## The Effects of Ethrane

To the Editor:-In a recent article,1 Drs. Shimosato, Sugai, Iwatsuki and Etsten, using a papillary muscle preparation, demonstrated that a greater concentration of Ethrane than of either halothane or methoxyflurane is required to depress several parameters of myocardial contractility (table 1). They state that "therefore, Ethrane was less depressant to myocardial contractility than methoxyflurane or halothane,"

Although, on a milligram-for-milligram basis, the above statement is accurate, it is, in a way, quite misleading, in that it ignores the relative narcotic effects of the three agents. If, for example, drug A and drug B depress the heart equally at equal concentrations and this same concentration represents a far greater anesthetic depth for drug A than for drug B,

Table 1. Concentrations of Ethrane, Methoxyflurane and Halothane to Produce 50 Per Cent Reductions of Parameters of Myocardial Mechanics

	Concentration (mg/100 ml)			
	Ethrane	Methoxyflurane	Halothane	
V <sub>max</sub>	20	17	6	
F <sub>m</sub>	16	12	7	
Maximal power	11	4	3	
Maximal work	9	6	3	
Maximal dF/dt	13	9	5	

Table 2. Multiples of MAC for Ethrane, Methoxyflurane and Halothane Required to Produce 50 Per Cent Reductions of of Parameters of Myocardial Mechanics

	Ethrane*	Methoxy- flurane**	Halothane***	
Vmx	2.2	3.4	1.3	
F <sub>m</sub>	1.S	2.4	1.5	
Maximal power	1.2	0.81	0.64	
Maximal work	1.0	1.2	0.64	
Maximal dF/dt	1.4	1.\$	1.07	

<sup>\*</sup> Ethrane MAC estimated to be 1.45 per cent from MAC x oil/gas partition coefficient = 143.2

then in terms of cardiac depression per levelo of anesthetic depression, drug B is more de pressing to the heart than drug A.

I have restated the data presented by the authors in terms of multiples of Minimum Alveolar Concentration (MAC),2 a standard of potency that can be used to compare relative cardiovascular or respiratory effects of anesthetic agents.3 The data now show that equals reductions of myocardial mechanics are pro-o duced by lighter levels of Ethrane than me-S thoxyflurane for all parameters measured except maximum power (table 2). If this relationship holds true in vivo, one could they say that Ethrane was more rather than less depressing to the heart than methoxyflurane.

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ering the relative narcotic effects of anesthetic agents, as pointed out by Dr. Saidman's letter, is well taken. However, it should be pointedo out that it is more appropriate to determine the percentage depression in myocardial mechanics at an equipotent level of anesthesia. A comparison of the degree of depression produced by three anesthetics: halothane, methoxyflurane and Ethrane, at a given MAC is ≥ more discerning than attempting to determine changes in multiples of MAC related to

<sup>\*\*</sup> MAC for methoxyflurane = 0.16 per cent.2 \*\*\* MAC for halothane = 0.77 per cent.2

Note, we have taken into account the relative narcotic effect by means of the MAC-1 level, and the concentrations of the anesthetics at a MAC-1 level for animals as described by Eger ct al. (Anesthesiology 30: 129, 1969). The percentage decreases in the components of myocardial mechanics of the isolated cat papillary muscle induced by three agents are shown in table 1.

The percentage decreases in Vmax, Fm, maximal power, maximal work and maximal dF/dt were calculated for a given MAC-1 level of each agent, expressed in mg/100 ml using

Table 1. Med			C-1 (%		1
	Vmax	F.	Max, Power	Max. Work	Max.d
Ethrane	12	36	44	50	37
Methoxyflurane	31	40	55	51	43 = 45 €
Halothane	39	35	51	52	45 €

component. Our data show that percentage decrements in all components of mechanics of contraction of the heart muscle exposed to Ethrane at the MAC-1 level are less than those in muscles exposed to either halothane of methoxyflurane. Evidently, these findings cor roborate our previous findings which showed that Ethrane is less depressant to myocardia contractility than methoxyflurane or halothane (Anesthesiology 30: 513, 1969).

## Drugs

en to 100 patients induction of anesthesia was reapnea occurred in ained with nitrous respiratory rate as when the respiratory rate as of Phenoperidine, NEW ANALGESIC Phenoperidine, a new analgesic, was given to 100 patients of various ages in a dose of one mg/30 kg body weight prior to induction of anesthesia. Sleep was not induced with this drug, but the patients became drowsy within five minutes. The dose of thiopental required for induction of anesthesia was reduced considerably. Respiratory depression was considerable; apnea occurred in 36 patients, most of whom were elderly. Anesthesia was maintained with nitrous oxide-oxygen and fractional doses of phenoperidine, using the respiratory rate as an indicator of the need for more drug (about every 30 minutes when the respiratory rate was faster than 10 beats/min). Tracheobronchial reflexes were also depressed, and the endotracheal tube was well tolerated. The cardiovascular system remained stable. (Viars, P., and Gaveau, T.: Report on the Use of Phenoperidine, Anesth. Analg. 25: 163 (March) 1968.)

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