also metabolizes serotonin and dopamine. The enzyme is inhibited by iproniazid. It is widely distributed in animal tissue such as liver, kidney, intestine, brain, blood vessels, placenta and lungs where it is found largely (or exclusively) in mitochondria. Because it is located in tissues where true cholinesterase levels are low, it has been suggested that just as cholinesterase is responsible for destroying acetylcholine so monoamine oxidase may inactivate a neurohormonal transmitter, such as noradrenaline or dopamine. Serotonin and monoamine oxidase show a parallel distribution in dog and cat brain, the levels being highest in the hypothalamus. Like acetylcholine, serotonin is apparently present in nervous tissue in a precursor state and seems to be active only when in a free form. It may have a role in central neurotransmission, being liberated momentarily in a free and active form and then rapidly destroyed. That monoamine oxidase may be responsible for the destruction of biogenic amines *in vivo* has been poorly supported by experimental evidence, but it probably plays a small but definite part in the *in vivo* metabolism of histamine, adrenaline and noradrenaline. (*Davison, A. N.: Physiological Role of Monoamine Oxidase, Physiol. Rev. 38: 729 (Oct.) 1958.*)

NEOSTIGMINE TOXICITY When the patient suffering from myasthenia gravis passes into a state of respiratory crisis, as the maintenance dose of neostigmine is increased to meet the rising demand, its input may rapidly overtake its elimination and a cumulative effect be produced. Neostigmine in high concentration may act as a neuromuscular blocker in its own right and produce respiratory failure from muscle paralysis. Controlled intermittent positive pressure respiration must be continued for some days while the drug is eliminated and gradual weaning practised. (*Shackleton, P.: The Management of the Apnoeic Patient, Ann. Roy. Coll. Surgeons (England) 23: 187 (Sept.) 1958.*)

PHENOTHIAZINE Preanesthesic medication with Pacatal alone or in combination with meperidine or pentobarbital provided increased sedative and analgesic effects of the latter drugs, antiemetic benefits (reduction of vomiting incidence from 21 per cent to 7 per cent) and drying of the respiratory tract. No marked respiratory or circulatory depression occurred preoperatively, during operation, or postoperatively. This drug is preferred to other phenothiazine derivatives for use in anesthesia. (*Corssen, G., and Allen, C. R.: Clinical Evaluation of 10(N-Methyl-piperidyl-(3)-Methyl) Phenothiazine (Pacatal) for Use in Anesthesia, South. Med. J. 51: 689 (June) 1958.*)

MEGIMIDE Megimide and Metrazole give essentially similar effects in producing electroencephalographic effects or clinical seizures in epileptics. There appeared to be less undesirable side effects from Megimide but convulsions occurred. (*Rodin, E. A., and others: Megimide and Metrazol, Electroencephalography and Clinical Neurophysiology, 10: 719 (Nov.) 1958.*)

OXYGEN Rats were anesthetized with chloralose or with a barbiturate given intraperitoneally. After the necessary cannulations or operative procedures they were placed in a steel chamber under a pressure of six atmospheres of oxygen. There was no rise in carbon dioxide tension in the arterial blood before the onset of cardiac or respiratory failures. After respiratory failure carbon dioxide tensions attained very high levels. (*Taylor, D. W.: Changes in Cardiac and Respiratory Rates, and in Carbon Dioxide Pressure and pH of Arterial Blood, in Anaesthetized Rats Exposed to Oxygen under High Pressure, J. Physiol. 143: 149 (Aug. 29) 1958.*)

LIVER FUNCTION In anesthetized dogs subjected to elevated carbon dioxide tension, arterial blood levels of carbon dioxide ranged from 58 to 86 volumes per cent. Liver function as determined by removal of Bromsulfalein was consistently impaired. Blood pressure determinations indicated that circulation was not altered appreciably during the test period. (*Holmes, E. L., and Barnhart, M. I.: Effect of Increased Carbon Dioxide Retention on Liver Function in the Dog, J. Appl. Physiol. 13: 184 (Sept.) 1958.*)

RESERPINE Experiments were undertaken to determine the effect of reserpine-induced

depletion of norepinephrine stores of the heart on the response of the heart rate to stimulation of the accelerans nerve. Pre-treatment with two doses of reserpine (each 0.1 mg./kg. intraperitoneally) sensitizes the heart to the positive chronotropic action of norepinephrine and reduces the response to stimulation of the accelerans nerve. Ganglionic transmission remains unaffected. The results indicate that the presence of certain stores of peripheral sympathetic transmitter is essential for the production of tachycardia by stimulation of the accelerans nerve. (*Trendelenburg, V., and Gravenstine, J. S.: Effect of Reserpine Pretreatment on Stimulation of the Accelerans Nerve of the Dog, Science 128: 901 (Oct. 17) 1958.*)

MIDBRAIN POTENTIALS The effects of chlorpromazine and pentobarbital on evoked potentials in the midbrain reticular formation were investigated in 23 cats. The potentials recorded oscillographically in the reticular formation were evoked by stimulation of the sciatic nerve. Chlorpromazine enhanced the amplitude of single evoked fast and slow potentials, and markedly slowed the rate of recovery of both of these potentials. Pentobarbital on the other hand decreased the amplitude of single evoked potentials, and increased the "absolute" refractory period and slowed the rate of recovery from the "fast" potential. Chlorpromazine may act selectively by depressing the frequency response of neurones responsible for the "slow" potential recorded in the medial reticular formation. (*DeMaar, E. W. J., and others: The Effects of Chlorpromazine II: The Effects of Chlorpromazine on Evoked Potentials in the Midbrain Reticular Formation, J. Pharmacol. & Exper. Therap. 124: 77 (Sept.) 1958.*)

EXTRACORPOREAL CIRCULATION A rotating drum film type oxygenator is described wherein filming takes place on a disposable sheet of corrugated polyethylene which is sterilized by boiling. The remainder of the apparatus is fabricated from stainless steel and is sterilized in the autoclave. The arterial blood is filtered by gravity flow through a fine mesh nylon fabric before leaving the oxygenator. Oxygen and carbon dioxide removal was excellent for the flows studied in 10 dogs. The capacity of the oxygenator exceeded the flow rates that were employed in the animal experiments. (*Schimert, G., and others: A Rotating Drum Film Oxygenator, Surg. Gynec. & Obst. 107: 527 (Nov.) 1958.*)

EXTRACORPOREAL CIRCULATION The bubble dispersion oxygenator and the stationary screen oxygenator were compared under experimental and clinical conditions. Experiments were conducted in 65 dogs and clinical experience was gained over a two year period in 94 patients. Experimental data indicated that the screen oxygenator had some advantages over the bubble oxygenator in that it reduced the possibility of microscopic air embolism. However, clinical experience indicated that factors other than the type of oxygenator employed were of major importance. The excellent clinical results obtained by the bubble oxygenator demonstrated the device capable of providing satisfactory extracorporeal circulation. Current preference for the screen oxygenator is due in part to the fact that a progressive increase in experimental mortality occurred with prolonged duration of bypass with the bubble oxygenator. (*Maloney, J. V., Jr., and others: An Experimental and Clinical Comparison of the Bubble Dispersion and Stationary Screen Pump Oxygenators, Surg. Gynec. & Obst. 107: 577 (Nov.) 1958.*)

EXTRACORPOREAL CIRCULATION Use of extracorporeal circulation for more than one to two hours has been successful only in few cases, one of the important time limiting factors being the effects on the formed elements of blood and on certain labile plasma proteins. Despite clean, smooth non-wetting surfaces, certain obstructions for recording oxygen tension, *p*H and temperature present opportunities for trauma to blood. Coagulation defects and excessive bleeding are the major problems encountered. Possible causes are (1) incomplete neutralization of heparin (2) loss of platelets (3) activation of fibrinolysins (4) loss of denaturization of some clotting element, (5) coagulation activated by trauma, (6) activation of some circulating anticoagulant, (7) loss of capillary tone. Proper maintenance of minimal blood dilution, near