Third, little is known about the interactions between psychotropic drugs such as haloperidol or chlorpromazine and ketamine in the central nervous system. What is appropriate and specific treatment for functional psychosis is not necessarily protective in the case of organic psychosis.

The indication for the use of ketamine by these authors was "repeated procedures." This is, I believe, not enough of an indication, given the possible disastrous consequences of ketamine psychosis. Although their patient did not seem to fit the category, there are patients who are severely burned who come to the operating room with a "Burn Unit (ICU)"-type of psychosis superimposed upon previous schizophrenia. In this author's experience, the addition of ketamine may make the already difficult postoperative management of such patients almost impossible.

Unless criteria for the safe use of ketamine in psychosis can be established or ketamine is clearly the best alternative despite severe emotional problems, its use should be proscribed in management of psychotic patients. If there is any question, a psychiatric consultant should be called in to help make the decision. Just because a patient "can be" anesthetized safely under certain conditions does not mean he should be. Let us not get caught-up in the—"All Indians walk in single file; at least the one I saw did"—approach to medical management.

Frank Guerra, M.D.
Assistant Professor of Anesthesia and Psychiatry
University of Colorado
Medical Center
Denver, Colorado 80262

REFERENCES

1. Reames E, Rosenblatt R: Ketamine anesthesia of a catatonic schizophrenic patient. Anesthesiology 51:577-8, 1979
(Accepted for publication February 29, 1980.)

Clonidine Withdrawal, Propranolol, and Rebound Hypertension

To the Editor:—Bruce et al. stated that the continued presence of beta-adrenergic blockade during preoperative cessation of clonidine administration in severely hypertensive patients would aggravate rebound hypertension. They attributed this to unopposed alpha-adrenergic effects of elevated catecholamine levels resulting from clonidine withdrawal. Consequently, they recommended withdrawal of beta blockers such as propranolol before discontinuation of clonidine administration. Their guidelines, however, ignore the significant contributions that beta blockers can make to the parenteral therapy of severe hypertension. Other authors have used beta blockers in combination with either alpha antagonists or other vasodilators during temporary interruption of clonidine therapy. Adequate doses of phenoxycbenzamine or hydralazine will minimize unbalanced vasoconstriction.

The authors also recommended the use of hydralazine as a substitute for clonidine. Reduced systemic vascular resistance during hydralazine therapy may lead to tachycardia and increased cardiac output and myocardial oxygen consumption (mediated by baroreceptor reflexes), together with renin release. These responses reduce the effectiveness of hydralazine and render it inadequate as a sole agent in the treatment of severe hypertension. Moreover, the agent is known to precipitate angina pectoris in hypertensive patients with coronary-artery disease. Beta blockers can minimize these unwanted reflex effects and thus serve as valuable adjuncts to hydralazine.

Finally, beta blockers potentiate the hypotensive effects of sodium nitroprusside, which is often used for control of perioperative rebound hypertension. This will significantly reduce the dose of sodium nitroprusside needed for this purpose.

Lawrence Kushins, M.D.
Assistant Professor
Department of Anesthesiology
New York University
New York, New York 10016

REFERENCES


(Accepted for publication February 29, 1980.)