Hypotension after Indigo Carmine

To the Editor—Recently Shir and Raja described four patients who became unpredictably hypotensive after the administration of indigo carmine intravenously during otherwise stable surgical and anesthesia conditions. Their anesthetic technique included a high epidural block produced by bupivacaine in three patients and lidocaine and tetracaine in one patient. The indigo carmine was injected 80 min after induction of anesthesia in one patient and 2 h after induction in the other three. In discussing the possible causes of hypotension induced by a drug that usually causes hypertension in intact awake humans, they list an idiosyncratic reaction as most plausible, and I would like to suggest another possibility.

Various agents cause both stimulant and depressant effects simultaneously, the predominance depending on the prevailing circumstances. For example, carbon dioxide is a central stimulator of the vasomotor center and a peripheral local depressant. In an anesthetized animal model, carbon dioxide causes hypertension, but when sympathetic blockade is induced, subsequent administration of carbon dioxide causes further hypotension. Weiskopf et al. have shown that ketamine, which normally results in sympathetic stimulation but also has depressant properties, causes hypotension comparable to an equipotent but small induction dose of thiopental in a hypovolemic model, despite a measured increase in circulating catecholamines. Presumably, in this instance, the stimulant effect is offset by the lack of volume, and the depressant component prevails. Nitrous oxide behaves similarly.

I suggest that the authors consider an effect similar to the above as one possible cause of the phenomenon that they have documented. Possibly, in this instance, the usually observed stimulant effect was prevented by the sympathetic block caused by epidural blockade by long-acting agents, leaving a depressant component of the drug unmasked. The pharmacology of indigo carmine is not described clearly, so one can only conjecture. I am unaware of the exact site of action at which the usually observed stimulant effect is initiated. Could it be at the vasomotor center? Could there always be a 5-HT-like effect such as the authors describe, which is unmasked only when sympathetic blockade prevents the usually more dominant stimulant effect? If my hypothesis is correct, this is hardly an idiosyncratic reaction. Should the title of the report have been “Indigo Carmine-induced Severe Hypotension in Patients with High Epidural Blockade”? Should the Food and Drug Administration indicate that the drug sometimes causes hypotension but define the circumstances? As is so often the case, we need to know more!

Professor of Anesthesia
Stanford University School of Medicine
300 Pasteur Drive
Stanford, California 94305-5115

References
4. Weiskopf RB, Bogert MS, Roizen MF, Reid IA: Cardiovascular and metabolic sequelae of inducing anesthesia with ketamine or thiopental in hypovolemic swine. Anesthesiology 60:214–219, 1984
5. Weiskopf RB, Bogert MS: Cardiovascular actions of nitrous oxide or halothane in hypovolemic swine. Anesthesiology 63:500–516, 1985

(Accepted for publication September 17, 1993.)