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Lidocaine Effects on Leukocytes and Erythrocytes

To the Editor:—It is known that lidocaine stabilizes the cell membrane and protects erythrocytes suspended in saline solution from hemolysis.[1] Our study was performed to investigate whether this property of lidocaine is of practical importance in improving the preservative quality of citrate–phosphate–dextrose (CPD) solution.

From each of five donors, a 450-ml volume of blood was collected into a standard plastic bag containing CPD, 63 ml. From every bag, nine aliquots of 50 ml each were withdrawn aseptically into small transfusion bags. One milliliter of CPD solution containing lidocaine was added to each of eight bags, and 1 ml without lidocaine was added to the control. The lidocaine concentrations in the study groups were 25, 50, 75, 100, 200, 300, 400 and 500 mg/l. On days 0, 7, 14, and 21, a 10-ml volume of blood mixture was aseptically withdrawn from each bag for the determination of plasma potassium, plasma and cell hemoglobin, hematocrit, and leukocyte count.

We found that all blood constituents except hematocrit and cell hemoglobin showed significant changes with time ($P < 0.05$). The slopes of the responses with time were similar at different dose levels. The addition of lidocaine did not cause a significant change in any of the constituents. The only statistically significant ($P < 0.05$) exception was a clinically unimportant decrease in plasma potassium at lidocaine concentrations of 300 to 500 mg/l (table 1). It was interesting to find that lidocaine did not increase lysis of leukocytes even at higher concentrations. We conclude that lidocaine does not improve the preservative quality of CPD solution; thus, its use is not recommended.

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TABLE 1. Effects of Lidocaine on Stored Blood

	Plasma Potassium (mEq/l)		Plasma Hemoglobin (mg/100 ml)		Erythrocytic Hemoglobin (g/100 ml)		Hematocrit (Per Cent)		Leukocyte Count (Cells/mm³ Blood)	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Control	14.675		83.400		12.55		37.775		4,995	
Lidocaine (mg/l)										
25	13.985		84.050		12.365		37.475		5,055	
100	13.945	0.244	77.400	5.77	12.380	0.079	37.405	0.272	5,000	0.093
300	13.439*		85.850		12.380		37.275		4,910	
500	13.490*		84.850		12.425		37.550		4,830	

* Significant at 0.05 level.

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Artifactual Metabolites of Halothane?

To the Editor:—Mukai *et al.*[1] ascribe the appearance of two volatile compounds, CF_2:CHCl and CF_3CH_2Cl, in the exhaled air of rabbits anesthetized with halothane to metabolism of halothane. Perhaps so, but the authors, according to their description of the methods they used, failed to take the precaution of running samples of halothane through the analytic process to assure that: a) the compounds in question were not artifacts generated by breakdown of halothane by the gas chromatographic techniques used; b)

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these two compounds were not originally present in the halothane as contaminants.

The possibility that the putative metabolites might be artifactual in origin is heightened by the fact that they appeared so rapidly, almost instantaneously, in the exhaled air. Metabolites of an inhaled anesthetic would be most rapidly detected in exhaled air if mixed-function oxidase systems in the lungs were responsible for biotransformation of the anesthetic. The rate at which other xenobiotics are known to be taken up by pulmonary microsomes and the rate at which they are known to be subsequently metabolized[2] are such that one would not expect the metabolites of halothane to appear almost immediately in end-tidal air. There should be a lag period. If the metabolites were formed by hepatic mixed-function oxidase systems the delay in their appearance in exhaled air would be even greater.

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In reply:—The present investigation was performed using a non-rebreathing anesthetic circuit. We chose this circuit because halothane vapor when repeatedly passed through soda lime can be converted partly into two substances: CF_2:CBrCl, which was reported originally by Raventos *et al.*[1] and CF_3CH_2Cl, whose concentration course in a closed anesthetic circuit with a dummy lung was reported by Morio *et al.** In the control gas chromatogram (fig. 1) of the gas sample from our nonrebreathing anesthetic circuit with a dummy lung, no obvious volatile material could be detected between the air and halothane peaks. This indicates that no artifact was generated by the breakdown of halothane in the chamber or during the gas chromatographic procedure. Furthermore, the halothane used in this study was demonstrated by gas chromatography to be pure. These two compounds were not originally present in the halothane used.

As to the microsomes in each organ, it is well known that there are large differences in drug-metabolizing abilities. In studying this problem, species difference should be taken into consideration. We have found in a subsequent study (unpublished observations) that CF_2:CHCl and CF_3CH_2Cl appear immediately after administration of halothane to a liver homogenate. A small amount of these metabolites appears when halothane is added to a kidney homogenate, but only a trace amount is found when halothane is added to lung or brain homogenate, and none in the case of whole-blood homogenate. These findings have led us to conclude that there is little delay in their appearance in the exhaled gas.

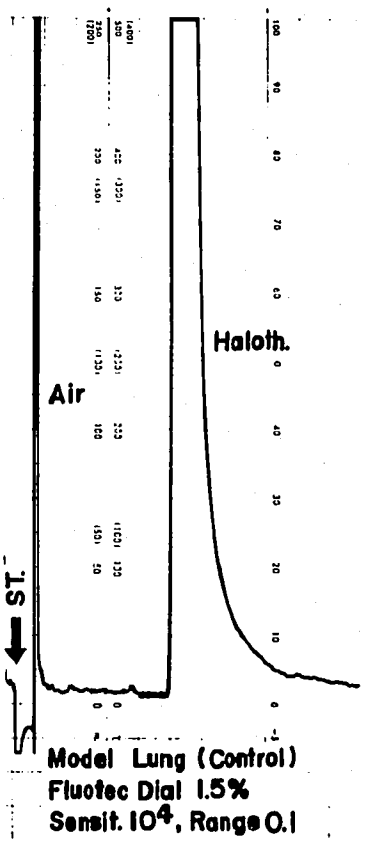

FIG. 1. Control gas chromatogram of the gas sample from the nonrebreathing anesthetic circuit. No obvious volatile material could be detected between the air and halothane peaks.

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