equipment design has now eliminated many of these hazards.[1] Pin-indexed oxygen flowmeters mounted on the right of the gas console nearest the outlet manifold are now standard features on many anesthesia machines.[1,2,6] Some machines even have distinct projections on the oxygen flow-control knob to permit rapid touch identification.[7] Human errors resulting in hypoxic inspired mixtures continue to occur, however, despite recent design and safety improvements.[8]

We report a combination of frequently occurring events that can cause inadvertent and insidious delivery of hypoxic inspired mixtures. The technical setting necessary for this potentially lethal situation includes any movable side-mounted anesthesia ventilator adjacent to an anesthesia machine tabletop and any antistatic rubber facemask (fig. 1*A*). Movement of the ventilator toward the anesthesia machine can easily slide the facemask along the tabletop toward a concealed wedge position under a flow control knob (fig. 1*B*). Contact between the sloping nasal end of the rubber facemask and the corrugated flow-control knob can easily rotate the knob clockwise reducing or terminating gas flow producing hypoxic or anoxic mixtures. Hyperoxic mixtures detrimental to newborns can occur if the nitrous oxide or other diluent (air, helium) flow control knob is rotated clockwise.[9] Unsatisfactory anesthesia may result if a Verni-Trol® or copper kettle flow control knob is rotated clockwise. The strong friction interface produced by opposing a rubber surface on a hard corrugated surface can rotate knobs in either direction with little work expended. By convention, clockwise knob rotation will reduce gas flow and counterclockwise rotation will increase gas flow on American-made anesthesia machines.[1]

Prevention of this often concealed hazard is simple and may include: 1) maintenance of an uncluttered tabletop with facemasks placed in drawers or on separate carts; 2) a ventilator mounting permitting unobscured view of the control panel; and most importantly, 3) continuous, visual surveillance of ventilator settings, flowmeters, gas flows, and inspired oxygen concentration.

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Nothing New Under the Sun—A Japanese Pioneer in the Clinical Use of Intrathecal Morphine

To the Editor:—Since the discovery of opioid receptors in the nervous system by Pert and Synder in 1973,[1] intrathecal or epidural administration of narcotics and their analogs has been widely utilized for the purpose of relieving intractable pain, postoperative pain, and pain during parturition. Morphine appears to be the best drug for this purpose, and Yaksh and Rudy[2] are considered to be the first to report the intrathecal administration of morphine in 1976.

But the true history of intrathecal morphine dates back to 1901, 75 years before Yaksh's report. Dr. Otojiro Kitagawa, a Japanese surgeon, presented a paper on the intrathecal injection of local anesthetics at the third annual meeting of the Japan Society of Surgery, held in Tokyo in April 1901. His report describes four cases of intrathecal administration of the local anesthetic "eucaine" for relieving severe pain due to vertebral inflammation which was not improved by intramuscular morphine. To two of these patients Dr. Kitagawa also gave 10 mg of morphine hydrochloride intrathecally in combination with 20 mg of eucaine. One patient was a 33-year-old man who was relieved of severe pain for

several days, and the other was a 43-year-old woman who was relieved of pain in the lower back for two days. Neither respiratory nor circulatory depression was observed following the subarachnoid morphine. The reason respiratory depression did not occur may in part be because Dr. Kitagawa used a large gauge needle which might have caused a significant leakage of CSF into the epidural space, thus minimizing any respiratory depression.

The details of his report appeared in a Japanese journal, *Tokyo Iji Shinshi*, issued on April 13, 1901 (1200:653–658, 1901).

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Potential Pitfalls in Measuring Regional Cerebral Glucose Utilization

To the Editor:—Crosby, Crane, and Sokoloff[1] note that conflicting results regarding the action of ketamine on cerebral metabolism have been reported. The authors used Sokoloff's 2-[^{14}C]deoxyglucose method[2] to estimate regional cerebral glucose utilization. With this method, 2-[^{14}C]deoxyglucose is introduced into circulation and 45 min are required to pass during which time continuous uptake by brain occurs. It is assumed that "steady-state" conditions exist throughout this period. Clearly, this was not the case when Crosby *et al.* studied the short-acting anesthetic ketamine. They gave a single injection of ketamine intravenously 2 to 5 min before 2-[^{14}C]deoxyglucose and waited an additional 45 min before sampling the brain. The dose used should have caused only a short period of anesthesia as borne out by their description: "There was generally at first a 5- to 10-min period of cataleptic-like unresponsiveness followed by a longer period of side-to-side head rocking, and eventually frantic, agitated hyperactivity." From this we can conclude that instead of the single "state of anesthesia" as suggested in the article, the condition being examined was in reality a mixture of anesthesia, awakening, and a post-anesthetic excited state.

It is not surprising that a variety of changes in glucose utilization were found with some areas of the brain being depressed, others excited, while still others were unchanged. What are the readers to conclude from this? It is not possible to assign these changes to any particular phase of anesthesia or post-anesthetic state. The circumstance is somewhat analogous to taking a single long-time exposure photograph of a football game and then trying to reconstruct the various plays from the single blurred image; it cannot be done.

Crosby *et al.* focused their discussion on the fact that they could not reconcile their observations with those of others. They were particularly critical of a study by Hawkins, Hass, and Ransohoff[3] who used a rival technique for measuring cerebral glucose utilization; one which takes only 10 min to complete. Two objections were raised. First, they point out that different results were found. However, they failed to emphasize that Hawkins *et al.* studied ketamine anesthesia over a 10-min period throughout which the rats remained anesthetized. This is in marked contrast to the kaleidoscopic levels of anesthesia that Crosby *et al.* studied. If their technique permitted a 10-min period of metabolic assessment, they might have found that the results were comparable. Second, they go on at some length with several very technical criticisms concerning the possibility of $^{14}CO_2$ loss and the velocity of glucose exchange between plasma and brain. Happily, their concerns are without foundation. Both of these points have been thoroughly considered before, theoretically and empirically, and the results published in respected, well-refereed journals.[3,4] Furthermore, the principles of the methods used by Hawkins *et al.* currently are accepted by several internationally recognized authorities.[5–8]

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