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 Anaesthesist 8: 350 (Dec.) 1959.*)

PRESSURE OXYGEN BREATHING Oxygen administration at 2.0 atmospheres during exercise lowered ventilation, restored arterial *p*H and pCO_2 toward resting levels and caused venous pCO_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 pO_2. (*Lambertsen, C. J., and others: Respiratory and Cerebral Circulatory Control During Exercise at .21 and 2.0 Atmospheres Inspired pO_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. (*Giarman, 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 Anaesthesist 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

viscero-cortical reflex arc with largactil (chlorpromazine). (*Mandl, D., and Kenedi, I.: Reflex Spasm of the Coronary Vessels in Acute Cardiac Infarction, Terap, Arkh. 5: 56 1958.*)

INTRA-ARTERIAL THIOPENTAL In rabbits thiopental given intra-arterially produces transient arterial spasm of the femoral artery. Tissue slough in rabbit ears following intra-arterial thiopental appears to be due to some property of the drug other than its alkalinity. More dilute solutions produce less necrosis. The area of necrosis is not lessened by intra-arterial vasodilators or local anesthetics, but is reduced by sympathectomy and anticoagulants. (*Kinmouth, J. B., and Shepherd, R. C.: Accidental Injection of Thiopentone into Arteries, Studies of Pathology and Treatment, Brit. Med. J. 2: 914 (Nov. 7) 1959.*)

NEW ANALEPTIC A study of Micoren (Prethcamid), a mixture of aliphatic amines, has shown its analeptic action sufficient to abolish barbiturate anesthesia in animals without causing convulsions or vomiting. It increases the depth of respiration and causes a slight rise in blood pressure. It has been shown to produce a persistent reversal of respiratory depression due to morphine, meperidine, thiopental, and hexobarbitone. (*Dobkin, A., and Mitchell, D.: Micoren in Barbiturate Poisoning, Canad. M. A. J. 81: 1009 (Dec. 15) 1960.*)

INSULIN AND LIVER The livers of anesthetized dogs were cannulated and perfused *in vivo*. Following a control period insulin was administered via the portal venous catheter. The perfusate was sampled at the outflow and analyzed for glucose content. Liver glycogen of biopsied specimens was determined. Glucose values rose slowly from 85 to 150 mg. per 100 ml. with insulin perfusion and plateaued at 200 to 300 mg. per 100 ml. by 60 to 90 minutes. Serum potassium levels were minimally changed. Liver glycogen values, although highly variable, were not significantly increased following insulin. These data are interpreted as an unchanged hepatic glucose output at approximately 2 to 3 mg. per kg. per minute. Chronic experiments were also performed on unanesthetized dogs whose portal, hepatic, and splenic arteries had been catheterized. Simultaneous blood samples were taken from each catheter at 5 to 15 minute intervals before and after insulin. A fall in glucose concentration was observed 5 minutes after administration of insulin, which became maximal in 30 minutes and was followed by a rapid recovery to near normal values. However, while increased uptake in nonhepatic splanchnic tissues occurred, no increase in uptake was noted by the liver. These two experiments suggests that insulin has no direct effect upon the liver but produces an uptake of glucose by the nonhepatic splanchnic tissues. (*Shoemaker, W. C., and others: The Hepatic Glucose Response to Insulin in the Unanesthetized Dog, J. Biol. Chem. 234: 1631 (July) 1959.*)

CATECHOLAMINES AND SKIN To differentiate between those changes in tissue oxygenation which were the result of stress per se and those of reflex release of epinephrine and norepinephrine occasioned by stress, tissue oxygen tension was measured polarographically in six normal adults given infusions of epinephrine and norepinephrine while breathing room air at rest. With norepinephrine the systolic blood pressure rose an average of 37 mm. Hg, the diastolic 31 mm. Hg, while the pulse rate decreased 18 beats per minute. With epinephrine, the systolic blood pressure rose an average of 34 mm. Hg; however, the diastolic pressure decreased an average of 6 mm. Hg and the pulse rate increased 14 beats per minute. Skin oxygen tension decreased an average of about 25 per cent during the infusions. The etiology of these decreases was not determined. However, the decrease seen with norepinephrine is interpreted to mean that the drug acts primarily by decreasing the rate of delivery of arterial blood to skin as a result of intense vasoconstriction, decreased cardiac output or both. The decrease associated with epinephrine may be attributed to increased oxygen utilization and possibly cutaneous vasoconstriction. (*Greene, N. M., Davis, M. T., and Bell, J. K. S.: Skin Oxygen Tension During Administration of Epinephrine and Norepinephrine to Normal Man, Yale J. Biol. & Med. 32: 93 (Nov.) 1959.*)