FIG. 1. The assembled rhinoscope and Endotrol® tube.

tube, thereby allowing visualization of the otherwise blind nasal intubation in the spontaneously breathing, sedated, and analgesic patient (fig. 1).

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."[2]

This technique is less traumatic to the paralaryngeal structures than the traditional blind nasal intubation. In a few patients, it is necessary to restablish 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.

SØREN ERIKSEN, M.D.
Department of Anesthesiology
Haderslev Sygehus
DK-6100 Haderslev
Denmark

REFERENCES

1. Smith BI: Anaesthesia and Still's disease. Anaesthesia 40:209, 1985
2. Gillespie NA: Endotracheal Anaesthesia. Madison, University of Wisconsin Press, 1948, pp 101–114

(Accepted for publication August 12, 1985.)

Anesthesiology
64:129–130, 1986

Cardiac Output Effects of Nitroglycerin in Experimental Lung Injury

To the Editor:—Benoit *et al.* reported that nitroglycerin (TNG) produced a 26% decrease in cardiac output when administered to dogs with oleic acid pulmonary edema[1]; this result contrasts with our previous report of a 40% increase in cardiac output in a similar model.[2] 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.[3] 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.[4] We found that TNG produced a 43% decrease in PVR and a 40% increase in cardiac output. The marked pulmonary vasodilator effects

of TNG in our protocol are consistent with data that vasodilators are most effective when baseline vascular resistance is high.[2,5]

The hypothesis that right and not left ventricular performance limited cardiac output in our protocol was confirmed by the effects of sodium nitroprusside (SNP) in our study. SNP is more potent than TNG as a systemic arterial vasodilator and should therefore result in a greater increase in cardiac output when left ventricular performance limits cardiac output. In our study, SNP produced only a 14% increase in cardiac output and did not affect pulmonary artery pressure or resistance at doses that produced similar decreases in systemic arterial pressure compared with TNG. The different hemodynamic effects of these two drugs in our study are explained by their relative potencies as pulmonary and systemic vasodilators in a model where pulmonary vascular resistance limits cardiac output.

The cardiopulmonary effects of TNG in humans will depend upon the patient population selected. When TNG is administered to patients with adult respiratory distress syndrome in whom cardiac output is not limited by pulmonary vascular resistance, the result will be arterial hypoxemia, and, if the patient is hypovolemic, decreased cardiac output and hypotension may occur. In contrast, when TNG is administered to patients with severe pulmonary hypertension in whom cardiac output is limited by right heart afterload, an increase in cardiac output may occur[6]; the effects on arterial oxygenation will depend upon the degree to which hypoxic pulmonary vasoconstriction was maintaining arterial oxygenation. Further studies are required to determine the risks and benefits of TNG therapy in selected subsets of patients with adult respiratory distress syndrome.

RONALD G. PEARL, M.D.
Assistant Professor of Anesthesia
Department of Anesthesia, S-278

MYER H. ROSENTHAL, M.D.
Associate Professor of Anesthesia, Medicine, and Surgery
Intensive Care Unit, P-2043

Stanford University
Medical Center
Stanford, California 94305

REFERENCES

1. Benoit A, Ducas J, Girling L, Schick U, Prewitt RM: Acute cardiopulmonary effects of nitroglycerin in canine oleic acid pulmonary edema. ANESTHESIOLOGY 62:754–758, 1985
2. Pearl RG, Rosenthal MH, Ashton JPA: Pulmonary vasodilator effects of nitroglycerin and sodium nitroprusside in canine oleic acid-induced pulmonary hypertension. ANESTHESIOLOGY 58: 514–518, 1983
3. Hill NS, Antman EM, Green LH, Alpert JS: Intravenous nitroglycerin: A review of pharmacology, indications, therapeutic effects and complications. Chest 79:69–76, 1981
4. Franciosa JA, Fischer HA: Right ventricular unloading: Lessons from the left (editorial). Ann Intern Med 95:647–648, 1981
5. Voss GI, Katona PG, Dauchot PJ: Effectiveness of sodium nitroprusside as a function of total peripheral resistance in the anesthetized dog. ANESTHESIOLOGY 62:130–134, 1985
6. Pearl RG, Rosenthal MH, Schroeder JS, Ashton JPA: Acute hemodynamic effects of nitroglycerin in pulmonary hypertension. Ann Intern Med 99:9–13, 1983

(Accepted for publication August 13, 1985.)

Anesthesiology
64:130–131, 1986

The Hemodynamic Effects of Positive End-expiratory Pressure

To the Editor:—Although Venus *et al.* have presented an explanation of the data from their study on the effect of hydration on renal function during ventilation with positive end-expiratory pressure (PEEP), I feel that several aspects require additional comment.[1] The study protocol was designed to compare renal function during PEEP in two groups of animals that differed only in the degree of hydration as measured by transmural left ventricular end-diastolic pressure ($LVEDP_{TM}$). After the addition of PEEP to controlled mechanical ventilation (CMV), $LVEDP_{TM}$ remained at 5 ± 1 mmHg in the normovolemic animals and at 10 ± 1 mmHg in the hydrated group as a result of infusion of lactated Ringer's solution. In spite of no change in $LVEDP_{TM}$ in the normovolemic group during ventilation with PEEP, there was a 35% decrease in cardiac output (CO) and a 20% decrease in mean arterial pressure (MAP). If ventricular contractility was not affected, the above results suggest that, although $LVEDP_{TM}$ was unchanged after instituting PEEP, there was a decrease in left ventricular compliance resulting in a decreased left ventricular preload. In reviews of their own data as well as that of other investigators, Robotham *et al.* have presented evidence that left ventricular compliance is altered during ventilation with CMV with PEEP while contractility remains unchanged.[2,3] Therefore, $LVEDP_{TM}$ is not an accurate measure of left ventricular