

jects suffering from asthma and emphysema. The effective dose of atropine was less than 0.4 mg. Its effects were complementary to those of ephedrine. Stramonium cigarettes and atropine aerosols were also effective. (Herxheimer, H.: *Atropine Cigarettes in Asthma and Emphysema*, *Brit. Med. J.* 2: 167 (Aug. 15) 1959.)

**LUNG MASSAGE** Following two deaths due to complete bronchospasm, possibility of its treatment by thoracotomy and massage of the lung was considered. Experimental work on dogs using bronchospasm produced by usually fatal doses of neostigmine showed 100 per cent survival by massage of the lungs. The method was also successful in one man. (Smolnikoff, V. P.: *About Lung Massage*, *Der Anaesthetist* 8: 350 (Dec.) 1959.)

**PRESSURE OXYGEN BREATHING** Oxygen administration at 2.0 atmospheres during exercise lowered ventilation, restored arterial pH and  $p\text{CO}_2$  toward resting levels and caused venous  $p\text{CO}_2$  to rise above the resting level; this suggests a slight elevation of cerebral blood flow or reduction in the rate of cerebral oxygen consumption during exercise breathing oxygen at 2.0 atmospheres, without gross elevation of cerebral  $p\text{O}_2$ . (Lambertsen, C. J., and others: *Respiratory and Cerebral Circulatory Control During Exercise at .21 and 2.0 Atmospheres Inspired  $p\text{O}_2$* , *J. Appl. Physiol.* 14: 966 (Nov.) 1959.)

**CENTRAL NEURO TRANSMITTERS** Minimal criteria to be met by any naturally-occurring substance suggested as a chemical transmitter at central synapses are: 1) The substance should be distributed in the central nervous system not in a uniform, but rather in a discrete pattern. It should be ascertained that it is elaborated in the neurone and released from the presynaptic nerve terminal. Local concentrations of the substance should be related to the function of a given neural structure, and fluctuation in local concentration will take place in response to functioning of such a structure or will lead to a quantitative change in its function. 2) Enzymatic mechanisms should exist for its synthesis and destruction. 3) Increase or decrease in local

concentration of the substance (produced either directly or by the systematic administration of a metabolic precursor or inhibitor of a destructive enzyme) should produce demonstrable effects. 4) Known blocking agents should also produce demonstrable effects. Acetylcholine, norepinephrine, 5-hydroxytryptamine, histamine, substance P and gamma-aminobutyric acid are considered in the light of these criteria. (Giarmán, N. J.: *Neurohumors in the Brain*, *Yale J. Biol. Med.* 32: 73 (Nov.) 1959.)

**OXYGEN CONSUMPTION** Brain slides of cats were used to measure oxygen consumption under the influence of hexobarbital and thiopental, respectively. Hexobarbital depressed to the same extent resting metabolism and the metabolism that had been stimulated by potassium. Thiopental, on the other side, depressed the potassium-stimulated metabolism more than the resting one. (Heeg, E., and Weis, K. H.: *About the Decrease of Oxygen Consumption of Brain Slides Due to Hexobarbital and Thiopental*, *Der Anaesthetist* 8: 318 (Nov.) 1959.)

**INTRAMUSCULAR CHLORPROMAZINE** Two ml. of 2.5 per cent chlorpromazine injected near the brachial artery in the upper arm caused arterial spasm, thrombosis of the artery and dry gangrene of the arm distal to the site of injection. Stellate block and local infiltration with procaine as well as tolazoline caused no improvement and intra-arterial papaverine little improvement. Probably only surgical excision would have been limb saving. (Hamer-Hodges, R. J.: *Gangrene of Forearm After Intramuscular Chlorpromazine*, *Brit. Med. J.* 2: 918 (Nov. 7) 1959.)

**CHLORPROMAZINE** Reflex spasm of a non-thrombosed coronary vessel may be one of the first signs of acute cardiac infarction. On the ECG this is reflected in shortening of the ST interval. An inverted T wave and shortening of the ST interval in leads  $V_2$ , 3 and 4 in a case of an infarct of the posterior wall are explained as being due to a reflex spasm of the left coronary vessel. The reflex spasm of a nonthrombosed coronary vessel may be warded off by blocking the abnormal