Special Article

Critique:

Occupational Disease among Operating Room Personnel

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The national study of occupational disease among operating room personnel was undertaken to determine whether there is validity to previous reports suggesting a hazard from exposure to trace anesthetic vapors.¹ According to the authors, the purpose of the national study was to define the problem as precisely as possible. The report required a two-year effort by an Ad Hoc Committee appointed by the American Society of Anesthesiologists. Questionnaires were sent to 73,496 individuals, and replies were analyzed.

The report states that while there was not, “unequivocal evidence concerning the several occupational diseases studied, it seems likely that a number of serious problems do exist in the operating room.” It further concludes, “Based on all the information gathered . . . an increase in disease rates in operating room personnel is present and that exposure to waste anesthetic gases in the operating room provides the most reasonable explanation.” The accompanying editorial declares that effective exhaust systems must be consistently used in all areas in which inhalation anesthetics are administered.² It suggests that failure to use these systems could raise major legal issues.

Because of the serious implications of the report in concluding that there is an occupational hazard if, in fact none exists, we believe the data should be critically reviewed. There are three areas of concern to us. First, we question the method of collecting data, which in the case of one disease results in bias and in the cases of other diseases results in information insufficient upon which to base valid conclusions. Second, we have reservations about the authors’ selection of 5 per cent as a level of statistical significance when comparing exposed subgroups to non-exposed control subgroups. When a set of 30 tests for comparison is performed (six tests, five diseases), the possibility of obtaining a significant finding in at least one test at the 5 per cent level, strictly by chance, is roughly 79 per cent.§ If a 1 per cent level is taken as significant for each separate test, then the overall false-positive rate is approximately 26 per cent. Considering the potential impact of the false-positive conclusion in the areas of legal liability and recruitment into the specialty, even a 1 per cent error may be too risky. Finally, we are disturbed by the logic used by the Ad Hoc Committee in deriving their conclusions. The data on spontaneous abortion, for example, would lead us to conclude that any increase in spontaneous abortion rate found in operating-room personnel is less likely to be due to inhalation of trace anesthetic vapors than to result from other environmental factors.

Spontaneous Abortion

1) If trace amounts of anesthetic vapor caused spontaneous abortion, then, to quote

§ This is only an approximation, since non-operating-room nurses were compared with both nurse anesthetists and operating-room nurses.
the text of the report, "it might be anticipated that the effect might also be evidenced in early abortion among operating room women and a decrease in sex ratio (male/female) of live-born babies. . . . " No statistically significant difference between exposed and unexposed women was found in those areas.

2) If exposure to anesthetic vapor caused an increased abortion rate, one would expect the rate would be highest among those women with greatest exposure. The text of the report states, "Levels of anesthetic exposure range from relatively high exposure among physician anesthesiologists (ASA) and nurse anesthetists (AANA) to substantially lower exposure among operating room nurses and technicians (AORN/T)." The rate of spontaneous abortion among the AORN/T, however, was greater than that of either the AANA or the ASA, and these differences were statistically significant. These comparisons indicate that the tendency toward spontaneous abortion is not dose-related to anesthetic vapor. The authors may object to our comparing nurse AORN/T to physician ASA; however, our comparison between nurse AORN/T and nurse AANA is no less appropriate than their comparison between physician ASA and physician pediatricians.

3) If the cause of the higher spontaneous abortion rate were exposure to anesthetic vapors, one would expect that leaving the operating room environment would lower the rate of spontaneous abortion. After having left the operating room one year or more prior to pregnancy neither ASA nor AANA had a significant reduction in spontaneous abortion rate. On the other hand, AORN/T, who were not exposed to the operating room environment during pregnancy, had a rate of abortion significantly lower than that of exposed AORN/T and equal to that of their control group. This leads us to conclude that if, indeed, there is any occupational hazard in the operating room contributing to an increased rate of spontaneous abortion the hazard may be something other than trace anesthetic vapors.

**Congenital Anomalies**

Using 5 per cent as a level of significance, neither the children of women in the ASA nor those of women in the AORN/T had a significantly higher risk of congenital anomalies compared with control groups. Further, when mothers from either of these two subgroups were absent from the operating room for a year or more prior to pregnancy the rate of congenital anomalies was not significantly reduced. Had the conservative 1 per cent level been used, the authors could not have claimed a significant difference between the congenital abnormality rates of children of nurse anesthetists and those of their control group or any subgroup that compared children of exposed men with their controls.

**Cancer**

There was no significant difference in cancer rates among men exposed to trace anesthetics vapors compared with control groups of unexposed men. Using a 1 per cent level, there was no significant difference between the cancer rates of female ASA or AORN/T and their control groups.

Corbett, a coauthor of the Ad Hoc Committee Report, previously published a paper on the incidence of cancer among Michigan nurse anesthetists. While the Ad Hoc Committee reported the overall cancer rate among nurse anesthetists to be 2.3 per cent, Corbett found the incidence among Michigan nurse anesthetists to be greater than 5 per cent. If the Michigan nurses were surveyed twice they are subject to sampling bias not found in nurses from other states. If the data from the Corbett study were included directly into the Ad Hoc Committee study, it was done in error, since the data-gathering techniques in the two studies differed. We believe it would be appropriate to recompute the cancer rate among nurse anesthetists omitting the Michigan data to see whether a significant increase would still be found.

The finding of a higher incidence of leukemia and lymphoma should be viewed in light of earlier reports. A retrospective study published by Bruce et al. showed a higher incidence of death from cancer of lymphoid tissue among anesthesiologists compared with the general population. When this was repeated six years later as a prospective study, no difference was found. The former study was cited by the Ad Hoc Committee; however,
Hepatic Disease

The report showed no significant difference in the rate of hepatic disease between male or female AORN/T and their control groups. Male nurse anesthetists had no significant increase in hepatic disease and, if a 1 per cent level of significance is used, neither did female members of the ASA group.

The authors state that O.R. personnel who administer blood run a high risk of serum hepatitis. For this reason the diagnosis of serum hepatitis was disregarded in calculating the hepatic disease rate. The survey form upon which the data were based had a space for diagnosis to be filled in by the respondent. We do not believe the information written in this space is reliable to separate the varieties of hepatic disease. A large number of cases reported merely as hepatitis may have been serum hepatitis. This would lead to an erroneous conclusion regarding the subgroups in which significant differences were found.

Renal Disease

Results again were contradictory and inconclusive. There was no evidence that anesthesia exposure in men led to a significantly higher rate of renal disease than found in control subgroups of non-exposed men. The rate of these diseases among male anesthesiologists was actually lower than that in pediatricians. Likewise, there was no significantly greater rate of renal disease in female anesthesiologists compared with women in their control subgroup. Using a 1 per cent level of significance, the only difference was that between the female AANA and their controls. No mention of the type of renal disease (i.e., congenital or acquired) is made in the report, nor did the data collection form allow determination of time of onset.

Comment

Throughout the study there were internal inconsistencies in the findings. In the cases of some diseases, the authors claimed a significant difference between one exposed subgroup and its controls, while there was no significant difference between another equally exposed subgroup and its controls. There was no evidence of a dose--response relationship in any disease category that showed a larger difference in the highly exposed ASA and AANA groups compared with their controls and a lesser difference when the lesser exposed AORN/T were compared with their control. Accepting a $P$ value of 1 per cent as a criterion for statistical significance, no subgroup had children with an elevated incidence of congenital abnormalities, one of six subgroups had an elevated incidence of cancer (possibly due to data collection bias), two of six subgroups had elevated incidences of hepatic disease (again a result of uncertain data), and one subgroup had an elevated incidence of unspecified types of renal disease. Several of the subgroup comparisons had $P$ values greater than 50 per cent.

The report has drawn conclusions concerning a medical problem and, together with the editorial, has made recommendations for “treatment” of this problem. In spite of these conclusions and recommendations, the authors by their own admission found no cause--effect relationship between the supposed offending agent and the supposed consequence of exposure. There is no certainty that the desired reduction in anesthetic concentrations would be achieved if we were to follow their advice and attach inexpensive gas-evacuation systems to the anesthesia machines and ventilators. Reasons for this are cited in the Ad Hoc Committee Report. In addition, evacuation systems, even ineffective, will not necessarily alter the supposed hazard unless a safe limit of exposure has been defined and concentrations maintained within the known safe limit. Thus, the legal liability and detrimental effect of resident recruitment will not be altered by the use of scavengers.

We are told that proof of a cause--effect
relationship between inhalation of trace amounts of anesthetic vapor and increased disease rates might be forthcoming after all operating rooms are equipped with gas-evacuation systems and the survey repeated. Yet, if the Ad Hoc Committee Report is to be believed, a repeated survey is unlikely to provide an answer. This report suggests that effects of trace anesthetic vapors linger for an indefinite period. Thus, there is no significant decrease in rate of spontaneous abortion or children with congenital malformations among anesthesiologists who have not been exposed to trace anesthetic vapors for longer than a year.

It is not our intention to dissuade anesthesiologists from venting the operating room of anesthetic vapors. We wish to object, however, to statements such as "failure to install exhaust systems and failure to use them at all times represents in the light of present knowledge an unconscionable practice, a practice of exposing oneself as well as others to a demonstrable hazard." The findings of the most extensive study on the subject, the study which defines the problem, "as precisely as possible" do not warrant this kind of statement at this time.

References


Renal Function

ANGIOTENSIN, DISSEMINATED INTRAVASCULAR COAGULATION (DIC), AND ACUTE RENAL FAILURE. The incidence and severity of renal glomerular capillary thrombosis in DIC can be influenced by certain vascular phenomena. Since the renin–angiotensin system plays an important role in the kidney, study of possible interaction between DIC and angiotensin was performed in six groups of rabbits. DIC was produced with an infusion of thrombin. Creatinine levels were determined before and 24 hours after drug infusion. The animals were then sacrificed and the kidneys examined microscopically. Group 1 served as control; group 2 received antithrombin II; group 3 received thrombin; group 4 received thrombin + angiotensin II; group 5 received thrombin + angiotensin II + phenoxycamphetamine; group 6 had bilateral ureteral ligation and served as non-filtering kidney controls. Groups 1 and 2 had normal creatinine levels and kidneys. Group 3 had normal creatinine; 7 of 10 had histologically normal kidneys. Groups 4 and 5 had elevated creatinine levels (similar to rabbits with non-filtering kidneys) and showed extensive microscopic renal changes. The combination of angiotensin II and DIC resulted in severe renal damage, an effect not altered by alpha-blockade. The authors postulate that this mechanism may contribute to glomerular fibrin deposits in acute ischemic renal failure. (Whitaker, A. N., and others: Interaction of Angiotensin with Disseminated Intravascular Coagulation. Am J Pathol 72:1–12, 1973.) ABSTRACTER’S COMMENT: The icing on the cake would have been no renal damage in a group of rabbits receiving angiotensin II + thrombin + an angiotensin blocking agent.