More Light on Rauwolfia

In the early days there was endless discussion on the choice of anesthesia for one problem or another, but for obvious reasons unanimity of opinion was seldom achieved. With greater sophistication most of these problems are settled today on an individual basis, choice depending upon knowledge of the disease in question, the patient's uncommon characteristics and an understanding of what anesthetics can do. No sooner had this approach been adopted than a new subject was substituted for debate—the possible untoward response to anesthesia of patients given potent therapeutic drugs beforehand. Just to mention a few, first came the adrenal steroids, then repercussions following the use of antibiotics, fleeting references to Antabuse and lately the commotion over tranquilizers and antihypertensive medications. Argument is not a peculiarity of anesthesiology as witnessed by the dispute over the proper surgical treatment of peptic ulcer, the internist's dilemma in reversing auricular fibrillation and perhaps the obstetrician's many approaches to relief of pain during labor. Preoccupation with these things makes for beneficial intellectual exercise, but all will admit that the patient must be treated sensibly until such time as a correct answer is found.

The *Rauwolfia* compounds have aroused the most interest. Antihypertensive drugs and many of the useful tranquilizers have actions at one locus or another on the delicate system of balances in the autonomic nervous system, especially on the sympathetic regulation of circulation. These drugs are often given to patients with a variety of cardiovascular diseases. It was anticipated, therefore, that their combination with the circulatory depressant properties of anesthetics would create hypotension. Yet to draw sweeping conclusions concerning the dangers of such medication was a big step to take on little more than the theoretical background. To deny a patient the benefit of these drugs preoperatively contradicts, in a sense, the attractive hypothesis that sympathetic reactivity may be harmful—the basis for the use of vasodilator drugs in shock, the application of controlled hypotension, artificial hibernation and neuroleptanalgesia—and acceptance of the primary circulatory depressant properties of halothane.

Two reports in this issue of the Journal, the clinical investigation by Katz *et al.* and the experiments by Bagwell and his co-workers, throw more light on *Rauwolfia* in anesthesia, and by implication demonstrate how similar problems should be approached. It is reassuring evidence of the growth of knowledge that we have arrived at an acceptable solution so soon after the first alarm was raised. In the interval since 1956 not only was the action of reserpine on tissue stores of catecholamines clarified, but methods become available and were applied to study the interrelation among anesthetics, the sympathetic nervous system and endogenous secretion of catecholamines. As a by-product, more has been learned of the action of sympathomimetic amines, for the use of reserpine, like cocaine used as a pharmacological tool, or denervation of receptors, permits a distinction to be made among vasopressors as to their direct or indirect actions. If there is a problem of hypotension in connection with reserpine and anesthesia the effective vasopressor now can be given.

The general conclusion of all studies in the past year is that anesthesia can be given safely to patients who have received reserpine in therapeutically effective dosage up to the time of operation. A parallel position probably can be taken with regard to other kinds of medicines. This does not imply that adverse phenomena will not be encountered nor that anesthesiologists can afford to be more complacent. Safe anesthesia depends upon the preparation for it, mentally and otherwise, as well as its thoughtful administration. This whole affair has been good exercise and a good lesson, for its examination, dispassionately, points the way toward solution of future problems.

Leroy D. Vandam, M.D.