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ABSTRACTS OF PAPERS

(An asterisk (*) following an author's name indicates "by invitation"; a (T) before a title indicates "read by title".)

The anticonvulsant activity and toxicity of 3-methyl-1-pentyn-3-yl sodium succinate. WILLIAM M. ALEXANDER*, LAWRENCE C. WEAVER, EARL R. BOCKSTAHLER*, ALICE B. RICHARDS* AND BENEDICT E. ABREU. *Research Dept., Pitman-Moore Company, Indianapolis, Ind.* The anticonvulsant effectiveness of 3-methyl-1-pentyn-3-yl sodium succinate (methyl pentynyl succinate) and six other anticonvulsive agents including methylparafynol was determined in mice utilizing the maximal electroshock seizure (M.E.S.), Metrazol seizure (Met.), and "psychomotor" seizure (PsM.) tests. The oral doses of each drug protecting 50% of the mice by each test (ED_{50}) and the doses producing minimal neurologic dysfunction in the same percentage of animals (TD_{50}) were determined at the time of peak anticonvulsant activity (PAT). The PAT for methyl pentynyl succinate was 3.5 hours and its TD_{50} was 980 ± 75 mgm./kgm., while the PAT for methylparafynol was 1.5 hours and its TD_{50} was 251 ± 32 mgm./kgm. Although the potency and toxicity of methyl pentynyl succinate was less than that of methylparafynol, their anticonvulsant profiles were quite similar. Comparative protective indices for methyl pentynyl succinate and methylparafynol were, respectively: M.E.S. 1.37, 1.26; Met., 2.48, 2.35; PsM., 2.20, 1.69. These data suggest that methyl pentynyl succinate may act by *in vivo* release of methylparafynol to produce its anticonvulsant effects.

Attempts at the objective evaluation of ataraxis and analgesia in patients and experimental animals. J. L. AMBRUS, C. M. AMBRUS, R. O. BAUER AND W. K. NOELL*. *Roswell Park Memorial Institute and Hosp., Buffalo, N.Y.* Volunteer medical students and patients with psychopathic disorders and various pain problems were studied weekly for periods up to 4 months. Galvanic skin reflex, skin temperature, skin oxygen tension, EEG, ECG and respiratory rate were simultaneously recorded. Anxiety reactions were provoked by the following means: auditory stimuli (sharp bell, clap), electric stimulation of the palm at the lowest noticeable level ("tingling") and at the level of pain or shocking threshold, warning the patient of an imminent shock verbally or by ringing a bell and thermal stimulation at the pain threshold. Anxiety reactions could be reduced by chlorpromazine in doses which did not alter the pain threshold to all stimuli except electric or thermal stimuli at the pain threshold level. Morphine in small doses (4–8 mgm. i.v.) exhibited an effect similar to chlorpromazine. Morphine in larger doses (8–24 mgm. i.v.) increased the pain threshold and decreased reactions to all stimuli. Various adrenergic and ganglionic blocking agents, barbiturates in subhypnotic doses and antihistamines in doses producing drowsiness were ineffective in altering anxiety reactions. Amphetamine decreased drowsiness and sleepiness produced by chlorpromazine without altering its effect on anxiety reactions.

Rats were trained to jump through a barrier from one compartment to the other if a bell is sounded or if electric stimulus (below the pain threshold level) is applied to their paws. Chlorpromazine or small doses of morphine (1–4 mgm./kgm.) obliterated these reactions, but did not alter pain threshold: the animals jumped if stimuli were applied at the level of the pain threshold. Larger doses of morphine increased the pain threshold.

The above procedures are correlated and