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FOCUS ON: CONSIDERATIONS DURING THE POSTOPERATIVE PERIOD

Pruritus and anaesthesia

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Summary Pruritus is a common complication of anaesthesia. In this article, current knowledge regarding the pathophysiology and treatment of the condition are presented. A suggested approach for treatment is given.

Itching is common in the postoperative period. Often it is no more than a minor nuisance, or a transient phase but it may be a severely troublesome problem for some patients. Despite this, even the largest of anaesthesia textbooks often devote only a paragraph or two to the topic. The aim of this article is to summarize current knowledge regarding the condition, and to suggest a practical approach to treating it.

Definition

Itch is defined simply as a sensation, arising from the skin or a mucous membrane, which produces the desire to scratch. Itch is benign, occurring spontaneously, or secondary to irritation from an external stimulus, such as a woolly jumper. If itch is pathological, it is termed pruritus. For that reason, the term pruritus is used in this article. Measurement scores using visual analogues scales and frequency of itching are commonly described. These are useful in scientific studies of the phenomenon, but are of less use in the day-to-day treatment of sufferers.

Transmission pathways of itch

The precise mechanism of itch transmission is incompletely understood. However, current knowledge has been comprehensively summarized.[1] Itch was previously taken to be simply a sub-modality of pain. This is now known not to be the case. Its transmission involves a sub-population of the nocioceptive C-fibres, which unlike their pain analogues, are insensitive to mechanical stimulation, but sensitive to histamine. These primary neurones travel to the ipsilateral dorsal horn of the spinal column and synapse there with a specific secondary itch neurone. These cross immediately to the contralateral anterolateral spinothalamic tract, and radiate to the thalamus. There, they synapse with a tertiary neurone, which projects to the somatosensory cortex, specifically the postcentral gyrus (Fig. 1). There is evidence of activation of the supplementary motor area, perhaps in preparation for the action of scratching. Although distinct from pain transmission, there is

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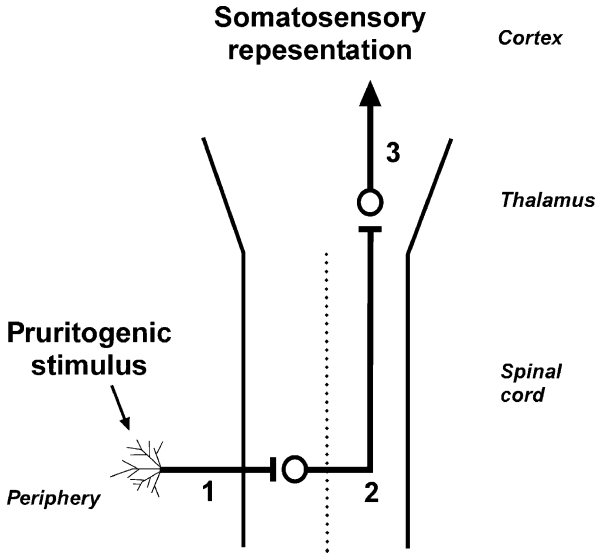

Figure 1 Diagrammatic representation of transmission pathway of itch. Primary, secondary and tertiary afferent neurones marked 1, 2 and 3, respectively.

some evidence of interaction between the two modalities, with each able to inhibit transmission of the other.

Substances mediating pruritus

A number of substances act or are postulated to act as mediators in the transmission or potentiation of pruritus. Knowledge of these aids understanding and planning of anti-pruritus treatments.

Histamine

This is the most commonly known potentiator of itch, and the one which is most commonly targeted in simple anti-pruritus treatment. Histamine is released in the epidermis from mast cells in response to a number of stimuli, and stimulates H_1 receptors in the epidermal C-itch fibres. Eighty-five percent of epidermal receptors are H_1 receptors; the rest are H_2.[2] Histamine is only implicated in itch due to mast cell degranulation, such as allergic, insect bite or following drug-related histamine release.

Arachidonic acid derivatives

Prostaglandins may fulfil the same role in potentiating and sensitizing itch receptors, as they do with pain receptors. Leukotrienes also appear to have a potentiating effect. Non-steroidal anti-inflammatory drugs (NSAIDs) have been shown to reduce pruritus in allergic conjunctivitis. Ninety percent of those given ketorolac reported improvement in their symptoms.[3] Following anaesthesia for abdominal surgery including epidural fentanyl, the effect of tenoxicam was studied.[4] The tenoxicam group had less pruritus, but also had lower pain scores and used less pethidine. This may explain the lower incidence of pruritus. However, when the cyclo-oxygenase 2 inhibitor celecoxib was given to women receiving spinal anaesthesia using bupivacaine and morphine, no effect on incidence of pruritus was shown.[5] These findings suggest that there may be a role for NSAIDs in the prevention and/or treatment of itch, but clearly there is much more to be researched yet.

Serotonin

The observation in some studies that 5-HT_3 antagonists can successfully treat resistant itch in a small group of patients has led to the postulation that 5-HT_3 receptor is involved in itch. Ondansetron is the most studied drug (see below).

Others

Intradermally, acetyl choline does not elicit itch in normal individuals (rather pain), but in atopic individuals, itch is elicited. Peptides can mediate itch, either via or without histamine release (such as bradykinin and Substance P, respectively). Protease enzymes liberated during tissue damage have been implicated. Cytokines are involved in some forms of itch.

Anaesthesia-related causes of itch

Opioids

These probably represent the most common anaesthetic-related cause of pruritus. Pruritus can occur as a result of systemic or neuraxial administration. It appears that systemic administration causes pruritus via peripheral and central mechanisms, whereas neuraxial route acts solely via central mechanisms.

Systemic mechanisms

Opioids cause histamine release via non-immune-mediated degranulation of mast cells, which in turn causes itch. Administration of simple anti-histamine drugs can reverse this type of pruritus, but not always. This implies that some of the pruritus is