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The Need for a Clearer Distinction between Anesthesia and Analgesia in Relation to the Opiate System

To the Editor:—The article published in your journal related to halothane and cerebral metabolic effects is of great interest.[1] However, the statement, "Naloxone has been reported to reverse the analgesic effects of a variety of drugs and therapies other than narcotic including nitrous oxide, halothane, enflurane, cyclopropane, pentobarbital. . . ."[1] raises a number of issues which we would like to mention.

There is some work which suggests that nitrous oxide, particularly in analgesic doses, does in fact have narcotic properties, closely resembling morphine.[2–5] This interaction could be mediated either by nitrous oxide causing the release of endogenous opiates or by direct receptor activation as suggested by Gillman and Lichtigfeld.[4] It is unlikely that nitrous oxide causes the release of endogenous opiate substances, since nitrous oxide does not seem to alter titers of these substances in human CSF (Y. Hosobuchi: personal communication). Agents with marked analgesic properties at subanesthetic concentrations may in fact be opiate receptor agonists. This analgesic property is most clearly seen with substances with high blood/gas solubilities such as methoxyflurane and trichlorethylene; nitrous oxide, however, is an exception to this rule as it acts rapidly and has a low solubility. The work of Artru *et al.*[1] highlights the considerable experimental evidence against naloxone acting as an antianesthetic agent. However, we know that naloxone reverses the analgesic effects of "non-opiate" agents. particularly nitrous oxide, as previously mentioned.

We, therefore, would like to suggest that more information might be derived from the study of agents in which the distinction between anesthetic and analgesic effects is more obvious, particularly as the latter effects may be opiate receptor mediated.

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The Dorsal Root Ganglion as a Site of Blockade during Epidural Anesthesia

To the Editor:—It is unfortunate that Cusick *et al.*[1] failed to examine the dorsal root ganglion as a site of blockade during epidural anesthesia, or even to cite our work on this subject.[2,3] Ironically, at the same 1979 ASA meeting where these authors presented their work, Galindo and Witcher[4] presented their findings on this subject. Galindo tested our results using complex neurophysiological techniques and confirmed our conclusion that the dorsal root ganglion is the most sensitive intradural structure to anesthetic block. These findings could account for threshold sensory anesthesia during both spinal and epidural block.

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