1971

# Year Book OF ANESTHESIA

ECKENHOFF

# THE YEAR BOOK
# *of*
# ANESTHESIA
# 1971

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## PHILOSOPHY, LOGISTICS AND UTILIZATION

**The Normality of the Abnormal** is discussed by William W. Mushin[1] (Cardiff, Wales). The practice of anesthesia is now dominated by a desire to maintain a "normal" state; "homeostasis" has become a key work in present practice. When the range of the physiologic functions of importance is considered, considerable differences are found on either side of an imaginary ideal state of health, but they are quantitative rather than qualitative. Some wide variations in function occur for no apparent reason, some from habits and others from natural factors such as age. The concept of "normality" as a fairly sharp physiologic point should be abandoned. Variation is the essential characteristic of living things. Surgical populations may be different from others in many respects. A great many surgically treatable conditions are associated with physiologic disturbances that are mostly compensatory or resultant, rather than causal in nature.

Postoperative personality disorders in aging patients may result from vigorous hyperventilation by an anesthesiologist attempting to maintain some form of homeostasis in terms of $P_{CO_2}$. Various effects of smoking may be important to the anesthesiologist, such as elasticity of the lung and a reduction in surfactant. The numbers that smoke make these changes "normal." Obesity is another matter on which an epidemiologic outlook must be developed. Circadian rhythm may modify the effects of anesthesia and surgery. Some of the difficulties or successes of induced hypothermia may be due to cyclic variations in temperature control. Circadian variations in blood pressure and in sensitivity of the central nervous system to various stimuli also occur. Persons who take sedatives may react differently than others to anesthesia. Age is important in reactions of patients to anesthetic procedures. Degeneration of aging affects neuronal tissue and also the blood supply to the central nervous system. Blood pressure changes with age may be of significance as may altering dose-effect ratios for drugs.

Programed anesthesia is expected in the future, with automated control of anesthetic gases modified by feedback of monitored physiologic events as they occur during anesthesia. Char-

(1) Anesth. & Analg. 49:667-679, Sept.-Oct., 1970.

acteristics of the "whole patient" must be identified, so that individual patients will not be matched against a theoretic, often unattainable physiologic idea to their possible disadvantage, but rather against a prototype drawn from actual knowledge of the particular defined population groups to which they belong.

► [We usually forget, and many never accept, that what is acknowledged as normal is but the average of the observations between the extreme.—Eds.]

**Analysis of Manpower in Anesthesiology** is presented by J. S. Gravenstein, J. E. Steinhaus and P. P. Volpitto.[2] Additional anesthesia services will be required as our medical resources are made available to the underprivileged. Anesthesiologists are assuming more responsibilities for patients in intensive care units, pulmonary care centers and obstetric services. It is not feasible to place an anesthesiologist with every patient requiring an anesthetic. Distinction may be made between simple standardized tasks, routine tasks responsive to general or specific direction, semiroutine tasks responsive to general direction, nonroutine tasks performed under little supervision and nonroutine tasks performed without supervision. Procedures vary in the degree of medical judgment required and the activities observed. Difficulties are more prone to arise during induction than during maintenance of anesthesia. Tasks have not yet been weighed in simulation experiments. Experience suggests that simple standardized tasks should be combined with routine tasks performed in response to general or specific directions. Semiroutine tasks may also be combined, as in the functions of nurses or nurse-anesthetists. Both nurse-anesthetists and anesthesiologists often perform tasks at lower levels than those who require their services.

Often the nurse-anesthetist is placed in an unfair position when asked to work independently or under the direction of a physician with little training in anesthesiology. Only a few anesthesia aides or technicians are presently available for tasks at the lowest level. The role of these persons must be better defined. A person should be able to rise to higher levels of responsibility as capability and desire permit or move laterally into related areas. Training of anesthesia technologists is directed at the increased use of technology in medicine, specifically, toward further use of physiologic measurement in anesthesiology. It is planned that the master technologist in anesthesi-

(2) Anesthesiology 33:350-357, September, 1970.

ology will complement the aide, nurse-anesthetist and anesthesiologist. An alternate approach is the training of assistants and associates in anesthesiology for specific tasks at the lower levels.

**Arterial Punctures by Nurses** were evaluated by Marvin A. Sackner, Wilbur G. Avery and Jean Sokolowski[3] (Mt. Sinai Hosp., Miami Beach, Fla.). Requests for arterial blood gas studies have risen markedly, and physicians in small hospitals have found it difficult to obtain such analyses readily as interns and residents are often unavailable. An attempt was made to train registered nurses to perform arterial punctures. Two nurses have performed 1,541 punctures since 1969 without morbidity or complications.

PROCEDURE.—With the patient supine or semirecumbent and the wrist extended, the area over the radial artery ½-1 in. above the wrist is cleansed and infiltrated with xylocaine. The artery is palpated with one hand. With the other hand, the needle is advanced into the artery at a 45-degree angle. A 3-5-ml. sample of blood is withdrawn. The brachial or femoral artery can be punctured if radial artery blood cannot be obtained.

Nurses were instructed for 1 week and have generally been able to perform all arterial punctures. Sampling usually required less than 2 minutes. The punctures done in 1969-70 included 127 brachial and 67 femoral punctures. No vasovagal reaction has occurred. The only morbidity was occasional minimal superficial hematoma formation over the puncture site.

Arterial punctures can be performed safely by nurses and ultimately by paramedical personnel, relieving the house staff of this burden. Local anesthesia should be used before arterial puncture. It reduces the tendency of patients to hold their breath or hyperventilate, interfering with a steady state, and reduces the frequency of vasovagal reactions. Nurses must be taught to ask about reactions to the "caine" type of drugs and to treat sensitivity reactions.

► [We suspect this will be greeted with cries of anguish by some. It makes sense to the editors.—Eds.]

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## BIOCHEMISTRY AND PHYSICAL CHEMISTRY

**Influence of Arterial Hypoxemia upon Labile Phosphates and upon Extracellular and Intracellular Lactate and Pyruvate Concentrations in the Rat Brain.** B. K. Siesjö and L. Nilsson[4] (Univ. of Lund) studied the effects of varying degrees of hy-

(3) Chest 59:97-98, January, 1971.
(4) Scandinav. J. Clin. & Lab. Invest. 27:83-96, February, 1971.

poxia on cerebral energy metabolism in male Wistar rats by relating arterial oxygen tension to brain tissue contents of phosphocreatine, ATP, adenosine diphosphate (ADP), adenosine monophosphate adenylic acid and inorganic phosphorus, as well as the cerebrospinal fluid lactate and pyruvate levels. Components of the glutamate-$\alpha$-ketoglutarate equilibrium were also analyzed in most brains.

Cisternal cerebrospinal fluid was sampled from animals anesthetized with divinyl ether and nitrous oxide. Hypoxia was induced for 30 minutes by reducing the oxygen flow at constant ventilation. Attempts were made to minimize variations in factors other than oxygen tension. The blood pressure tended to fall at a $Pao_2$ below about 25 mm. Hg; below 20 mm. Hg it almost always declined, and the exposure to hypoxia had to be reduced to 15 minutes.

Progressive monrespiratory acidosis developed with a $Pao_2$ below 45-50 mm. Hg. The whole blood lactate concentration and the lactate-pyruvate ratio increased with a $Pao_2$ below about 50 mm. Hg. A fall in base excess was correlated with these changes. Similar changes occurred in cisternal cerebrospinal fluid at low $PO_2$ levels. Brain phosphocreatine levels fell significantly and inorganic phosphorus increased with a $PO_2$ below about 35 mm. Hg. The ATP-ADP ratio fell moderately at a $PO_2$ below about 25 mm. Hg; changes were more pronounced in hypotensive animals.

Intracellular lactate levels increased at a $Pao_2$ not associated with significant changes in labile phosphates (35-45 mm. Hg). Cerebrospinal fluid changes exceeded those occurring in the intracellular space at low $Pao_2$ levels. At extremely low levels the greatest lactate accumulation was intracellular. The tissue $\alpha$-ketoglutarate content did not decrease until the $PO_2$ fell below 20 mm. Hg, and the drop was most marked in animals with hypotension.

The earliest tissue changes with arterial hypoxemia were increased in the intracellular lactate concentration and the lactate-pyruvate ratio. The energy metabolism of the brain is remarkably resistant to pure hypoxemia. The finding that cerebrospinal fluid changes were less marked than those in the cells at extreme degrees of hypoxemia may have been due to an insufficient equilibration time.

► [Interpretation of intracellular metabolic events is difficult even under the best circumstances. This study documents a disparity between intra-

cellular and cerebrospinal fluid lactate-pyruvate ratios and draws attention to the limitations of extracellular lactate-pyruvate measurements as indicators of intracellular hypoxia. More importantly, however, tissue levels of high energy phosphate compounds were well maintained in markedly hypoxemic states. The brain tolerated a $Pao_2$ of 30 mm. Hg or less without causing major changes in cerebral energy reserves and without evidence of threat to viability of cerebral cells, provided hypotension did not occur. It appears that maintenance of cardiovascular function is the critical factor in severely hypoxemic states.—Eds.]

**Reduced Red Cell Glycolysis, 2,3-Diphosphoglycerate and Adenosine Triphosphate Concentration and Increased Hemoglobin-Oxygen Affinity Caused by Hypophosphatemia** were studied by Marshall A. Lichtman, Denis R. Miller, Jules Cohen and Christine Waterhouse[5] (Univ. of Rochester). The red blood cell glycolytic rate and glycolytic intermediates are intimately related to red blood cell survival in the circulation and to the function of hemoglobin as an oxygen donor at tissue partial pressures of oxygen. Extracellular organic phosphate can influence the red cell glycolytic rate.

A woman aged 52 was seen for progressive peripheral neuropathy and intractable steatorrhea. An intestinal biopsy specimen indicated a sprue-like disorder, but there was no response to gluten proscription or high doses of prednisone. Intravenous feedings with 12% glucose and 4% casein hydrolysate were given. Positive nitrogen balance was observed during the period of parenteral feeding. The patient died of infection and gastrointestinal bleeding after 5 months. There was extensive necrosis of the gastrointestinal mucosa. Red cell metabolism was examined during the illness.

Plasma inorganic phosphate levels fell to extremely low levels when parenteral nutrition was instituted and were below 1 mg./100 ml. by 72-96 hours. Urinary phosphorus excretion fell to zero. There was a 60% drop in red cell glucose utilization and in lactate production. Lactate-glucose ratios remained constant. The red cell 2,3-diphosphoglycerate (2,3-DPG) level fell to 45% and the ATP level fell to 52% of baseline, corresponding temporally to the fall in the glycolytic rate. Phosphate infusion intravenously increased glucose utilization and lactate production, and the 2,3-DPG and ATP levels increased to normal in close parallel with the increase in plasma phosphate concentration. The low glycolytic rate of the red cells from the patient was partly corrected when the red cells were placed in normal plasma. The glycolytic rate of normal red cells declined

(5) Ann. Int. Med. 74:562-568, April, 1971.

in the plasma from the patient. No important blood pH changes were noted. Oxygen affinity of hemoglobin was reduced in normophosphatemia and increased during protracted hypophosphatemia. Over half the red cells were spherocytes during protracted hypophosphatemia. Moderate anemia and slight reticulocytosis were present during the period of parenteral feeding. A direct Coombs test was negative.

Extracellular phosphate is important in modulating the glycolytic rate and the red cell ATP and 2,3-DPG levels in vivo. Red cell energy stores are critically reduced during marked hypophosphatemia, threatening red cell survival. Tissue oxygen delivery may be impaired under these circumstances.

► [The work of Benesch and Benesch on the regulatory effects of intracellular organic phosphate on oxygen release by hemoglobin is assuming greater clinical significance. Both 2,3-DPG and ATP bind to hemoglobin and decrease the affinity of hemoglobin for oxygen over the physiologic range of oxygen tensions. Since there is about 3 times as much 2,3-DPG in the red cell, its effect on Hb-$O_2$ binding is more important. In this case the observed hypophosphatemia was accompanied by a decrease in red blood cell 2,3-DPG, which in turn interfered with the ability of hemoglobin to release oxygen at the tissue level.—Eds.]

**Effect of Chronic Intake of Ethanol on Pentobarbital Metabolism** was studied by H. Kalant, J. M. Khanna and Joan Marshman.[6] Ethanol was given for 2 weeks to male rats on a liquid diet; ethanol constituted 5.1% of the diet and provided 36% of the total calories. The daily intake of ethanol was 10-12 Gm./kg. Control animals received an equicaloric amount of sucrose instead. Liquid diets were replaced by tap water 24 hours before the study. Phenobarbital sodium was given in a daily dose of 100 mg./kg. for a week, by gastric intubation, as a 1% aqueous solution. Sleep time was determined after the injection of pentobarbital, 30 mg./kg. in saline intraperitoneally; the interval between loss and return of the righting reflex was recorded. Plasma and brain levels of pentobarbital were determined. Pentobarbital metabolism also was studied in liver slices.

Ethanol-treated and control rats had similar growth. Sleep was of slower onset and shorter duration in the ethanol group. At 15 minutes after a pentobarbital dose, plasma and brain pentobarbital levels were lower in the study group, but the blood-brain ratios were almost identical in the two groups of rats. Pentobarbital disappearance in plasma or brain did not differ 30-60 minutes after administration. The rate of pento-

(6) J. Pharmacol. 175:318-324, November, 1970.