physema, J. Clin. Invest. 39: 724 (May) 1960.)

PULMONARY DIFFUSION Carbon monoxide equilibration tests indicate a nonuniformity of diffusion throughout the lung. Diffusion is quantified in terms of compartments which vary in their diffusing characteristics. In normal subjects, the lung behaves as though 6 to 18 per cent of its volume contained 12 to 46 per cent of its diffusing capacity. Total diffusing capacity calculated by compartmentation methods is not significantly different from that calculated by the conventional breath-holding technique. In patients, variable degrees of diffusing capacity/lung volume nonuniformity are noted. This phenomenon appears to produce an unpredictable error in the breath-holding method in abnormal subjects. True over-all diffusing capacity cannot be accurately assessed by conventional techniques in patients with marked pulmonary abnormalities. *(Burrows, B., and others: Non-Uniform Pulmonary Diffusion as Demonstrated by Carbon Monoxide Equilibration Technique: Experimental Results in Man, J. Clin. Invest. 39: 943 (June) 1960.)*

HYPOVENTILATION By interfering with the chest bellows mechanism, marked obesity alone may cause alveolar hypoventilation to the extent that cyanosis, polycythemia, hypercapnea, pulmonary hypertension, and right sided heart failure develop. The management of this problem may require artificial ventilation in an attempt to achieve lower levels of carbon dioxide and resensitization of the respiratory center. *(Scalettar, R., and others: Alveolar Hypoventilation and Cardiopulmonary Failure in Obesity, U. S. Armed Forces Med. J. 11: 774 (July) 1960.)*

OXYGEN THERAPY DANGER Oxygen was administered to 40 dogs during the recovery phase after clinical death lasting 5 minutes and resulting from massive loss of blood. It was found that hypoxia following clinical death results in increased sensitivity to high concentration of oxygen with rapid development of hyperoxia and death of animals. This was the cause of death (within 24 hr.) of 13 of the 40 dogs. Fifteen dogs were not affected by oxygen therapy and the life of 8 dogs was prolonged by oxygen therapy to 5–97 days. Oxygen therapy was effective only in the case of 4 dogs the spontaneous respiration of which was reestablished only after some delay (4.5–7 min.). The use of 100 per cent oxygen for the purposes of artificial respiration during resuscitation is considered inadvisable. During the recovery period the dosage of oxygen should be strictly controlled. It is useful only as a 40–50 per cent mixture in an oxygen tent in which the animal is placed not earlier than 80–112 minutes after resuscitation and for not less than 4 hour. *(Smirenskaya, E. M., and Romanova, N. P.: Oxygen Therapy During Period of Recovery after Clinical Death, Byull. Eksper. Biol. i Med. 46: 66, 1958.)*

RADIATION THERAPY Radiosensitivity of cells with low oxygen supply may be reduced by two thirds. Such tumor cells can thus survive the largest possible doses of X-ray. With increasing oxygen tension the radiosensitivity increases rapidly up to a certain point beyond which there is only slight further increase. Patients were given oxygen under 4 atmospheres of pressure in a pressure chamber (45 pounds per square inch gauge pressure). Intravenous anesthesia was used. Bilateral myringotomy was done prior to the first treatment. Results over a three year period have been encouraging. *(Foster, C. A., Churchill-Davidson, I. F. J., and Thomlinson, R. H.: Anesthesia for Radiation Therapy Under High Oxygen Pressure, Der Anaesthesist 9: 157 (May) 1960.)*

AMINE INHIBITORS Reduction in the formation of amines is associated with the lowering of blood pressure and inducing transient sedative effects in hypertensive patients. α-Methyl-3,4-dihydroxy-*d,l*-phenylalanine (α-methyl-dopa), a compound known to be an effective inhibitor of aromatic amine acid decarboxylation in man, was given and urinary serotonin, tyramine, and tryptamine were determined. Urinary levels of these amines were significantly decreased over controls. Patients who received the drug showed significant reduction in blood pressure, and evidence of sedative and tranquilizing effects. This study demonstrates the relationship of

amine metabolism and regulation of blood pressure. The sedative effects are also in line with the current thoughts concerning the role of amine metabolism in relation to brain function, *e.g.* decreased serotonin levels lead to decreased brain function and therefore sedative effects. (*Oates, J. A., and others: Decarboxylase Inhibition and Blood Pressure Reduction by α-Methyl-3,4-Dihydroxy-*dl*-phenylalanine, Science 131: 1890 (June 24) 1960.*)

THIOPENTAL AND BLOOD LIPID Thiopental anesthesia in dogs and rats was accompanied by a sharp fall in blood nonesterfied fatty acids and a small increase in blood sugar. No pronounced changes in the blood concentrations of cholesterol, phospholipids or fatty acid esters were observed. Ether anesthesia had no effect on the blood nonesterfied fatty acids in rats. Hence, the fall in nonesterfied fatty acids during thiopental anesthesia is not related to the anesthesia itself. (*Fodor, J., and Grafnetter, D.: Influence of Thiopentone Anesthesia on Blood Lipid and Blood Sugar Level, Brit. J. Pharmacol. 15: 282 (June) 1960.*)

HYDROXYDION Hydroxydion (Viadril) causes a moderate constriction of the renal afferent and efferent arterioles similar to ether, cyclopropane and thiopental. There is a transient moderate reduction of glomerular filtration rate and renal plasma flow rate. (*Kunz, F., and Reubi, F.: The Behavior of Renal Hemodynamics during Hydroxydion Narcosis, Der Anaesthesist 9: 197 (June) 1960.*)

BLOOD ETHER LEVELS Figures concerning ether content of venous blood of 29 patients during the main forms of ether anesthesia are provided. Analysis of the results shows that under certain conditions the concentration of ether in the blood can serve as an index of the depth of anesthesia. The stage of analgesia was associated with a blood ether content of 19–32 mg./100 ml. The corresponding figures for the stages of excitement and surgical anaesthesia were 32–62 mg./100 ml. and 70 mg./100 ml., respectively. During combined ether-oxygen anesthesia similar depths of anesthesia were associated with similar blood ether contents. (*Zhilis, B. G.: Ether Content of Blood During Various Types of Ether Anaesthesia, Khirurgiya 6: 34, 1959.*)

ALCOHOL After ingestion of a single dose of ethanol there is an early diuresis accompanied by increased urinary 17-hydroxycorticoid output and lower plasma levels, followed by decrease output in urine and an increase in plasma 17-hydroxycorticoids to base-line values within 12 hours. Active adrenal stimulation is suggested by the finding that a significant drop in blood eosinophils at 4 hours after ingestion occurs. Therefore, increased adrenocortical function after ethanol ingestion in man was demonstrated. (*Kissin, B., Schenker, V., and Schenker, A. C.: Acute Effect of Ethanol Ingestion on Plasma and Urinary 17-Hydroxycorticoids in Alcoholic Subjects, Amer. J. Med. Sc. 239: 690 (June) 1960.*)

ALCOHOL Intravenous infusion of 2 g./kg. of ethanol depresses both serotonin and norepinephrine levels in the brain stem of the rabbit. The parallel decrease of both neurohormones persists long after ethanol disappears, an effect somewhat similar to that of reserpine. Chronic infusion of alcohol for 7 days produces a 50 per cent decrease in both serotonin and norepinephrine levels in the rabbit brain stem. (*Gursey, D., and Olson, R. E.: Depression of Serotonin and Norepinephrine Levels in Brain Stem of Rabbit by Ethanol, Proc. Soc. Exp. Biol. & Med. 104: 280 (June) 1960.*)

MEPROBAMATE Meprobamate overdosage in a 19 year old girl caused stupor, decreased muscle tone and disproportionate hypotension, the blood pressure falling until both it and the pulse were unobtainable. Mephentermine, and also dilute norepinephrine drip which was continued for eleven hours, raised the blood pressure promptly. No evidence of shock occurred, the skin remaining warm and pink. (*Stevens, A. E.: Hypotension Due to Meprobamate Overdosage, Brit. Med. J. 1: 1029 (April 2) 1960.*)

PHENOTHIAZINE ATARAXICS Extrapyramidal reactions to phenothiazine lead to hospitalization of 39 patients at Los Angeles General Hospital during 1958 to 1960. Most