sential neurotransmitters.[19] For that reason, severe impairment of neurological function might occur in the presence of a normal energy pool.

The quest for protection from hypoxia of the central nervous system will continue. the authors have raised a number of significant questions and are to be complimented for the meticulous development and presentation of some fascinating data.

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Analgesia, Anesthesia and Chest Wall Motion

CHANGES in chest wall (diaphragm, thoracic plus abdominal wall) shape and motion under the influence of analgetics, anesthetics, or positive airway pressure during muscular paralysis are of interest both to the practicing anesthesiologist, who can use them in staging the depth of anesthesia[1] and to the pysiologist,[2] who can use them as a tool. In this issue of ANESTHESIOLOGY appears a stimulating report[3] documenting a reduction in tidal volume and respiratory frequency during CO_2 rebreathing after morphine administration in humans, and also demonstrating reduction in the tidal volume contribution from rib cage motion at equivalent end-tidal CO_2 concentrations, comparing the sedated to the unsedated state. When the tidal volume contributions from rib cage or diaphragm were plotted as a function of the tidal volume rather than of carbon dioxide tension, no effect of morphine could be detected comparing the sedated and unsedated states. Since the effects of halothane[4] on the rib cage appear similar, the authors suggested that both morphine and halothane act by exerting a direct action on respiratory neurones in the medulla. This conclusion implies a model of the respiratory control mechanisms in which a central pattern generator in the brainstem drives the diaphragm and intercostal/accessory muscles in set patterns which vary as tidal volume changes, in response to change in chemical stimulus. Analgetic and anesthetic agents then act by decreasing the gain of the central pattern generator in response to a given change in the chemical stimulus without altering the set pattern for a given tidal volume. This attractively simple unifying hypothesis requires careful examination. Examination of the receptor sites for endogenous or exogenous opiates in the central nervous system[5,6] and observations of the actions of opiate antagonists on the H-reflex at spinal

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cord level,[7] as well as on respiratory control mechanisms in the brainstem itself,[8] suggest that many sites of action may exist. Therefore, it appears that to confine thought to one mechanism may be an oversimplification, but is nevertheless a valuable starting point for investigation.

Changes in chest wall shape and motion during anesthesia are further illustrated by a second report[9] in this issue describing changes in functional residual capacity and in the anterior-posterior diameter and circumference of the chest wall after induction of anesthesia with thiopental and during lung inflation with and without muscle paralysis.

It is important to note that both groups of investigators[3,9] measured changes in only one diameter of the chest wall. Changes of dimension in only one axis may not completely reflect changes in chest wall motion, hence caution must be exercised when extrapolating these measurements to volume contribution by rib cage or abdominal-diaphragmatic components of the chest wall. For this reason Hedenstierna *et al.*[9] are careful not to draw conclusions about the contribution of the rib cage to tidal volume during spontaneous breathing with thiopental anesthesia. If their finding[9] of an increased excursion of the rib cage during spontaneous breathing under anesthesia were to mean that there was an increased rib cage contribution to tidal volume and if tidal volume itself had been reduced at equivalent end-tidal CO_2 concentrations after thiopental, this would contradict the hypothesis of Rigg and Rondi.

Possible links between alteration in motion and shape of the chest wall and pulmonary gas exchange have not been addressed by either of these two reports in this issue. A different shape of the lung may lead to an altered distribution of inspired gas (ventilation/unit lung volume) depending on the shape change. An altered distribution of inspired gas from that seen during awake spontaneous breathing has been shown to occur during general anesthesia, both with[10] and without[11] muscle paralysis. This altered distribution of inspired gas is consistent with a different pattern of motion of the respiratory system. If the distribution of inspired gas is altered and if the distribution of perfusion does not adjust, ventilation-perfusion mismatching and impaired gas exchange may ensue.

From a review of the available evidence one of us[12] suggested that it is the change in the shape of the lungs resulting from changes in chest wall shape and motion, which is the initial factor responsible for impaired gas exchange during anesthesia, either with spontaneous breathing or mechanical ventilation.

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