BRIEFS FROM THE LITERATURE

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Briefs were submitted by Drs. Gerald Allen, C. M. Ballinger, Lee S. Binder, John P. Bunker, M. T. Clarke, J. E. Eckenhoff, Martin Helrich, S. J. Martin, R. E. Ponath, William Rabenn, Alan D. Randall, Lawrence Reichmann, and H. S. Rottenstein. Briefs appearing elsewhere in this issue are a part of this column.

CARBON DIOXIDE Fifteen young mongrel dogs were lightly anesthetized either with pentobarbitone sodium or with one of the inhalants (ether, halothane or cyclopropane). Increasing concentrations of carbon dioxide were then added to the gas mixture in steps of five per cent every five minutes to a maximum of 75 per cent.

The various inhalants differed in the degree to which they reduced the stimulatory effect of carbon dioxide on minute volume. The reduction among the inhalants was most marked with ether and least marked with cyclopropane. Pentobarbitone caused even less reduction. The peak stimulatory response to ventilation was reached when carbon dioxide concentration was about 25 per cent. The increase in minute volume was achieved in some dogs principally by an increase in rate, in others by an increase in tidal volume. A further marked increase in minute volume occurred, if the inhalant was withdrawn at a carbon dioxide concentration of about 20 to 25 per cent. The stimulating effect of carbon dioxide then gradually decreased. At a concentration of about 40 per cent respiratory arrest supervened. If artificial ventilation was then maintained, while the carbon dioxide concentration was further increased, spontaneous respiration then recommenced, usually at a carbon dioxide concentration of 60 to 70 per cent, and at a minute volume which approximated control values. At this stage ("supercarbia") arterial and venous pressures, heart rate and rhythm, and respiratory minute volume remained essentially constant for more than an hour. Cutting of the vagi during this stage in three dogs produced the typical slowing and deepening of respiration.

Slow decrease in the carbon dioxide concentration to complete elimination led to the re-establishment of a normal respiratory pattern, after a short period of stimulation when the carbon dioxide concentration was reduced below 30 per cent (mirror-image of the respiratory response at the onset, when the carbon dioxide had been increased). (*Graham, G. R., Hill, D. W., and Nunn, J. F.: The Effect of High Carbon Dioxide Concentration on Respiration and Circulation, Der Anaesthesist 9: 70 (Feb.) 1960.*)

CARBON DIOXIDE A new carbon dioxide electrode separated from the blood or tissue by a membrane permeable only to carbon dioxide can be calibrated directly to read P_{CO_2}. P_{CO_2} on the surface of the brain in dogs was found to be about 55 mm. Hg. Liver and gut mucosa have high P_{CO_2} (about 80 mm. Hg). The rate of rise of arterial P_{CO_2} during apnea was studied in seven patients and found to average 4 mm. per minute after the initial rise from arterial to mixed venous level. The prominent effects of carbon dioxide on the circulation are three. The myocardium is depressed by high carbon dioxide concentrations and this depression may be partially reversed by adrenalin or noradrenalin, acetylstrophanthidin, or by buffering the *p*H to normal levels. Secondly, high concentrations of carbon dioxide cause direct vasodilitation of smooth muscle. Thirdly, the sympathetic nervous system is stimulated partly mediated through peripheral chemoreceptors, in part directly through the adrenal medulla, and in part through the central nervous system. Increased carbon dioxide also increases the irritability of the ventricle. Withdrawal from high carbon dioxide concentrations usually produces hypotension, which may be a complication in post-