BRIEFS FROM THE LITERATURE

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Briefs were submitted by Drs. C. M. Ballinger, Lee S. Binder, John P. Bunker, M. T. Clarke, R. A. Devloo, J. A. Eastwood, J. E. Eckenhoff, Martin Helrich, J. R. Householder, S. J. Martin, R. E. Ponath, Alan D. Randall, R. W. Ridley, and H. S. Rotenstein. Briefs appearing elsewhere in this issue are a part of this column.

MONOAMINE OXIDASE INHIBITOR
Central nervous system stimulants are found in compounds such as: (1) sympathomimetics, particularly the phenylalkylamines (ephedrine, amphetamine, methamphetamine), and the newer phenethylamines (pipradol, methylphenidate, phenmetrazin); (2) variations of certain antihistaminic structures which have yielded both tranquilizers and central stimulants (phenyltoloxamine); (3) anticholinergic cogeners of atropine (benactyzine); (4) local anesthetics; and (5) ganglionic stimulants (nicotine, lobeline). However, the majority of these central stimulants have certain structural features contained in norepinephrine, acetylcholine, histamine and serotonin. The replacement of an amino group by a hydrazine moiety in a series of sympathomimetic amines has yielded a number of potent central stimulants which produce their effect both by direct stimulation of the central nervous system (amphetamine-like), and by monoamine oxidase inhibition in the brain. This prevents metabolic destruction of endogenous central excitatory amines (norepinephrine and dopamine). (Biel, J. H., Nuhfer, P. A., and Conway, A. C.: Structure and Activity Relationships of Monoamine Oxidase Inhibitors, Ann. New York Acad. Sc. 80: 568 (Sept. 17) 1959.)

MONOAMINE OXIDASE INHIBITORS
Side effects associated with chronic use of monoamine oxidase inhibitors are: orthostatic hypotension, periorbital and dependent edema, subclinical and possibly clinical jaundice, constipation, delayed micturition, impotence (and its opposite), pruritis, gastritis, anemia, and in one instance, uremia. In roughly 70 per cent of psychiatric patients, a reerudescence of psychopathology occurs accompanied by hyperactivity, irritability, agitation and assaultiveness. Neuromuscular systems include flaccidity, paresthesias and twiching. (Bailey, S. d’A., and others: Comparison of Iproniazid with other Amine Oxidase Inhibitors, Including W-1544, IB-516, RO 4-1018, and RO 5-0700, Bull. New York Acad. Sc. 80: 652 (Sept. 17) 1959.)

MONOAMINE OXIDASE INHIBITORS
The histological changes in the liver of patients who developed jaundice following the intake of iproniazid represent different stages and degrees of a diffuse hepatic parenchymal disease, the picture being similar to that considered characteristic for viral hepatitis. The main difference is the higher incidence of more severe alterations in the iproniazid group. (Popper, H.: Hepatic Injury in Patients Who Have Received Iproniazid, Bull. New York Acad. Sc. 80: 928 (Sept. 17) 1959.)

MONOAMINE OXIDASE INHIBITORS
Monoamine oxidase inhibitors in large doses led to a decrease in hemoglobin and in the number of red cells observed. Histological examination revealed other signs of erythrocyte destruction: hemosiderosis of spleen, liver and kidney; increased cellularity of bone marrow; and hyperemia of the spleen. Some compounds were also hepatotoxic, including severe swelling, fatty infiltration, and necrosis of the liver. Liver damage was usually accompanied by some degenerative changes and fatty infiltration of the kidneys. Prolonged treatment with therapeutic doses produced no changes in reproductive organs of rats. Higher doses, however, caused