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$P_{aO_2}$  was calculated as  $P_B - P_{H_2O} - P_{aCO_2} - P_i$  anesth.  $P_{aO_2}$  was measured by polarographic analysis of an iced arterial sample corrected for the cooling time by the average change of Fletcher and Barber.<sup>15</sup>  $CaO_2 - C_{vO_2}$  was measured by the Van Slyke manometric method.

During the inhalation of low oxygen mixtures ( $F_{IO_2} = 0.25$ ), physiologic shunt was calculated by:

$$\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{(0.0032 \times P_{aO_2}) + (Sc \times 1.34 \times Hb) - CaO_2}{(0.0032 \times P_{aO_2}) + (Sc \times 1.34 \times Hb) - C_{vO_2}}$$

$P_a$  was obtained by the physiologic deadspace method of Filley<sup>16</sup>:

$$P_a = (P_B - 47) \times F_{IO_2} - P_{aCO_2}/P_{ECO_2}(F_{IO_2} - F_{EO_2})$$

#### APPENDIX

During inspiration of the high oxygen mixtures ( $F_{IO_2} = 0.80$  to  $0.90$ ) the physiologic shunt was calculated by:

$$\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{(A - aDO_2) \times \lambda \text{ body temp.}}{(A - aDO_2) \times \lambda + CaO_2 - C_{vO_2}}$$

$$Hb \text{ was calculated from } \frac{[CaO_2 - P_{aO_2} \times 0.0031]}{1.34 \times SaO_2}$$

$ScO_2$  and  $SaO_2$  were taken from the Severinghaus slide rule, knowing  $P_{aO_2}$ ,  $P_{aCO_2}$ ,  $pH_a$  and body temperature.

## Drugs

**ISOPROTERENOL** The effect of intravenous isoproterenol upon cardiorenal hemodynamics was studied in ten patients with, and three patients without, heart disease. Although cardiac output was increased in every patient in response to the drug, no significant change in glomerular filtration rate or renal blood flow was seen, and the percentage of cardiac output delivered to the kidney decreased. The data suggest that there is either a weak beta receptor response in the kidney or none. (Rosenblum, R., and others: *Effect of Acute Intravenous Administration of Isoproterenol on Cardiorenal Hemodynamics in Man*, *Circulation* 38: 158 (July) 1968.)

**IRREVERSIBLE HYPOGLYCEMIA** The glucose-lowering action of alcohol augments that of other hypoglycemic agents and may induce severe hypoglycemia with irreversible neurologic changes. In six healthy subjects infusion of alcohol during a standard insulin-tolerance test inhibited the usual rebound of glucose after hypoglycemia. Alcohol interferes with hepatic glycconeogenesis and induces hypoglycemia whenever glycconeogenesis is required to maintain normal glucose levels. Diabetics receiving other hypoglycemic agents should be warned about the blood-glucose-lowering action of alcohol. (Arky, R. A., and others: *Irreversible Hypoglycemia: A Complication of Alcohol and Insulin*, *J.A.M.A.* 206: 575 (Oct.) 1968.)