as well. This kind of dual protection can come only from hypothermia. In addition, hypothermia can limit the amount of general anesthetic required. Decrease in dose of anesthetic drugs may become a vital issue, as the increase in intracranial pressure reported during general anesthesia appears to be far more pronounced with anesthesia at depth. The increase in intracranial pressure probably will prove to be minimal or nonexistent during very light anesthesia.

These considerations lead to the conclusion that in our present state of knowledge the anesthetic technique of choice for carotid vascular surgery should include hypothermia and a light plane of anesthesia.

While we work toward clinical application of rCBF measurements, the interim solution is—cool it!

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Dimensions of Anesthetic Uptake

Our current theories of anesthetic uptake and distribution had simple beginnings. In his classic paper, Kety made several assumptions which permitted the first reasonable simulation of anesthetic uptake. One of these assumptions was that all tissues receive equal perfusion per unit volume. Kety was aware that this was incorrect, but to assume otherwise presented an almost overwhelming mathematical obstacle. Later, Price, then McKrell, Mapleson, Severinghaus, and Eger, discarded this assumption and proposed new models which assumed differential perfusion of tissues (i.e., the visceral tissues receive a high flow, muscle less, fat still less). These analyses were made possible by the development of analog and digital computers. One by one the remaining simplifying assumptions made by Kety also have been abandoned in favor of more complex but more realistic assumptions. For example, the impact of ventilation/perfusion abnormalities on uptake has been analyzed. Similarly, the effect of varying inspired concentrations of anesthetic (the "concentration effect") has been accounted for. We have calculated the effects of proportionate and differential changes in blood flow to tissues. Even anesthetic metabolism has its counterpart in simulation.

Several surviving assumptions are patently false, yet have remained unchallenged. Until the work by Ashman, Blesser and Epstein (A Nonlinear Model for the Uptake and Distribution of Halothane in Man) which appears in this issue of Anesthesiology, we had assumed that induction of anesthesia did not affect the initial physiologic conditions given the computer. Thus, ventilation, circulation, metabolism, etc., were presumed to remain constant once the initial conditions had been set: if we began with a cardiac output of 5 l/min, then we finished with a cardiac output of 5 l/min. As Ashman et al. note, this conflicts with reality. For example, halothane decreases cardiac output, and this decrease (at least initially) should decrease halothane uptake and thereby allow achievement of a higher alveolar concentration. Ashman and his co-workers addressed themselves to the problem of calculating the effect of a progressive decrease in output with increasing partial pressure of halothane. Their solution suggests that an hour of inspired halothane at 1.2 per cent produces an alveolar halothane concentration 6 per cent above that produced when cardiac output is constant. This is the first computation of the effect of an anesthetic on its own uptake through its influence on a physiologic variable. It therefore represents a significant advance over our previous assumptions.

Ashman's paper points up the value of electrical and mathematical models that simulate
uptake and distribution. Often such models have taken the place of experiments in vivo and, in fact, experiments in vivo are used less frequently than models in the initial investigations of new hypotheses. The slight degree of the change predicted by Ashman would have precluded its quantitative assessment in vivo because of concomitant changes in other variables. For example, cardiac output is not the only factor influenced by halothane. Metabolism decreases and must cause a reduction in alveolar ventilation if $P_{\text{CO}_2}$ is held at a normal level. At light planes of anesthesia this reduction in ventilation would oppose and probably completely reverse the effect of the decrease in cardiac output upon the alveolar halothane concentration. Ashman points out another difficulty in the interpretation of an in-vivo approach. Anesthetic levels of halothane affect not only cardiac output but also distribution of blood flow. Distributional changes probably would decrease uptake just as would a reduction in output. Finally, we also know that increased duration of anesthetic antagonizes the depression of cardiac output produced by halothane, thus adding a nonlinear fourth dimension to the reality Ashman wished to simulate. 

Of what importance are such changes? Why study a factor which, at most, causes a 6 per cent deviation from the norm? As noted above, part of the answer is that Ashman’s work represents the first challenge to an unrealistic assumption. Another part of the answer is that the 6 per cent figure is a minimum rather than a maximum estimate of what is likely to occur clinically. Ashman chose a 1 per cent inspired concentration because it produces very light of anesthesia. A higher concentration would cause greater depression of cardiac output and uptake. Similarly, the data relating halothane concentration and cardiac output were gathered in young, healthy unmedicated volunteers. Elderly or sick patients or patients who have received other depressant drugs might respond to a given concentration with a far larger reduction in cardiac output. Additionally, an increase in anesthetic solubility would cause a greater change in the impact of the reduced cardiac output on uptake: methoxyflurane thus would affect its own uptake more than would halothane. The ultimate rationale for focusing on such “6 per cent” changes is that the sum of such changes describes the total picture of anesthetic uptake. 

**References**