increases in body temperature or heart rate cause concern during the time of the illness. Ataxia occurred early, dysarthria, restlessness, hallucinations, coma and convulsions have all been described following this method of administration of atropine. The dosage of atropine varied from 4.3 mg. to 15 mg. in the cases reported in this series. While no fatalities occurred in this series, deaths have occurred with as little as 1.6 mg. of atropine. A possible relation is suggested between the occurrence of these reactions and a high environmental temperature and humidity. (*Hoefnagel, D.: Toxic Effects of Atropine and Homatropine Eye Drops in Children, New Engl. J. Med. 264: 168 (Jan. 26) 1961.*)

HYDROCORTISONE FOR HYPOTENSION Fourteen patients who had cranial trauma, brain tumor, epilepsy, subarachnoid hemorrhage and meningitis who subsequently developed an acute fall in blood pressure were treated successfully with hydrocortisone. The stress resulting from these conditions seems to indicate the need for hydrocortisone when unexplained acute hypotension occurs. The evidence of a failing circulation in such patients should not be falsely interpreted as atypical signs of cerebral edema or compression. (*Shenkin, H. A.: Acute Hypotension Treated Successfully with Hydrocortisone, New Engl. J. Med. 264: 645 (Mar. 30) 1961.*)

CURARE EFFECT ON CORTEX The effect of curare on the cerebral cortex was studied by recording the direct cortical response to stimulation. Microelectrodes were used to record from single cortical cells of the exposed brains of cats. Curare administered intravenously caused variable degrees of depression of direct cortical response. Topical application of curare caused consistent augmentation of the response. Curare and strychnine have similar effects on single cortical neurons. The specific action of curare on the central nervous system is considered to be excitatory, perhaps as a result of blockade of inhibitory synapses. (*Morlock N., and Ward, A. A., Jr.: Effects of Curare on Cortical Activity, Electroenceph. Clin Neurophysiol. 13: 60 (Feb.) 1961.*)

RELAXANT DRUG THERAPY A 12 year program of relaxant drug therapy in cerebral palsy was reviewed. In four of the relaxants tested, none of which included the muscle relaxants commonly used in surgery, the placebo effect was greater than the drug effect. Decisions regarding relaxant drug therapy must be made with caution as in discriminate acceptance of a new relaxant drug must be rigidly evaluated in terms of the neural, motor and behavioral effects against placebo responses. (*Denhoff, E., and Holden, R. H.: Relaxant Drugs in Cerebral Palsy: 1949–1960, New Engl. J. Med. 264: 475 (Mar. 9) 1961.*)

EXTRAPYRAMIDAL REACTIONS Phenothiazine derivatives may cause toxic symptoms of extrapyramidal tract involvement even with low doses well in the therapeutic range. Reactions may be characterized by jitteriness, constant movements, insomnia, muscle spasm, torticollis, opisthotonos, carpopedal spasm, dysphagia, trismus, protrusion of the tongue, oculogyric crises, dysphonia and tonic seizures. Reactions usually are not dangerous and subside in from 4 to 48 hours after drug withdrawal. Sedation with barbiturates alone often produces alleviation of symptoms. In severe reactions, an antiparkinsonian drug such as benztropine (Cogentin) methanesulfonate is indicated. The muscle relaxant, methocarbamol (Robaxin), has also been found effective in controlling symptoms. (*Sobel, A.: Treatment of Extrapyramidal Reactions to Phenothiazine Derivatives, U. S. Armed Forces Med. J. 11: 12 (Dec.) 1960.*)

Abstractor's Note: Much to the nostalgic regret of many an "Old Vet," the Armed Forces Medical Journal was discontinued with the December 1960 issue.