An Alarming Problem

To the Editor:—The derivation of the word monitor is familiar to anyone who has attended or read any of the American Society of Anesthesiologists' refresher courses. Monitoring in anesthesia has grown rapidly from the early days of relatively simple monitoring, such as a finger on the radial pulse or a precordial stethoscope. Nowadays the well-trained anesthesiologist is faced by a battery of monitoring devices that may tower to alarming heights over the anesthesia machine (fig. 1). Some warn of airway disconnects, some of blood pressure, either too low or too high. Some even have small slave monitors with their own readouts, printouts, and perhaps in the not too distant future, takeouts. What this battery of warning devices have in common is that they warn, some with a blinking light, some with an audible alarm, and herein lies the source of yet another problem. It is sometimes impossible to tell among the massed bank of monitors which one is in fact alarming. This is because most of the audible warning devices sound very similar in tone, possibly because most alarm systems are made by the same company. I would make a plea to the manufacturers to build into their systems a very different audible alarm such that it would be immediately recognizable as coming from, say, an oxygen saturation monitor or an automatic blood pressure device. At best we could perhaps play "Galway Bay" on the massed bank of monitoring devices; at worst such a system of different audible sounds and pitches would help in measurably to find which monitor was alarming and perhaps help to cut down one’s perioperative anxiety.

![Fig. 1. Arrows indicate some of the independent alarm systems.]

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The Fiberoptic Rhinoscope in Adult Intubation

To the Editor:—The use of the fiberoptic bronchoscope or laryngoscope in patients where difficult intubation is present or expected is well described. The fiberoptic rhinoscope (Olympus ENF-P®) has been used in children with juvenile chronic arthritis as a guide wire for tracheal intubation, allowing a 4.5-mm tube to be inserted over it.1 However, the rhinoscope also can be used with advantage for adult intubation in situations where the fiberoptic technique is considered appropriate.

The rhinoscope, which has an outer diameter of 3.7 mm, has a working length of 25 cm when combined with an Endotrol® tube (National Catheter Corp.). The tube (no. 6, 7, or 8) is cut at this length and the scope inserted, with its distal end corresponding to the distal end of the
The maneuverability of the scope and tube is, due to the guide-hook of the Endotrol® tube, nearly as good as that of the longer broncho/laryngoscope but can be supplemented with changes in head position or external manipulation of the thyroid cartilage in “threading the trachea on to the tube.”

This technique is less traumatic to the paralaryngeal structures than the traditional blind nasal intubation. In a few patients, it is necessary to reestablish the original length of the tube after intubation. This is easily done by inserting an internal metal connection between the two separated parts of the tube.

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REFERENCES

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Cardiac Output Effects of Nitroglycerin in Experimental Lung Injury

To the Editor—Benoit et al. reported that nitroglycerin (TNG) produced a 25% decrease in cardiac output when administered to dogs with oleic acid pulmonary edema; this result contrasts with our previous report of a 40% increase in cardiac output in a similar model. These opposite effects may be explained by differences in the two protocols interacting with the three different vasodilator effects of TNG. TNG is a systemic arterial dilator, a systemic venodilator, and a pulmonary arterial dilator, so that the overall effect on cardiac output is a function of baseline volume status, systemic and pulmonary vascular tone, right and left heart function, and dose of TNG. Cardiac output usually is limited by left ventricular performance, and the predominant effect of TNG is usually systemic venodilation. Therefore, under normal hemodynamic conditions, TNG will decrease left ventricular preload and cardiac output; these effects will be most pronounced in hypovolemic subjects. The oleic acid protocol used by Benoit et al. created similar conditions, and the predominant hemodynamic effect of TNG was a reduction of cardiac output. Our protocol was designed to produce a model of pulmonary hypertension rather than simply acute lung injury. We therefore chose an experimental design that produced an increase in pulmonary artery pressure (PAP) as well as in pulmonary vascular resistance (PVR). Oleic acid has a very steep dose–response curve. In preliminary experiments, we found that doses similar to those used by Benoit et al. increased PVR but did not increase PAP. We therefore used a higher dose of oleic acid (0.10 ml/kg), which, prior to TNG administration, resulted in a 46% increase in PAP and a 155% increase in PVR; the protocol used by Benoit et al. produced only an 18% increase in PAP and a 78% increase in PVR. We believe the pulmonary hypertension that occurred with our protocol produced a hemodynamic situation similar to chronic pulmonary hypertension. In this situation, cardiac output is limited by right rather than left ventricular performance, so that pulmonary vasodilation may reduce right ventricular afterload and result in an increase in cardiac output. We found that TNG produced a 43% decrease in PVR and a 40% increase in cardiac output. The marked pulmonary vasodilator effects