thrombolysis or fibrinolysis as a result of increased fibrinolysin activity and consequent hemorrhage, particularly following pulmonary, cardiac, pancreatic, and prostatic surgery. Administration of certain drugs or hormones also increase this tendency—epinephrine, vitamin K, nicotinic acid, PABA, and others. Lysis was determined by dissolution in a test tube of the organized clot over a period of 4 to 8 hours. In five patients who had a prostatectomy, use of fat emulsion intravenously resulted in subsequent control of hemorrhage and reduction of *in vitro* clot lysis. (*Biggs, A. W., and others: The Use of Fat Emulsion IV in Control of Hemorrhage Due to Thrombolysin Activity, South. Med. J. 54: 1252 (Nov.) 1961.*)

ANTICOAGULANT ANTAGONIST Coumarin anticoagulant therapy is frequently accompanied by the administration of other drugs, including barbiturates. Results obtained in guinea pig, dog and man indicate that pretreatment with barbiturates may antagonize the hypo-prothrombinemic effect of coumarin anticoagulants. This effect has been correlated with lower plasma levels of the anticoagulant drugs. In man, the route and timing of dosage significantly influence this barbiturate effect. It may be necessary to pay more attention to the mutual influence of concomitant therapy on the physiological disposition of drugs employed. (*Dayton, P. G., and others: Influence of Barbiturates on Coumarin Plasma Levels and Prothrombin Response, J. Clin. Invest. 40: 1797 (Oct.) 1961.*)

CATECHOLAMINES Catecholamines have a variety of metabolic effects. The one that has received the most emphasis is the stimulation of glycogenolysis as a consequence of the activation of glycogen phosphorylase in liver, muscle, myocardium, intestinal smooth muscle, adipose tissue, and other tissues. The glycogenolytic effect of epinephrine results in a parallel increase in blood glucose and lactate. Other metabolic effects are hyperkalemia, inhibition of glucose uptake by tissues, mobilization of fat involving an increase in plasma free fatty acids, inhibition of the incorporation of amino acids into muscle protein, and inhibition of cholesterol and fatty acid synthesis. A beta-adrenergic antagonist (dichloroisoproterenol) was used to study the metabolic aspects of adrenergic blockade. DCI has previously been shown to prevent both epinephrine-induced augmentation of contractile force and activation of phosphorylase in the dog heart *in situ*. It was found that DCI almost completely abolishes the increase in blood glucose and free fatty acids produced by epinephrine, norepinephrine, and isoproterenol in the dog. The hyperlactic acidemic effect of epinephrine is partly blocked. However, DCI does not block epinephrine-induced hyperglycemia in mice. (*Mayer, S., Moran, N. C., and Fain, J.: Effect of Adrenergic Blocking Agents on Some Metabolic Actions of Catecholamines, J. Pharmacol. Exp. Ther. 134: 18 (Oct.) 1961.*)

THIOBARBITURATES The extent to which the liver of humans or other species can carry out the reaction C=S yields C=O in the barbiturates remains somewhat uncertain. The reaction occurs with thiourea and many of its aryl derivatives. Thiourea is formed in small yield from thiopental metabolism along with a considerable yield of inorganic sulphur. This reaction may be of no more than minor importance in the metabolic disposition of thiobarbital. Barbital is well known to be stable in the animal body and to be excreted practically quantitatively in the urine, so that its quantitative determination is an uncomplicated indicator of the extent of desulfurization of its thioanalogue. It was found that barbital is excreted in the urine of man very slowly and in very small amounts after the intravenous injection of moderate doses of pure thiobarbital. The 5 to 7 per cent barbital yield and the 1 to 5 per cent thiobarbital excreted shows that some other metabolic reactions are responsible for the fate of about 90 per cent of the thio compound. (*Bush, M. T., Mazel, P., and Chambers, J.: Metabolic Fate of Thiobarbiturates: Thiobarbital in Man, J. Pharmacol. Exp. Ther. 134: 110 (Oct.) 1961.*)

ATROPINE Reflex parotid secretion and heart rate were measured in nine volunteers at intervals before and after injection of graded

doses of atropine sulfate, ranging from 0.5 to 2.0 mg. Effective inhibition of the salivary reflex and vagal blockade were observed after doses of 2.0 mg. of atropine sulfate per 70 kg. of body weight. Doses of 0.5 mg. per 70 kg. were almost completely ineffective. Doses of 1.0 mg. per 70 kg. caused about 50 per cent inhibition of reflex parotid secretion which lasted not longer than about 45 minutes. (*Stewart, W. C., and Currie, H.: Relationship Between Dosage of Atropine and Effects on Reflex Parotid Secretion and Heart Rate in Man, Canad. Med. Ass. J. 85: 780 (Sept. 30) 1961.*)

ANALGESICS On comparing the analgesic efficacy of meperidine (Demerol) and phenazocine (Prinadol) in obstetrical patients, no statistically significant difference was found. Phenazocine caused less nausea and vomiting. (*Corbit, J. D., and First, S. E.: Clinical Comparison of Phenazocine and Meperidine in Obstetric Analgesia, Obstet. Gynec. 18: 488 (Oct.) 1961.*)

OXYGEN TOXICITY The demonstration of the efficacy of trishydroxymethyl aminomethane ('tris') in buffering tissue carbon dioxide and acidity suggested that it might provide protection against the toxic action of oxygen at high pressures. Rats administered 'tris' before exposure to oxygen at high pressures demonstrated that protection was provided. The onset of oxygen seizures was postponed and their incidence and severity decreased. Lung damage was either absent or much less severe than in control animals. Lung weight was lower and the mortality rate much decreased. The results not only demonstrated the protection provided by 'tris,' but redirected attention to increased tissue carbon dioxide tension and tissue acidity as possible contributors to the precipitation of the toxic reaction to oxygen at high pressures. (*Bean, J. W.: Tris Buffer, Carbon Dioxide and Sympatho-adrenal System in Reactions to Oxygen at High Pressures, Amer. J. Physiol. 201: 737 (Oct.) 1961.*)

ANALGESIA POTENTIATION In the study of the efficacy of giving an ataractic drug in addition to an analgesic drug for relief from the discomfort of parturition, a phenothiazine derivative (promethazine) and a benzoquinolizine derivate (Nitomar) were compared with each other and with meperidine alone. The employment of a tranquilizer in addition to meperidine was significantly more effective that meperidine alone in decreasing the patient's pain or anxiety responses. (*Fromhagen, C., and Carswell, A. P.: Potentiation of Analgesia During Labor: Study of Two Tranquilizers, Obstet. Gynec. 18: 483 (Oct.) 1961.*)

ANTIEMETIC Sixty-one patients developed nausea and vomiting after a variety of operations under barbiturate-cyclopropane-nitrous oxide-oxygen anesthesia. They were treated with intravenous administration of 200 mg. trimethobenzamide (Tigan). Eighty-five per cent had complete relief in one to ten minutes, average four minutes. Duration of relief was five hours. Blood pressure, pulse and respiration were unchanged. (*Kolodny, A. L., and Shane, S.: Trimethobenzamide, A. M. A. Arch. Surg. 83: 775 (Nov.) 1961.*)

HYPOTHERMIA A marked reduction in the flow of pancreatic secretions takes place in dogs under hypothermia of 34 to 28° C. Complete cessation of flow takes place at 28° C. A moderate decrease in the enzyme concentration in the serum and pancreatic juice also is seen during hypothermia. (*Symbas, P. S., and others: Influence of Hypothermia on Pancreatic Function, Ann. Surg. 154: 509 (Oct.) 1961.*)

HYPOTHERMIA Hypothermia from 14 to 28° C. in dogs produced a metabolic acidosis with excess lactate due to hypoxic metabolism. There was a fall in *p*H with the P_{CO_2} low or normal. The metabolic acidosis was not due to decrease in cardiac output or to arterial oxygen unsaturation. During warming, there was a further drop in *p*H and a rise in P_{CO_2} owing to increased carbon dioxide production. At the same time, excess lactate fell. Employing a special animal preparation, it was shown that the metabolic acidosis of hypothermia results from different rates of cooling of various parts of the body. (*Ballinger, W. F., and others: Acidosis of Hypothermia, Ann. Surg. 154: 517 (Oct.) 1961.*)