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Application of Practice Guidelines to Anesthesiology

FOUR recent studies in the anesthesia literature have reported successful and similar programs to change anesthesiologists' practice patterns.[1–4] This issue of ANESTHESIOLOGY contains one such report.[1] Two studies (by Freund *et al.*[1] and Lubarsky *et al.*[2]) tested the success of their efforts to change practice patterns by comparing drug use during a historical control period to that in a later intervention period at single hospitals. The other two studies (by Cohen *et al.*[3] and Rose *et al.*[4]) performed such an analysis at a control and a study hospital. Increases in the proportion of patients treated using the practice guidelines were then compared between the two hospitals. All four studies simultaneously monitored changes in patient demographics and outcomes. The studies examined practice guidelines for neuromuscular blocking drugs,[1] expensive anesthetic drugs,[2] prevention of nausea,[3] and management of postoperative pain.[4]

Comparison of how these groups achieved changes in anesthesiologists' practice patterns are important. Two studies[1,2] succeeded at changing anesthesiologists' practice patterns by combining education, practice guidelines, and paper barriers. Completion of a form at an operating room pharmacy[1] or advance approval of the attending anesthesiologist[2] was required to obtain the more expensive drugs. The former study[1] showed that the paper barriers produced changes that were maintained for 2 yr. The latter study[2] discussed that maintenance of the changed behavior was achieved, in part, by providing anesthesiologists with individualized feedback on their (1) deviation from the practice guidelines and (2) drug administration compared with their peers. The other two studies[3,4] succeeded at changing practice patterns by combining education, practice guidelines, and individualized feedback. The individualized feedback included the number of patients cared for during the promoted measures.

Together, the results of these[1–4] studies stand in contrast to some previous studies in anesthesiology that failed to show that education and practice guidelines change physicians' practice patterns. Anesthesiologists'

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practice patterns can be changed[1-4] to decrease costs,[1,2] to try to improve patient outcome,[3,4] or both. However, changing practice patterns requires more than just education and practice guidelines. Either "negative" barriers limiting undesired practice (*e.g.*, signatures, voice release, or forms),[1,2] "positive" individualized feedback,[3,4] or both[2] should be combined with education to change physicians' behavior.

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"Exciting" Aspects of Opiate Receptor Signal Transduction

THE process that causes a cell to produce a particular response after binding of an agonist to its receptor is called *signal transduction.* Of particular interest to anesthesiologists is the transmembrane signaling that follows agonist binding to ligand-gated ion channels (*e.g.*, γ-aminobutyric acid$_A$ receptors) or G protein-coupled receptors (*e.g.*, adrenergic and opiate receptors). In the former case, the receptor and the channel that translocate chloride anions are on the same protein, whereas in the case of the opiate receptors at least three separate proteins participate in the signal transduction; *i.e.*, the receptor, the G protein, and the effector. With that many "moving parts," the opportunity to modulate such a system abounds. When one considers that each family of receptors has many subtypes (*e.g.*, there are nine adrenergic receptor subtypes), which can couple to more than 20 different G proteins and nearly 100 effector mechanisms, it becomes easy to understand how a single species of agonist can give rise to a plethora of diverse biologic responses.

In this issue of ANESTHESIOLOGY, Gutstein *et al.*[1] tested the hypothesis that opiate receptors are coupled to mitogen-activated protein kinase cascades, which induce changes in cellular function by phosphorylation of cytoplasmic and nuclear proteins. Until a few years ago, such a notion would have seemed farfetched because the hyperpolarization effects of opioids in neuronal cells could be easily explained by a well-characterized activation of potassium channel (promoting escape of intracellular cations), inhibition of calcium channels (preventing calcium from entering into the cell), or both. Although these transduction mechanisms can explain the inhibitory effect of opioids on neuronal excitability, they do not provide an answer for the multitude of excitatory effects (*e.g.*, tolerance, dependence/addiction, muscle rigidity) that opioids also exhibit at the cellular level.

The authors have developed cDNAs for each of the rat opiate receptors, which, when spliced into an appropriate vector, can be introduced into cells, which then express "pure" populations of these receptor subtypes. The fidelity of transfection and protein expression en-

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