ABSTRACTS

Editorial Comment: A fixed style of presentation for this department of ANESTHESIOLOGY has purposely not been defined. It is the wish of the Editorial Board to provide our readers with the type of abstract they desire. Correspondence is invited offering suggestions in regard to the length of abstracts, character of them, and source of them. The Board will appreciate the cooperation of the membership of the Society in submitting abstracts of outstanding articles to be considered for publication.

GELLHORN, ERNST, AND LAMBERT, E. H.: *The Vasomotor System in Anoxia and Asphyxia: A Study of the Adjustment Reactions of the Mammalian Organism.* In: Illinois Medical and Dental Monographs. Urbana, Illinois, The University of Illinois Press, 1939, vol. 2, p. 71.

"The present investigation was carried out in order to clarify the mechanism by which oxygen deficient gas mixtures with and without carbon dioxide influence the central nervous system. It had been observed by one of us (E. G.) that various cortical functions in man which are greatly impaired by oxygen want are not altered at all when the same oxygen-nitrogen gas mixture is inhaled with a small admixture of carbon dioxide. Our observations made on the blood pressure in man under these conditions had to be supplemented by detailed experiments with animals in which a finer analysis of the mechanisms could be made.

"We were, however, not satisfied to describe our results attained by analytical methods but tried to add to these investigations a synthetic description which allows an insight into the physiologic activities of the organism as a unity. The problems we discuss seem to us of great interest not only for the physiologist but also for the neurologist and physician. This, we hope, will become clear from our investigation and discussion of the relationship between anoxia and asphyxia. The latter term has been used in such different ways that a definition for our purposes seems indispensable. We speak of anoxia as the condition resulting from a diminished oxygen supply to the tissues. If, however, anoxia is combined with an increased carbon dioxide tension in the blood and the tissues, a state of asphyxia results. This is a frequent consequence of anoxia when it impairs the heart action, thus preventing the removal of carbon dioxide from the tissues. It seems important to distinguish sharply between anoxia and asphyxia because under the clear-cut conditions of physiological experimentation the effects of asphyxia may be fundamentally different from those of anoxia. . . .

"The effect of various gas mixtures (1 to 10 per cent. oxygen and 4 to 15 per cent. carbon dioxide) has been studied on the blood pressure of dogs anesthetized with sodium amytal, sodium barbital, or chloralosane. The results are as follows:

"By comparing the effect on the blood pressure of the inhalation of oxygen deficient and carbon dioxide containing gas mixtures on dogs with normal and artificial respiration, it is found that the blood pressure rises more in the latter condition than in the former. After carotid sinus denervation, respiration fails and the blood pressure usually falls during oxygen deficiency, but, if artificial respiration

is maintained, the blood pressure rises during a period of anoxia. These experiments indicate the necessity of maintaining a constant respiration when the reaction of the blood pressure to oxygen deficiency is being studied. The role of the carotid sinus and aortic chemoreceptors in the reaction of the blood pressure to oxygen deficiency was determined in dogs with artificial respiration. (*a*) Bilateral carotid sinus denervation, alone, did not alter the nature of the blood pressure reaction to oxygen deficiency, although the height of the rise of blood pressure was usually diminished by this procedure. Additional elimination of the abdominal fibers of the vagi by section of the esophagus at the level of the diaphragm had no effect on the blood pressure reaction. (*b*) Bilateral cervical vagotomy, alone, had no effect on the nature of the blood pressure reaction to oxygen deficiency. (*c*) Carotid sinus denervation, plus section of the nerves of Cyon, changed the reaction to oxygen deficiency from a rise to a fall of blood pressure. Additional section of the remaining vagosympathetic trunks in the neck further increased the fall of blood pressure obtained during oxygen deficiency. (*d*) The reversal of the blood pressure reaction to oxygen deficiency by total denervation of the chemoreceptor zones is not the result of deterioration of the experimental animal nor of disorganization of the vasomotor center by acute denervation. After the reversed reaction to oxygen deficiency has been obtained in animals with bilateral removal of the carotid sinus areas and unilateral vagotomy with the impulses in the contralateral vagus nerve blocked by cooling instead of cutting, the normal reaction of the blood pressure is reestablished when the nerve is again warmed. (*e*) The above experiments indicate that oxygen deficiency causes a rise of blood pressure only by action on the peripheral chemoreceptor zones. It depresses the 'isolated' vasomotor center.

"The role of the carotid sinus and aortic chemoreceptors in the reaction of the blood pressure to carbon dioxide excess was determined in dogs with artificial respiration. It was found that total denervation of the chemoreceptor zones does not alter the nature of the blood pressure reaction to carbon dioxide excess. Usually, the rise of blood pressure obtained is increased by this denervation. This is probably due to the removal of the pressor reflexes. The experiments indicate that carbon dioxide acts mainly on the vasomotor center and has only a slight action on the blood pressure through the chemoreceptors. In dogs with their carotid sinuses denervated and vagus nerves cut, asphyxia, carbon dioxide excess, and injection of lactic acid cause a rise of blood pressure, while oxygen deficiency causes a fall of blood pressure. Increasing the depth of anesthesia diminishes the blood pressure reaction to carbon dioxide and injection of lactic acid, and frequently changes the reaction from a rise to a fall of blood pressure both in the intact and the denervated dog. Ether, chloralosane, sodium amytal, and urethane were found to have this effect on the intact dog. The blood pressure reaction to oxygen deficiency is not reversed under these circumstances. The similarity of the mode of action of oxygen deficiency and carbon dioxide on the respiratory and circulatory systems and the relation between anoxia and asphyxia under various experimental conditions is discussed. That the carotid sinus pressor reflexes are impaired by the inhalation of gases with low oxygen tension is shown by the following results: (*a*) On tilting a narcotized dog into a vertical 'feet down' position the blood pressure in the carotid artery falls more during the period of anoxia than under control

conditions which precede and follow this experimental period. (*b*) On increasing the endosinusal pressure in a dog whose contralateral carotid sinus has been denervated it is found that the blood pressure falls less during anoxia than in the control period. (*c*) On tilting into the 'feet down' position the blood pressure in the carotid artery falls more in a dog after denervation of both carotid sinuses than was observed prior to this operation in the same animal during inhalation of oxygen deficient gas mixtures. This indicates that anoxia does not eliminate these reflexes completely.

"Under conditions in which carbon dioxide and oxygen deficiency cause a rise in blood pressure, i.e., in the normal dog with and without artificial respiration, and further after vagotomy *or* removal of both carotid sinus areas with artificial respiration, the effect of carbon dioxide plus low oxygen tension is greater than corresponds to the algebraic sum of the individual effects. In the dog deprived completely of its buffer nerves and artificially ventilated, it is found that carbon dioxide completely offsets the fall of blood pressure produced by the inhalation of a gas with a low oxygen tension. The experiments indicate that the cause of this potentiation lies in the fact that the effect of carbon dioxide is increased in anoxia. This is in part due to the weakening of the carotid sinus pressor reflexes. In addition to that it is assumed that the intracellular metabolites formed during short periods of anoxia may interact with the effects of carbon dioxide and thereby cause the potentiating effect described above."

J. C. M. C.

REID, L. C.: *Cellular Respiration.* Brit. J. Anesth. **17**: 41–56 (July) 1940.

"Cellular respiration may be defined as those biological processes and chemical mechanisms by which the cell converts the bound, radiant energy of the sun, stored in foodstuff molecules, to free utilizable biotic energy, thereby making possible cellular activity and even cellular existence. . . . The cell . . . has two methods for liberating the energy present in the foodstuff molecules: (1) fermentation; (2) oxidation. . . . Fermentation is the term used to cover those processes by which glucose is converted into simpler substances in the absence of oxygen with the liberation of energy. . . . Glycogenolysis . . . represents the conversion of glycogen to glucose and, strictly speaking, does not form a part of cellular respiration, but is added for the purpose of rounding out the picture with some degree of continuity and completeness. . . . It is well known that hydrogen and oxygen do not combine with a measurable velocity at ordinary temperatures but will do so under special conditions, such as the presence of inorganic catalysts, for example, palladium or platinum or enzymes, so-called organic catalysts. . . . The enzymes, dehydrogenases, were first described by Wieland and have since been extensively investigated by Thunberg, who has recently published an extensive review. These dehydrogenases are responsible for the activation of the hydrogen of the foodstuff molecules, which is transferred to a suitable acceptor or carrier substance. They are highly specific in their actions; that is, some will only transfer hydrogen to cytochrome, others to flavoprotein, and it is interesting in this connection to note that this specificity is due to the variability of the bearer portion of the molecule. Their prosthetic groups are all the same. The peculiar specificity of reaction depending on changes in the bearer portion rather than the active prosthetic group seems, at first sight, to be unusual, but the well-known substance, hemoglo-