

CORRESPONDENCE

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In Reply:—We appreciate the comments of Snyder and Dwersteg, particularly their statement that the presence of an interpreter and clear communication cannot be overemphasized. However, we disagree that our discussion presented "stereotypes and misinformation gleaned from a limited number of research references." Our extensive literature search revealed the paucity of articles on the management of the deaf-mute patient. None of the reports we cited came from laboratory research but were clinical monographs dealing with audiology.

Despite our clear statement that there was no proof of association between paranoia and deafness, Snyder and Dwersteg stated, "One is left with the distinct feeling that there is." We examined the references carefully on this point. Though some authors imply an association between deafness and paranoia, the association has not been demonstrated but has not been disproved either.

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Believe Your Monitors

To the Editor:—In early December 1992, a potentially fatal episode occurred at our 120-bed community hospital when the oxygen pipeline suddenly became contaminated with 2,200 psi nitrogen.

We had a four-room schedule that morning, with two rooms to start at 7:30 AM and the other two at 8 AM. Before 7:30 AM, the anesthesia equipment in the two early rooms was confirmed to be in order. In one of the rooms, a patient was to undergo a breast biopsy under monitored anesthesia care, and as the intravenous catheter was being inserted in the holding area, the high-pressure-oxygen alarm went off. Since this was illogical and there was no known reason why this should have occurred, it was ignored initially. The patient was taken into the room, the monitors were attached, and it was noted that the in-line oxygen monitor was now indicating 30% O₂, whereas earlier the reading had been 100%. When the patient was given supplemental oxygen *via* nasal prongs, her SpO₂ decreased into the 80s but promptly returned into the 90s when the nasally administered "oxygen" was discontinued. Supplemental oxygen subsequently was administered from the reserve cylinder, and the case proceeded uneventfully. At some point during this event, the wall hose to the gases was disconnected, but subsequently no one was able to remember when this was done.

Unknown to anyone in the operating room suite, a construction crew was starting to work on the oxygen pipelines in the attached Skilled Nursing Facility at about 7:30 AM. This crew did not notify anyone that they intended to work on the oxygen lines, they did not

shut off the zone valve between the skilled nursing facility and the rest of the hospital, and they purged the pipeline by connecting a 2,200-psi nitrogen cylinder directly to the pipeline without benefit of a reduction valve between the cylinder and the pipeline. This high-pressure nitrogen quickly contaminated all the oxygen lines in the hospital.

Shortly after 7:30 AM, surgery began in the other early room with a thoracoscopy in an otherwise healthy woman with a persistent pneumothorax. Although ventilation *via* mask was somewhat difficult, we were surprised that her SpO₂ decreased into the 60s in the first minute after induction. About this time, it was reported that the overpressure alarm had gone off and the oxygen pressure was being indicated as 64 psi. This initially did not concern us nearly as much as her decreased SpO₂ did until the in-line oxygen monitor alarmed shortly thereafter, indicating a low oxygen concentration that eventually decreased to 7% O₂.

At this point, we concluded that something other than oxygen was in our pipeline. The reserve cylinder was turned on, her SpO₂ increased into the 70s, and about a minute later, the wall hose was disconnected, after which her SpO₂ promptly increased into the upper 90s. The surgery was completed without further mishap, and the patient suffered no ill effects from this episode.

The construction crew made at least three major mistakes. First, they did not notify anyone in the operating room suite that there was to be work done on the gas lines. Second, they did not shut off the

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zone valve that isolates the Skilled Nursing Facility from the rest of the pipeline system; and third, they did not use a reduction valve between the nitrogen cylinder and the pipeline. Subsequently, we were not able to detect a leak in the pipeline, despite the severe overpressure to which it had been subjected.

A point of note for anesthesia personnel: most pipelines have a functional range between 50 and 55 psi, with a low-pressure alarm set to go off at 45 psi and a high-pressure alarm set to go off at 60 psi. If the supply fails and the line-pressure drops, it is only necessary to open the reserve cylinder and the manifold on the anesthesia machine will repressurize at about 48 psi. But in an overpressure situation such as occurred in this case, the oxygen reserve cylinder reduction valve will not open until the manifold pressure is less than about 48 psi. Therefore, it is necessary to disconnect the wall hose

in an overpressure situation for the reserve cylinder to function properly.

We were lucky in that, during this brief episode, no patients were undergoing mechanical ventilation of their lungs while in the intensive care unit, nor were any patients receiving nasal oxygen anywhere in the hospital.

Our advice therefore is, no matter how illogical it seems and no matter that it has never occurred previously to you, be very suspicious when your monitors provide unexpected data.

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Periarterial Lidocaine/Hydralazine Aids Arterial Catheter Insertion in Patients with Preeclampsia

To the Editor:—Patients who need intraarterial pressure monitoring often are those who pose the greatest difficulty with catheter insertion, especially parturients with severe preeclampsia. Part of the pathophysiology of this disease is arterial vasoconstriction. In some of these patients, although the blood pressure is increased, the arteries are constricted, hard to palpate, and prone to developing spasm. Periarterial lidocaine injections do not seem to help. After several unsuccessful attempts to cannulate a constricted artery in a patient with severe preeclampsia, I mixed approximately 1 mg hydralazine with 1 ml 1% lidocaine in a syringe and reinjected around the artery. Five minutes later, the same artery had a bounding pulse, and I was able to insert the catheter without difficulty.

Before attempting arterial catheter insertion in patients with preeclampsia in whom the artery is difficult to palpate, I now add 2 mg hydralazine to 2 ml 1% lidocaine. A small amount of this mixture is

injected around the radial artery. If the radial artery cannot be palpated, I inject around the brachial artery in the antecubital fossa. The injection is performed 5 min before attempting catheter insertion.

Although this technique was successful in seven of eight patients with preeclampsia, it did not dilate the arteries in three older, chronically hypertensive patients. In no case has any change in blood pressure been associated with this technique.

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