**Balanced and Multimodal General Anesthesia: A Modern Approach for an Old Art**

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*Then the Lord God caused the man to fall into a deep sleep and, while he slept, he pulled out a rib and closed Me wound* (Genesis, 2: 21).

The three pillars on which the current anaesthetic management of patients is based are, from a clinical point of view, hypnosis/amnesia, immobility (neuromuscular blockade, NM-13) and the control of the response of the autonomic nervous system (ANS; sympathetic - SNSy-parasympathetic - SNPa) to intraoperative nociception (antinociception).

![Antinociception: ANS control Hypnosis/amnesia: Unconsciousness](image)

Physiologically we can categorize these components according to their "location": components of mainly cortical (hypnosis/amnesia) and subcortical (immobility, ANS control) origin.

The aggression that surgery implies to the organism will be translated into a response mediated by the ANS in its two components (SNSi - SNPa).

The more or less current concept of “antinociception” would replace the classic “intraoperative analgesia”, if we stick to the more or less universal definition of “pain” as “unpleasant emotional sensation and experience associated with actual or potential tissue damage”, the patient’s analgesia only becomes of interest when he is conscious, not under the state of diminished consciousness secondary to general anaesthesia. Thus, we must be concerned that the patient is optimally analgesic “when awake”.

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The elements that make up the nerve pathway that mediates nociception are generically called the “Nociceptive Medullary Autonomic Circuit” (NMA pathway). It is deduced that the intraoperative stimulation of the NMA pathway (nociception) is clinically translated into alterations of the heart frequency, blood pressure, that could compromise the function of the different organs and tissues.

This nociceptive response, classically interpreted as “intraoperative pain”, was treated or managed by administering systemic analgesics, mainly opioids (fentanyl, remifentanil...) which often led to an overdose of these drugs with an increased incidence of their side effects (nausea, vomiting, tolerance, hyperalgesia, urinary retention, decreased level of consciousness, delayed awakening, hypoventilation...). Many of these side effects are of particular interest in certain subspecialties such as neuroanesthesia, where a delay in awakening, a decreased level of consciousness, or the occurrence of perioperative nausea and vomiting from the use of opioids could lead to misdiagnosis or compromise the outcome of surgery.

Therefore, the application of analgesic techniques/drugs must be done:

1. With the aim of guaranteeing optimal analgesia when the patient is awake (with the level of consciousness preserved, in the absence of hypnosis)
2. Ideally from the preoperative period (“preventive analgesia”: analgesic intervention prior to the appearance of a harmful stimulus aimed at reducing or avoiding subsequent pain) and throughout the perioperative period.

By acting in this way, we will avoid that in the period of “awakening from the anesthesia” the nociception is transformed into pain. Pain is a too complex phenomenon to be tackled with only one type of drug (opioids). Thus, there are different classes of drugs that act at different levels to achieve the best possible degree of analgesia:

1. Loco - regional techniques with local anesthetics. From surgical wound infiltration to neuraxial blocks.
2. Agonists a - 2: Clonidine, Dexmedetomidine. They modulate the noradrenergic response at the central nervous system level.
4. NMDA receptor antagonists: Ketamine, Magnesium sulfate. They are drugs with multiple effects: analgesics, antihyperalgesics, opioid - sparing.
5. Intravenous Lidocaine: Immunomodulatory effect, analgesic, antihyperalgesic, Glucocorticoids (Dexamethasone): multiple effects, modulate the systemic inflammatory response.
6. NSAIDs. Anti-inflammatory and analgesic effect.
7. Opioids: Very effective in established pain (not in all types) but should not be used to modulate intraoperative nociception [1-3].

Bibliography