Anesthesiology 59:365, 1983

Operating Room Hypoxia

To the Editor:—A 42-year-old woman was undergoing a total ankle replacement for rheumatoid arthritis. She was under general anesthesia with an endotracheal tube in place. A tourniquet was on her left leg and was powered by the usual tourniquet regulator, which in turn received its driving pressure from a wall control panel.

About 30 min into the surgery, a loud gas leak occurred behind the high-pressure wall control panel. This panel not only drives the tourniquet but also the two sets of power saws the surgeons were using. All attempts were made to stop the hissing gas leak by manipulating the controls but, short of shutting down the whole system, which would have shut down the tourniquet, we could not stop the leak. The surgeons continued to work, even with the hissing behind the panel, because they needed the tourniquet and the power tools to do the operation. The operating room door was kept closed, and traffic was restricted in and out of the room to reduce contamination.

About 30 min later, I noticed that I was getting a headache and investigated the leak that I assumed was

compressed air. Lo and behold it was a pure nitrogen leakl

The patient's inspired oxygen was 40% on the IL meter, the air in the vicinity of the nitrogen leak was 2%, and the general room oxygen measured 18%.

Once we knew the problem, the solution was easy. We plugged oxygen flow meters into the wall oxygen outlets in the operating room and turned them on. That and the room turnover ventilation did the trick. The operating room oxygen went up to 21%, my headache went away, and the case finished uneventfully 1 h later. The cause was found to be a leaky gas regulator behind the panel, which was replaced.

This certainly must be a rare complication but may require a new piece of operating room equipment . . . a canary in a cage!

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(Accepted for publication March 8, 1983.)

Anesthesiology 59:365, 1983

Succinylcholine Tachyphylaxis with Isoflurane

To the Editor:—In their recent article on prolonged succinylcholine infusions, ¹ Donati and Bevan reported accelerated onset of tachyphylaxis and phase II neuromuscular blockade in patients anesthetized with isoflurane when compared with patients receiving N₂O-fentanyl anesthesia. However, in both the abstract and results sections, they stated that this occurred "without affecting succinylcholine requirements."

This apparent contradiction probably resulted from application of inappropriate statistical methods for analysis of the infusion rate data: the authors used unpaired t tests, which not only lack the power of analysis of variance, but also may introduce the problems inherent in performing multiple comparisons. It is apparent from the data of their figure 1 that at all nine times of determination, the infusion rates required with isoflurane anesthesia exceeded the corresponding rates with N₂O-fentanyl anesthesia. Using the simple, nonparametric "sign test," we determined that this corresponds to a value of P < 0.002—highly significant by any standard.

Although we agree with the authors' conclusion that tachyphylaxis and phase II block occur earlier, we contend that based on their own data, there is a highly significant increase in succinylcholine dose requirement during isoflurane anesthesia: a finding that would be expected if tachyphylaxis indeed occurs earlier with this technique.

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(Accepted for publication March 8, 1983.)