls, or in tendons. These form as much as 75% of the sensory innervation of skletal muscle.[10]

Joint nociceptors: Nerve fiber counts in the articular nerves of cats reveal that the predominant population of joint afferents is formed, not by large afferents, but rather by small myelinated A-delta (group III, conduction velocity 2.5-20 m/s) and unmyelinated C-fiber afferents (group IV, conduction velocity < 2.5 m/s).[11]

ROLE OF CHEMICAL MEDIATORS IN PAIN PRODUCTION

The pain producing properties of several endogenous vasoactive substances were studied extensively by keele and Armstrong.[12] By applying substances to blister bases in volunteers, they showed that pain was produced by peptides such as angiotensin, bradykinin and substance P, and amines such as 5-hydroxytryptamine, serum, and various inflammatory exudates. Considerable evidence has since been presented that some of these substances, espicially bradykinin and eicosanoids promote the pain and hyperalgesia associated with inflammation.[13] Prostaglandins (PGs) have also been observed

to produce prolonged hyperalgesia when injected in the rat paw.[14] It has been suggested that PG-mediaed hyperalgesia is a C-AMP/Ca^{++} mediated process.[15] When given intradermally or intramuscullarly in concentrations higher than those that occur in inflammation, PGE, causes long-lasting pain.[16]

The PGs must be considered to be different from other inflamatory mediators, because their principal role appears to be to enhance the inflammatory effects of other mediators, such as vaso-active amines and kinins.[17] Electrophysiological studies of cutaneous nociceptive afferents have provided direct evidence for the sensitizing effects of prostaglandins on unmyelinated nociceptors, as well as, A-delta mechanoceptors.[18] Continous intraarterial infusions of PGE_1 and PGE_2 sensitize C-nociceptors and intensify brodykinin-induced discharges of these efferents.[19]

HISTORY OF LOCAL ANALGESIA[20]

The origin of modern local Anlagesia is credited to **Carl Koller**, an ophthalmologist, who demonstrated the use of cocaine for surgical anesthesia of the eye in 1884. Cocaine had been isolated from the coca plant in 1855 by Gaedicke and later purified by Albert Neimann in 1860. The surgeon **William Halsted** demonstrated in 1884 the use of cocaine for