CORRESPONDENCE

CARBON DIOXIDE HOMEOSTASIS DURING ANESTHESIA

To the Editor.—Doctors Elam and Brown [Carbon Dioxide Homeostasis During Anesthesia. III. Ventilation and Carbon Dioxide Elimination, ANESTHESIOLOGY 17: 116 (Jan.) 1956] have pointed out the important role of carbon dioxide production in the maintenance of carbon dioxide homeostasis and offer evidence of the difference in the effect of ether and meperidine on carbon dioxide production. However, their data do not support the following statements labeled as conclusions:

"Alveolar ventilation required to maintain carbon dioxide homeostasis may, therefore, vary between 1.0 and 8.6 LPM. During Demerol-scopolamine anesthesia normal alveolar carbon dioxide tension may be maintained with an alveolar ventilation as low as 1.0 LPM and a respiratory rate of 3.3 per minute. With plane 3 ether anesthesia normal alveolar carbon dioxide tension was not maintained with an alveolar ventilation of 4.9 LPM and a respiratory rate of 34 per minute. Carbon dioxide accumulation was consistently found during ether anesthesia at surgical planes necessary to provide abdominal relaxation."

The patient in whom it is contended that normal alveolar carbon dioxide tension was maintained with an alveolar ventilation of 1 liter per minute and a respiratory rate of 3.3 per minute had at this time an alveolar carbon dioxide tension of 56 mm. of mercury. The patient purported to be unable to maintain normal carbon dioxide tension with ether anesthesia had an alveolar CO_2 tension of 44 mm. of mercury. The data presented were obtained from 9 patients. Three received meperidine but adequate data were collected on only two. One of these showed a marked increase in alveolar carbon dioxide tension, the other a slight increase. Five patients were given ether anesthesia. Of these, one demonstrated a marked increase in alveolar carbon dioxide tension, two slight increases, one no change, and one a decrease in carbon dioxide tension. The data seem at odds with the conclusions.

There is good reason to believe that carbon dioxide production would be increased by ether anesthesia. Brewster, Isaacs, and Andersen (Am. J. Physiol. 175: 399, 1953) have demonstrated increases in oxygen consumption up to 25 per cent of preanesthesia values in the dog anesthetized with ether. This is probably secondary to the epinephrine released by ether and it is reasonable to assume that carbon dioxide production would increase concomitantly. Recent data which we have collected using morphine tends to support the observation that meperidine decreases carbon dioxide production. Six convalescent surgical patients were given 10 mg. of morphine intravenously. Their alveolar ventilation and alveolar carbon dioxide tensions were measured before and after morphine. The data (table 1) indicate a decrease in carbon dioxide production following morphine, *but also an accumulation of carbon dioxide* (increase in alveolar carbon dioxide tension) in every patient as was the case in Elam and Brown's two patients. Meperidine as well as morphine is well known to depress respiration by an action on the respiratory center (Loecheke *et al.*, J. Pharmacol. & Exper. Therap. 108: 376, 1953). I am certain that Drs. Elam and Brown did not wish to imply that meperidine-scopolamine exerts its major effect on carbon dioxide production rather than the respiratory center. Yet they minimize the effects of these two drugs on the respiratory center and the consequences of decreased ventilation following meperidine. Both meperidine and plane 3 ether anesthesia will depress the respiratory center and produce accumulation of carbon dioxide.

The data available are not sufficiently quantitative to permit accurate estimate of how much carbon dioxide production is increased or decreased by these two drugs. One wonders whether such figures as 50 cc. per minute for carbon dioxide production are meaningful. (Assuming an RQ of 0.7 in this patient the oxygen consumption for this normothermic adult would be only 70 cc. per minute.) Such low estimates could have been obtained from measurements made before a "steady state" had been achieved and

TABLE 1

Patient	CO_2 Production			Change in Alveolar CO_2 Tension mm.Hg.
	Before Morphine cc./min.	After Morphine cc./min.	Difference cc./min.	
JG	238	187	−51	+2.5
JP	239	197	−42	+5.0
HP	312	286	−26	+6.0
LC	233	165	−68	+4.0
RR	238	229	− 9	+2.5
JA	204	162	−42	+5.0

while CO_2 storage was occurring following the decreased alveolar ventilation (respiratory center effect) and the resulting decreased alveolar capillary diffusion gradient. The role of the anesthetic agent in the production of carbon dioxide accumulation during thoracic surgery remains to be evaluated.

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TOXIC REACTIONS IN EPIDURAL ANESTHESIA

To the Editor.—May I make a rather late comment on the paper of Dr. Blundell and others on Xylocaine and Cyclaine for epidural analgesia (ANESTHESIOLOGY, May 1955)? Convulsive reactions in over 1 per cent of patients caused them to abandon Xylocaine despite its otherwise good characteristics for this technique.

They found that adding epinephrine, 1 part in 100,000, to the analgesic solution had no sustaining effect on the blood pressure, and though it is not actually stated it appears that they did not normally use epinephrine. The absence of influence on the blood pressure agrees with our experience, and we take it as evidence that the epinephrine in this dilution has a local vasoconstrictor effect which hinders its own absorption into the circulation and that of Xylocaine with which it is mixed. This is confirmed by our experience of toxic effects. When using Xylocaine, 2 per cent, without epinephrine, we repeatedly found it to cause drowsiness and even unconsciousness or mental agitation though never convulsions. In one case epidural *procaine*, 2 per cent, without epinephrine did cause convulsive twitching. Since adding epinephrine, 1 part in 100,000, we have never seen these signs of generalization, though initial doses of 2 per cent Xylocaine have been up to 40 ml., and continuous analgesia has been maintained up to 24 hours.

In the three cases detailed by Dr. Blundell, convulsions occurred two minutes, one minute, and almost immediately after the injection of the main dose of Xylocaine. The severity and speed of these reactions suggests another possibility other than absorption from the tissues—accidental cannulation of an epidural vein. This would seem an unlikely event, but it occurred here recently. Immediately after insertion of a polyvinyl catheter, venous blood flowed down the catheter and dripped from the end. The rate of flow was increased by aspiration with a syringe. On withdrawing the catheter a little the flow ceased. Since this occurrence we take precaution of aspirating before injecting anything through the catheter.

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