induced preparations. These data are interesting in light of the report of Haugen *et al*.,[7] who have demonstrated four electrophoretically distinct cytochrome P-450 proteins. Phenobarbital treatment induces only one of these proteins. It is possible that the protein induced by phenobarbital treatment catalyzes the pathway primarily leading to free fluoride formation. The effects of any of the hundreds of other enzyme-inducing agents on the quantitative and qualitative aspects of anesthetic biotransformation are unknown.

These reports reflect a growing interest in anesthetic biotransformation and the increasing experimental sophistication needed to obtain useful information from these studies. It is clear that an important factor in anesthetic toxicity relates to metabolism and, it seems only reasonable, that the more that is known about anesthetic biotransformation the safer will be our anesthetic practice. We have come a long way from the era when inhalation anesthetics were thought to be classic examples of pharmacologically active but metabolically inert compounds.

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Circulation

HYPERTENSION AND MENTAL STATUS The authors examined the course of modest (mean arterial pressure 110 torr) hypertension in 19 patients. Five received no therapy. Fourteen patients were instructed in "a technique based on Buddhist meditation exercises designed to elicit a relaxation response." They were told to repeat the technique twice a day for 10–15 minutes. After six months, no change was observed in the control patients. In the experimental group, an average decrease of 12 torr in mean arterial pressure had occurred. The plasma level of dopamine-beta-hydroxylase (an indicator of sympathetic nervous system activity) was significantly decreased at the end of six months in the experimental group but showed no change in control patients. No change in either plasma volume or peripheral venous renin level was found in either group. There was a significant increase in furosemide-stimulated renin activity in the treated group. Since adrenergic activity may influence renin secretion, the authors hypothesize that this decrease resulted from diminished sympathetic activity. The authors conclude that for certain patients, a psychotherapeutic modality of therapy may be efficacious in treating hypertension. Furthermore, the decreased blood pressure appears to be associated with reduced peripheral adrenergic activity. (*Stone RA, DeLeo J: Psychotherapeutic control of hypertension. New Engl J Med 294:80–84, 1976.*)