reduced arterial carbon dioxide tension on the cerebral vasculature. This study was undertaken to note the acute effects of hyperventilation on the oxygen and carbon dioxide tensions, and the *p*H of lumbar cerebrospinal fluid. *Method:* The investigation was performed on patients undergoing various neurosurgical operations who required cerebrospinal fluid drainage by inlying spinal catheter. Premedications consisted of pentobarbital 100 mg. and atropine 0.6 mg. given intramuscularly one hour preoperatively. Anesthesia was induced with thiamylal sodium 2½ per cent and succinylcholine, followed by endotracheal intubation. The patients were then allowed to breathe spontaneously a mixture of nitrous oxide and oxygen (3:2 or 2½:2½ liters/minute) and halothane (0.5–1.5 per cent) for 20–30 minutes before cerebrospinal fluid and arterial blood samples were taken for analysis. Then the patients were curarized and hyperventilated by means of a positive pressure respirator, the tidal volume being approximately double that read off the "Radford Nomogram," the inspired gas concentration remaining unaltered. After a minimum of 45 minutes, sampling of arterial blood and cerebrospinal fluid was repeated, the dura still being intact. *Results:* With hyperventilation the mean arterial carbon dioxide tension fell from 50 ± 9 mm. to 29 ± 8 mm. of mercury, this being paralleled by the mean cerebrospinal fluid carbon dioxide tension dropping from 50 ± 8 mm. to 39 ± 12 mm. of mercury. Associated with this, the arterial *p*H rose from a mean of 7.34 ± 0.04 to one of 7.52 ± 0.07 and the cerebrospinal *p*H from 7.32 ± 0.04 to 7.44 ± 0.05. Although the mean arterial oxygen tension (128 mm. Hg) did not change with hyperventilation, the mean oxygen tension of the cerebrospinal fluid fell from 73 ± 18 mm. to 59 ± 13 mm. of mercury, a decrease occurring in eight out of the nine patients. *Discussion:* Cerebrospinal fluid neither uses oxygen nor produces carbon dioxide and its gaseous content is the result presumably of diffusion from the tissues surrounding it. Any variation in the tension of carbon dioxide and oxygen in the cerebrospinal fluid is likely, therefore, to be a reflection of similar variations in the brain, spinal cord and their blood vessels. *Summary:* It has been demonstrated in this study that with hyperventilation, a fall in arterial carbon dioxide tension was accompanied by a similar fall in the carbon dioxide tension of the lumbar cerebrospinal fluid, the *p*H changing appropriately. The associated fall in the oxygen tension of the cerebrospinal fluid in 8 out of 9 patients, in spite of no change in the mean arterial oxygen tension, would indicate a fall in the oxygen tension of those tissues with which the cerebrospinal fluid is in contact. This could be due either to reduced perfusion or increased oxygen utilization.

The Effect of Halothane on Neuromuscular Transmission. A. J. GISSEN, M.D., J. H. KARIS, M.D., and W. L. NASTUK, Ph.D., *Departments of Anesthesiology and Physiology, Columbia University, College of Physicians & Surgeons, New York City.* *Method and Results:* Halothane was studied in the amphibian nerve-muscle preparation to determine its effect on peripheral neuromuscular transmission and at what site it acts. The desired anesthetic concentration was obtained by equilibrating Ringer's solution with a gas mixture of halothane plus oxygen. The blocking concentrations of halothane were found to be: 1.5 per cent for the nerve stimulated twitch, 4 per cent for axonal conduction and 4 per cent for the directly stimulated muscle. From this it was concluded that the peripheral blockade produced by halothane occurs at the neuromuscular junction. The neuromuscular junction was studied by microelectrode penetration of single fibers. Transmembrane resting potential was unchanged following exposure to 4 per cent halothane although the neurally evoked action potential was completely blocked. This was entirely reversible. The magnitude of postjunctional depolarization produced by applied carbachol in a bath and iontophoretically applied acetylcholine (ACh) were decreased by exposure to halothane. Miniature endplate potentials (MEPPs), which reflect postjunctional depolarizations produced by random quantal release of ACh from the nerve ending, were rapidly decreased by application of halothane, and returned to previous levels following its removal. These experiments show postjunctional membrane desensitization by halothane.

We examined the prejunctional effects by the following experiments. While recording from a postjunctional intracellular electrode a nerve stimulus was alternated with an iontophoretic pulse of ACh. Halothane depressed the depolarization response to both forms of stimulation equally. Hubbard and Schmidt (J. Physiol. *166:* 145, 1963) showed that if an external recording electrode is carefully placed one can record the action potential of the terminal nerve filament and distinguish it from the response of the postjunctional membrane. Exposing such a preparation to halothane resulted only in depression of the postjunctional membrane response. *Summary:* Halothane, even in moderate concentrations, gives evidence of causing peripheral neuromuscular blockade. The postjunctional membrane at the neuromuscular junction is the structure most sensitive to the blocking action of halothane.

Effect of Vasodilatation on Metabolic and Hemodynamic Parameters During and After Cardiopulmonary Bypass. THOMAS M. GLUSHIEN, M.D., and LEROY C. HARRIS, M.D., *Department of Anesthesiology, University of Pittsburgh School of Medicine and Presbyterian-University Hospital, Pittsburgh, Pennsylvania.* Reduction in oxygen consumption during, and acidosis following hypothermic cardiopulmonary bypass reflect a depression of metabolism and an impairment of tissue blood flow. Active vasodilatation should modify the peripheral vascular response. *Method:* Eight dogs were anesthetized with pentobarbital (20 mg./kg.), paralyzed with succinyldicholine, intubated, and ventilated (100 per cent O_2) with a piston ventilator (tidal volume 15 ml./kg.; rate 20/minute). The following parameters were monitored before, during and after bypass: (1) Esophageal temperature; (2) urine output; (3) heart rate (lead 2 ECG); (4) arterial pressure; (5) central venous pressure; (6) oxygen consumption (closed system spirometer technique); (7) cardiac index (Hamilton, W. F., and Remington, J. W.: Amer. J. Physiol. *148:* 14, 1947); (8) hemoglobin and hematocrit; (9) arterial *p*H, P_{O_2} and P_{CO_2}; and (10) standard bicarbonate and base deficit (calculated). After control measurements, cardiopulmonary bypass was conducted for 60 minutes at 32° C. (±1.3° C.). Perfusion was maintained at 100 ml./kg./minute (2.34 ± 0.11 liters/m.^{2}/minute) with an occlusive, load insensitive pump, resulting in a mean arterial pressure of 80 to 110 mm. of mercury. Oxygenation was maintained with a disc oxygenator. Four animals not actively vasodilated were used as controls (untreated animals). Four other animals were treated identically, except that vasodilatation was produced during bypass with trimethaphan camphorsulfonate (Arfonad) infusion to a mean arterial pressure of 50–60 mm. of mercury. Post-bypass, the animals were allowed to rewarm spontaneously and monitoring was continued for 180 minutes. *Results and Discussion:* During bypass there was no significant difference in oxygen consumption in the two groups; but the treated animals had lower standard bicarbonate, higher base deficit, and higher urine output. Thus with vasodilatation there was earlier and greater expression of anaerobic metabolism. During the post-bypass period, treated animals had higher oxygen consumption (115–118 per cent, $P < 0.001$) and urine output (170–190 per cent) despite a lower cardiac index (67.5–75 per cent, $P < 0.01$). Three hours post-bypass the treated animals developed a higher standard bicarbonate and lower base deficit (a reversal of the immediate post-bypass situation). Pre-bypass correlation of oxygen consumption with cardiac index showed wide scatter in both groups ($r = 0.240$, $P > 0.01$). Post-bypass correlation was significant in the untreated animals ($r = 0.797$, $P < 0.05$) and extremely close in the treated animals ($r = 0.960$, $P < 0.001$). Correlation of standard bicarbonate with both oxygen consumption and cardiac index was positive in the pre-bypass period, but became negative post-bypass in both treated and untreated animals. These correlations were generally not statistically significant. The close correlation of oxygen consumption with cardiac index, and the altered correlation of standard bicarbonate to these parameters suggests the "washout" of previously formed acid metabolites. After bypass the treated animals displayed a significantly greater decrease in arterial P_{O_2} (21.3–29.2 per cent of untreated animals, $P < 0.001$) and increase in P_{CO_2} (140–186 per