tension, or damage by hypoxia. It is always associated with severe pulmonary-artery hypertension. The patchy, generally hilar distribution, periartral hemorrhages, alveolar hyaline membranes and predilection for high-altitude-native healthy young men (who presumably have the most reactive pulmonary arterioles) all suggest an overdistention and traumatic rupture at the arterial level. A second possibility is overperfusion of some capillary beds whose arterioles are forced open by the severe hypertension. Milledge, iliff and I succeeded in obtaining perivasculer edema by hypoxic pulmonary hypertension in a dog lung even after complete unilateral blockade of the distal arterial bed with polystyrene microspheres 12 to 35 μ in diameter such that no capillary flow or hypertension was possible. However, the mechanism still needs study, since the microspheres could have been responsible for damage and leakage. The possibility of pulmonary edema and hemorrhage following severe hypoxia should be considered, since adequate ventilation and oxygenation are generally sufficient therapy.

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Meeting of the Austrian, German, and Swiss Societies of Anesthesiology and Reanimation

The Twelfth Joint Meeting of the Austrian, German, and Swiss Societies of Anesthesiology and Reanimation was held in Berne, Switzerland, September 1–3, 1971. In his introductory comments, B. Tschirren (Berne) noted the increased recognition of anesthesiology as a university discipline, manifested by the recent proliferation of medical school chairs of anesthesiology. He stressed the need for anesthesiologists to demonstrate their role in patient care by making themselves active rather than passive members of the surgical team, and by actively making patients aware of their participation.

A round-table discussion of Anesthesia and Respiration began with a review of respiratory physiology by J. P. Haab (Fribourg), whose O₂-flow diagrams clarified many factors involved in oxygen transfer from ambient atmosphere to tissues. He emphasized that oxygen flow is a function of gas conductance to the alveolus as well as of pressure differences across alveolar, vascular, and tissue membranes. H. Benzer (Vienna) reviewed the characteristics of mechanical ventilators and showed that volume-limited machines were more effective than pressure-limited ones in maintaining adequate ventilation when compliance or resistance was increased. The best machines are volume-regulated with pressure plateaus. H. Waversit (Heidelberg) discussed monitoring in the operative and postoperative periods and made a plea for attention to ventilation, hemodynamic status, blood–gas and air–gas studies, fluid balance, and electrolyte replacement. A similar emphasis on total patient care was underscored by P. Safar (Pittsburgh) in describing his experiences with prolonged artificial respiration in 2,000 intensive-care-unit patients and in 36,000 general anesthetics. Prolonged artificial ventilation must be tailored, depending upon whether a “healthy” or “sick” lung is involved. The healthy lung does well with most types of ventilation. Ventilation of the “sick” lung, for example, associated with massive trauma, must take into account and correct changes in the renal, circulatory, and central nervous systems.

G. Hössli (Zurich) directed a round-table discussion on Anesthesia and Circulation and examined normal circulatory physiology and the impact of cardiocirculatory abnormalities on anesthetic management. The importance of the preoperative visit in cardiovascular evaluation was emphasized. The postoperative death rate from a fresh infarction is less than 10 per cent in patients with minimal angina or one-year survival following a previous myocardial infarction. However, the presence of severe angina or a recent myocardial infarction increases this rate to 40–60 per cent. It was the view of the panel that prophylactic digitalization is rarely indicated.

Among the 100 individual papers presented, Y. Bapaci et al. (Geneva) discussed electron microscopic and morphometric changes in oxygen pneumonitides in humans. The earliest changes are seen within a few hours and con-
sist of capillary dilatation and accumulation of alveolar edema fluid, followed by massive swelling of epithelial cells. Within three days hyaline membranes form, edema spreads to the interstitium, and epithelial and endothelial cells degenerate, leading to a decrease in cell volume. After 6–13 days interstitial fibrosis occurs, with a decline in vital capacity. Initially, capillary volume increases, then decreases to 73 per cent of preoxygenation values after 14 hours and to 27 per cent after 13 days. Cessation of O₂ therapy prior to the twelfth day leads to considerable functional recovery. However, lung reserves are decreased and some degree of permanent change is always seen after more than 14 hours of O₂ therapy.

Several papers dealt with the effects of ketamine. L. Audi-Kolaric (Zagreb) reported its use in children, especially in burn patients. The circulatory stimulation seen with ketamine leads to increased bleeding and to arrhythmias. U. Braun et al. (Gottingen) reported that ketamine increased myocardial O₂ requirements, a finding also reported by H. Sonntag et al. (Gottingen).

Much interest in the clinical use of pancuronium was evidenced. J. M. Kapferer (Innsbruck) reported that a pancuronium–Ketalar mixture was highly successful in the poor-risk geriatric patient. K. Widemann et al. (Heidelberg) uses pancuronium in infants and children. Initial doses of 0.12 mg/kg produce good conditions for intubation in 10–15 seconds, and relaxation lasts 60 minutes.

In general, the meeting met its goals of providing an opportunity for the dissemination of general medical knowledge to anesthesiologists, as well as indicating the level and direction of anesthesia research in central Europe.

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Drugs and Their Actions

PROPRANOLOL The effects of propranolol on several responses to orthostatic and exercise stress were examined in healthy young men. Propranolol significantly reduced the heart-rate response to orthostatic stress, while systolic and diastolic blood pressures were unaffected. Propranolol reduced heart rate and minute ventilation significantly during bicycle exercise. However, oxygen consumption and mean exercise endurance times were not altered. The findings suggest that in healthy subjects the cardiac depressant effects of propranolol are compensated for, so that oxygen uptake and work capacity are not seriously compromised. (Maksud, M. G., and others: Effects of Propranolol on Several Physiological Responses during Orthostatic and Exercise Stress in Healthy Male Subjects, Canad. J. Physiol. Pharmacol. 49: 867–872, 1971.)