tion (MAC) for each agent are: halothane 0.74 per cent; fluroxene 3.4 per cent; and cyclopropane 9.0 per cent (Saidman, L. J., and others: Anesthesiology, in press). Constant alveolar concentrations were maintained for each agent at multiples of MAC ranging from 1.1 to 2.5 (1.1 MAC equals 1.1 times MAC). End-expired halothane and fluroxene concentrations were monitored with a Beckman infrared analyzer. Alveolar cyclopropane concentration was estimated from inspired cyclopropane concentration determined by oxygen difference. Alveolar anesthetic and CO₂ partial pressures were maintained constant for an interval calculated to allow for cerebral equilibration. Ventilation was measured with a recording ventilimeter. The slope of CO₂ response (liters/minute/mm. PaCO₂) during anesthesia was expressed as a fraction of the awake control slope. No surgical stimulation was present during this study.

Results: Mean ventilatory slope values (liters/minute/mm. PaCO₂) awake were: halothane 1.08 ± 0.42; fluroxene 1.32 ± 0.64; and cyclopropane 1.59 ± 5.3. With a few exceptions (cyclopropane) all subjects showed a progressive reduction in CO₂ response with increasing depth of anesthesia. Mean fraction of awake slope values for halothane were 0.48 ± 0.22 (P < 0.05) at 1.1 MAC; 0.17 ± 0.17 at 2.0 MAC, and 0.06 ± 0.09 at 2.5 MAC. Values for fluroxene were 0.72 ± 0.28 (P > 0.05) at 1.1 MAC; 0.45 ± 0.18 (P < 0.05) at 2.0 MAC, and 0.03 ± 0.02 at 2.5 MAC. Values for cyclopropane were 0.92 ± 0.21 (P > 0.05) at 1.3 MAC; 0.67 ± 0.24 (P < 0.05) at 1.9 MAC; and 0.54 ± 0.09 at 2.4 MAC. Differences between halothane and cyclopropane were significant at each level of MAC. Similarly a difference was demonstrated between halothane and fluroxene at 2.0 MAC and between fluroxene and cyclopropane at 2.5 MAC. Mean PaCO₂ values during awake determinations were: halothane 35.5 ± 2.9 mm. of mercury; fluroxene 34.4 ± 3.7 mm. of mercury; and cyclopropane 37.7 ± 2.2 mm. of mercury. PaCO₂ values during spontaneous ventilation increased progressively with increasing levels of anesthesia in almost every instance. Mean PaCO₂ values for halothane were 48.2 ± 6.5 mm. of mercury (P < 0.05) at 1.1 MAC; 63.5 ± 11.2 mm. of mercury at 2.0 MAC, and 74.3 ± 11.6 mm. of mercury at 2.5 MAC. Values for fluroxene were 40.9 ± 3.7 mm. of mercury (P < 0.05) at 1.1 MAC; 41.9 ± 5.6 mm. of mercury at 2.0 MAC, and 54.2 ± 8.8 mm. of mercury at 2.5 MAC. Values for cyclopropane were 45.0 ± 4.3 mm. of mercury (P < 0.05) at 1.3 MAC; 52.1 ± 12.9 mm. of mercury at 1.9 MAC, and 52.6 ± 13.1 mm. of mercury at 2.4 MAC. Differences between halothane and fluroxene were significant (P < 0.05) at each level of MAC. Similarly a difference was demonstrated between halothane and cyclopropane at 2.5 MAC.

Conclusion: At equipotent anesthetic concentrations halothane and fluroxene are more potent ventilatory depressants than cyclopropane.

Effects of Anesthetics on Cardiovascular Responses to Hypothalamic and Mesencephalic Stimulation in Dogs. S. H. Ngai, M.D., and Per Bolme, M.D., Department of Anesthesiology, College of Physicians and Surgeons, Columbia University, New York City, and Department of Pharmacology, Karolinska Institute, Stockholm, Sweden. Available evidence indicates that anesthetic action on neural regulatory mechanisms plays an important role in producing circulatory changes. While there is general agreement that halothane and barbiturates depress the vasomotor centers, opinions differ as to the mechanism of action of cyclopropane (Price, and others; Anesthesiology 24: 1, 1965; Markee and Wang: Fed. Proc. 22: 187, 1963; and Bartelstone: Fed. Proc. 23: 179, 1964). The use of basal narcosis or decerebration in acute experiments has often led to difficulties in the interpretation of observations. Method: In the present study, dogs with chronically implanted aortic catheters, electromagnetic flowmeter probes around the external iliac artery and stimulating electrodes in the hypothalamus and mesencephalic tegmentum, were used. The electrodes were placed stereotaxically to give a pressor response or to activate the sympathetic cholinergic vasodilating mechanisms to skeletal muscles upon stimulation. In some animals bilateral carotid loops were also prepared. After recovery from these operations, arterial pressure, external iliac arterial blood flow and their responses to central stimulation or caro-
tid occlusion were measured before and during anesthesia with cyclopropane, halothane, Nar
total (isopropyl-β-bromallyl-N-methylmalonyl-
carbamide sodium), 25-27 mg./kg., or after intramuscular administration of morphine sul-
fate, 1-1.5 mg./kg. Results: "Light" cyclo-
propane anesthesia caused no change or a slight increase in the arterial pressure, reduced
the blood flow and increased the calculated
vascular resistance in the hind limb. The
pressor response to central stimulation was
reduced and sometimes changed to a depressor
one. The cholinergic vasodilating response
persisted. Response to carotid occlusion did
not change or decreased slightly. "Deep" cy-
clopropane anesthesia caused variable changes
in the arterial pressure, markedly reduced the
blood flow and increased the vascular resist-
ance. All responses to central stimulation and
carotid occlusion were abolished. Halothane
anesthesia, depending on the "depth," reduced
or abolished the pressor response to central
stimulation and carotid occlusion. The cho-
linergic vasodilating response was not altered
significantly. Narkotal, in the dose employed,
produced "light" narcotics. It reduced the
arterial pressure, blood flow and the pressor
response to central stimulation and carotid
occlusion. The cholinergic vasodilating re-
sponse was not altered significantly. Mor-
phine produced sedation. It did not signifi-
cantly change the arterial pressure but reduced
the blood flow and increased the vascular
resistance. Pressor and cholinergic vasodilat-
ing responses to central stimulation were not
altered. Conclusions: These observations sug-
gest that anesthesia generally suppresses the
excitatory vasomotor mechanisms, confirming
the concept of depressant action of halothane
and barbiturates, but denying the thesis that
the circulatory effect of cyclopropane is due
to its selective sparing action on these mecha-
nisms. These results also indicate that the
sympathetic cholinergic vasodilating mechani-
sm in the hypothalamus can be activated
during sedation and "light" anesthesia. (Sup-
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Effect of Postural Variations on Anatomic
Dead Space Before and After Atropine.
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thesiology, Columbia-Presbyterian Medical
Center, New York City. The observation
(Tomlin, P. J., Conway, C. M., and Payne,
J. P.: Lancet 1: 14, 1964) that patients de-
veloped hypoxemia after atropine and before
operation suggested that atropine acted by
markedly increasing the dead space. In ad-
dition, interpretation of the data accumu-
lated by many investigators depends upon
whether the dead space is fixed or variable.
Among the factors which influence dead space
are the effects of posture and belladonna
drugs. While the effects of posture and of
atropine have been measured independently,
itis seemed reasonable to examine the combined
effects of posture plus atropine. Methods
and Results: Eight healthy male subjects were
studied in the supine, sitting, right and left
lateral positions before and 20 minutes after
the intramuscular injection of 0.5 mg. atro-
pine. Pilot studies revealed that 0.8 mg.
atropine produced a greater dead space change
than 0.5 mg. atropine and the same change as
1.2 mg. atropine. Anatomic dead space was
measured using the Fowler single-breath tech-
nique with nitrogen as the indicator gas. The
method is based upon the simultaneous meas-
urement of expired gas volume and expired
N₂ content following the change from breath-
ing air to inhaling 100 per cent oxygen. There
was no statistically significant difference in
the tidal volume measured in the various
positions before or after atropine administra-
tion. There was no significant difference
among the dead spaces in the various positions
before atropine administration. However,
atropine administration caused a significant
increase in anatomic dead space of about 25
per cent greater than the control. We were
curious to determine if the observed 25 per
cent increase in anatomic dead space would
cause a clinically significant fall in arterial
oxygen saturation. Twenty patients in good
physical condition were studied. Control arte-
rual blood samples were obtained from semi-
recumbent patients, by percutaneous arterial
puncture from the brachial artery into 10 ml.
heparinized syringes. The blood was allowed
to fill the syringe under its own pressure for
one to two minutes, and during this period
the patient was encouraged to relax and
breathe naturally. This procedure was re-