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Nervous System

MONAMINE OXIDASE INHIBITORS The duration of action of norepinephrine released from cytoplasmic storage granules of neurones upon neuronal stimulation is largely limited by uptake again into nerve cytoplasm and storage granules and to a much lesser extent by diffusion away from the nerve ending and enzymatic destruction by catechol-O-methyl transferase. The chief function of monamine oxidase present in the mitochondria of adrenergic nerve endings may be to metabolize cytoplasmic norepinephrine that leaks from storage granules and escapes re-uptake. Monamine oxidase inhibitors cause an increase in neuronal stores of norepinephrine, but the mechanisms for disposing of norepinephrine released as a neurotransmitter or injected into the circulation are not disturbed other than by the limitation on tissue uptake imposed by the greater tissue stores. In experiments with human volunteers taking monamine oxidase inhibitors it was found, as expected, that injected norepinephrine is not potentiated. Ephedrine acts by releasing stored norepinephrine and since these stores are increased by monamine oxidase inhibitors the action of ephedrine is substantially potentiated. Phenylephrine acts chiefly directly on receptors and only to a small extent by release of stored norepinephrine and little potentiation occurs after intravenous administration, as expected. Substantial amounts of monamine oxidase are found in the intestine and liver and since phenylephrine acts as a substrate for monamine oxidase, administration of an inhibitor must be expected to increase absorption of phenylephrine. In 3 subjects, amounts of phenylephrine which barely caused blood pressure rise when given orally before monamine oxidase inhibitors, caused such dramatic blood pressure rise after inhibitors that the administration of the alpha blocking agent, phentolamine, was necessary. The inhibitors used in this study were phenelzine and tranylcypromine. (*Elis, J., and others: Modification by Monoamine Oxidase Inhibitors of the Effect of Some Sympathomimetics of Blood Pressure, Brit. Med. J. 1: 75 (April) 1967.*)