from the supplementary oxygen flowmeter should always be verified when a machine is placed into service.

Our department developed a policy that states that operating room circulating nurses who apply supplemental oxygen must obtain their supply from separate flowmeters connected directly to wall-mounted oxygen fittings. Only anesthesiologists and certified registered nurse anesthetists may use the anesthesia machine for an oxygen source because of the potential problems described in this letter.

Harvey J. Woehlck, M.D.
Assistant Professor of Anesthesiology
Department of Anesthesiology
Medical College of Wisconsin
9200 West Wisconsin Avenue
Milwaukee, Wisconsin 53226

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Hypotension and Spinal Anesthesia

To the Editor:—The recent study by Carpenter *et al.*[1] imposes a few specifications concerning the definition and prevention of hypotension during spinal anesthesia.

First, as pointed out by the authors, the definition of hypotension during spinal anesthesia is controversial.[1] Nevertheless it seems to be unsafe to define hypotension as a systolic blood pressure of less than 90 mmHg. Rather than an arbitrary value, hypotension usually is defined as a decrease of systolic blood pressure of more than 30% from the baseline.[2]

Second, the authors have found many risk factors for hypotension during spinal anesthesia, but they omitted discussing the role of fluid loading in preventing hypotension. Venn *et al.*[3] found a tendency toward a more stable systolic blood pressure after fluid loading in patients in whom the block extended to T5 or above, whereas others found it less effective.[4] It seems that different volumes of fluid loading are the reason for these contradictory results.

P. Szmuk, M.D.
T. Ezri, M.D.
V. Priscu, M.D.

Department of Anesthesia
Kaplan Hospital
76100, Rehovot, Israel

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In Reply:—Szmuk *et al.* state that our definition of hypotension *seems* unsafe and then suggest that it would have been more appropriate to define hypotension as a 30% decrease from baseline. Szmuk *et al.* may be correct. However, it is not possible to define the lowest acceptable blood pressure for any individual patient using routine anesthetic monitoring techniques. Furthermore, the absence of data on this topic precludes a definitive conclusion and leaves our study open to criticism of what *seems* to be appropriate.

In defense of our definition, we do not agree that relative definitions of hypotension would have reduced the risk or improved our ability to interpret the data. For example, a recent epidemiologic publication utilized ≥20% decline in blood pressure from baseline (measured immediately prior to induction of anesthesia) as a relative definition of hypotension.[1] Although this is a concise definition, the clinical relevance of a 20% (or even a 30%) decline is questionable for at least two reasons.

First, blood pressure normally decreases by an average of 20% with sleep each night,[2] yet the effect of sleep on blood pressure is not considered to place individuals at risk for morbidity or mortality. Second, selection of an accurate and representative baseline blood pressure is difficult (a critical factor that is often ignored). Blood pressure measurements made immediately prior to induction of anesthesia can be considerably higher or lower than those measured in the clinic or hospital and thus may not accurately represent the

baseline blood pressure. This variability in preanesthetic blood pressures results in part from the variability in premedication practices, in part from the variability in patient anxiety levels, and in part from normal variations in blood pressure (*e.g.*, systolic blood pressure noted to range from 75 to 200 mmHg during waking hours in normotensive individuals).[2] Thus, the ability to predict those patients in whom a particular blood pressure decrease is potentially detrimental is not as clear as it first seems.

Although the cerebral autoregulatory curve is known to shift in chronic hypertensives, the magnitude of the shift and the relevance of previous studies to our patient population are unclear. Most of the previous studies were performed in patients with severe arterial hypertension and mean pressures ≥ 150 mmHg.[3] However, none of our patients had mean arterial pressures greater than 150 mmHg, and only 18 of our patients had diastolic blood pressure greater than 100 mmHg, at baseline. Thus, the significance and relevance of this factor in our patient population is unknown.

We conclude that there is no definition of hypotension that will satisfy all audiences. Relative definitions of hypotension have the advantage that they have been utilized in many previous studies. However, we believed that the limitations, described above, outweighed the benefits. After much deliberation, we chose an absolute definition based on systolic blood pressure because we thought this definition had the most clinical relevance for this study.

The authors also question the role of fluid loading in preventing hypotension. Although we did not prospectively assign patients to receive different volumes of intravenous fluids prior to initiating spinal anesthesia, we did record the amount of fluid preload administered and attempted to correlate these volumes with the incidence of hypotension. None of these correlations proved to be statistically significant. Consequently, these findings were reported only in the results section of the paper and not expanded upon in the discussion.[4]

Randall L. Carpenter, M.D.
Robert A. Caplan, M.D.
Department of Anesthesiology B2-AN
Virginia Mason Medical Center
P.O. Box 900
Seattle, Washington 98111

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Nitrous Oxide—It's Enough to Make You Vomit

To the Editor:—After tabulating the results of eight studies that examined the effect of N_2O on vomiting,*[1–7] Watcha and White recently concluded: "The evidence at present suggests that nitrous oxide does not significantly effect [sic] the incidence of postoperative emesis in adults when halogenated inhalation agents are used (table 3)."[8] However, examination of Watcha and White's table shows that half of the studies listed found a statistically significant association between N_2O and emesis,*[1,6,7] and all of the "negative" studies found a higher incidence of vomiting in their N_2O groups, though not by a statistically significant amount.[2–5]

One of the "negative" studies found that twice as many N_2O patients vomited, but because the sample size was small, the P value was .054, so the association was discounted.[2] Another of the "negative" studies lumped vomiting, retching, and nausea together, but when vomiting alone is considered, the result is significant at $P < .006$ (chi square).[3] The one nongynecologic "negative" study excluded patients at high risk for emesis from preexisting conditions and excluded surgical procedures associated with emesis, but included patients who had experienced "nausea and/or vomiting with previous general anesthesia."[5] These exclusion criteria limit the clinical generalizability of the study's findings but do not challenge their validity. Inclusion of individuals with a history of vomiting subsequent to surgery also would be valid if both the N_2O and non-N_2O groups had contained approximately equal numbers of such individuals, but that was not the case. Muir and coauthors' non-N_2O group was assigned significantly more patients known to vomit subsequent to surgery ($P < .03$, chi square), biasing their result from finding a high incidence of vomiting associated with N_2O.[5] Nevertheless, when vomiting is distinguished from nausea, Muir and coauthors found the association, but it was not statistically significant.

Instead of noting an apparent trend, Watcha and White discounted the four statistically significant findings because "none . . . was controlled for the day of the menstrual cycle"—which might be a

* Alexander GD, Skupski JN, Brown EM: The role of nitrous oxide in postoperative nausea and vomiting (abstr). Anesth Analg 63:175, 1984