BRIEFS FROM THE LITERATURE

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CEREBRAL BLOOD FLOW Cerebral perfusion is controlled by regulation of perfusion pressure and by cerebral vascular resistance. Until recently, it was believed that cerebral vessels did not possess significant capacity for intrinsic control of vascular tone so that perfusion passively followed changes in arterial blood pressure. Recent quantitative studies in man have demonstrated that—within a wide pressure range—cerebral blood flow is independent of changes in arterial blood pressure. Only in marked hypotension is cerebral vasodilatation inadequate; and when cerebral blood flow falls to about 60 per cent of the control value, clinical signs of cerebral hypoxia become evident. Vascular reactivity to pressure changes appears to be unaffected by sectioning the vagus, cervical sympathetics, sinus and aortic nerves, or to local application of sympathomimetic drugs. Thus active regulation of cerebral vascular tone to pressure change may be a kind of autoregulation, possibly due to a direct effect of pressure change on vascular smooth muscle. Another impeding factor is local vasomotor activity of intracranial vessels. This activity is influenced largely by chemical or humoral factors. Carbon dioxide causes pronounced cerebral vasodilatation. Inhalation of 5 per cent carbon dioxide increases cerebral perfusion by about 50 per cent while 7 per cent carbon dioxide causes an increase of about 100 per cent. Conversely, hypocapnia produced by marked hyperventilation decreases blood flow to about 60 per cent of normal. This is the same low critical level as was found associated with clinical signs of cerebral hypoxia in severe hypotension. Although it is generally believed that the partial pressure of carbon dioxide is the important variable, no clear cut data are available to distinguish the action of carbon dioxide tension from that of associated changes in bicarbonate. Inhalation of air mixtures of low oxygen tension causes dilatation whereas oxygen tensions at 3 to 4 atmospheres cause a moderate vasoconstriction. Thus oxygen seems especially important in the homeostatic regulation of cerebral perfusion in the face of severe anoxia. Consciousness is lost when the oxygen tension of cerebral venous blood reaches 15–20 millimeters of mercury, and at this level, abnormal cortical electrical activity also starts abruptly. The same critical level of cerebral venous oxygen has been found to limit the vasoconstriction of hypocapnia in animal experiments. The effect of *p*H and of nervous influences is not clear. The influence on cerebral blood flow and oxygen uptake of physiological variables such as exercise, apprehension and sleep, pathologic variables such as coma, hypertensive encephalopathy and cerebral arteriosclerosis, and drugs such as depressants, convulsants and hormones is analyzed. Drugs which cause a depression of consciousness, *e.g.*, barbiturates, all cause a reduction in cerebral oxygen uptake proportional to the degree of cerebral depression. Surgical anesthesia reduces cerebral oxygen uptake by about 40–50 per cent. (*Lassen, N. A.: Cerebral Blood Flow and Oxygen Consumption in Man, Physiol. Rev. 39: 183 (April) 1959.*)

CEREBRAL CIRCULATION Carbon dioxide is a more powerful cerebral vasodilator and can produce greater increases in cerebral blood flow than any other chemical agent yet studied. Thiopental anesthesia and old age tend to lessen this effect. Therapeutic effects of carbon dioxide rebreathing, particularly in presence of hypoxemia, are discussed. The effects of most general anesthetic agents with the exception of ether, which has a specific vaso-