The anesthetic management of patients with this condition has been extensively reviewed.[1] One problem for the anesthesiologist is to correctly locate the endotracheal tube (ETT) tip distal to the origin of the fistula but proximal to the carina. Appropriate positioning results in minimal gastric distension, minimal loss of tidal volume, and minimal environmental pollution when ventilation is assisted. Recently, Baraka *et al.*[2] described two newborns safely anesthetized with selective bronchial intubation who underwent surgical repair of TEF. Unfortunately, these authors reported only one arterial blood gas and no pulse oximetry readings during the period of one-lung ventilation, raising concern as to the safety of their technique. Furthermore, left-sided endobronchial intubation in the newborn is technically very difficult, while a right-sided intubation is frequently associated with right upper lobe collapse due to the short distance between the carina and the origin of the right upper lobe bronchus.

Two techniques have been described for placing the ETT in the correct location. The first requires insertion of a gastrostomy tube under a water seal.[3] When positive pressure is applied to the ETT, bubbles will be seen emerging from the gastrostomy tube if the ETT tip is proximal to the fistula. This method is slow, requires an assistant, and increases the risk of infection and hypothermia due to spillage of the fluid used. A second technique deliberately places the ETT tip distal to the carina and into one of the mainstem bronchi. Subsequently, the ETT is withdrawn to the point where bilateral breath sounds are heard, thereby locating the ETT tip just proximal to the carina.[4] With this technique the patient is at risk of bronchospasm unless deep levels of anesthesia are maintained during the procedure.

We report a simple technique that we have found useful for confirming the position of the ETT in an infant with TEF who had a gastrostomy tube in place. The method involves loosely connecting the gastrostomy tube to the sampling catheter of a side-stream type capnograph or dedicated anesthesia gas analyzer, such as the Datex/Puritan Bennett PB 254 (Puritan Bennett Co., Wilmington, MA). When the ETT tip is proximal to the fistula, the presence of $CO_2$ and inhaled agents is detected by the analyzer. When the tip is immediately distal to the fistula, no $CO_2$ or inhaled agents are detectable. Thus, during gentle positive pressure ventilation *via* the ETT, the intermittent analysis of the gases emerging from the gastrostomy tube facilitates the precise location of the ETT. Once the ETT tip is correctly positioned, the ETT is secured in the usual way. In our experience, this technique facilitates the rapid determination of the optimum position for the ETT tip. This is a desirable situation in an infant prone to hypoxia and is reproducible intraoperatively when even small movements of the head may easily displace the ETT proximal to the fistula or into a main stem bronchus.

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## Influence of Anesthetics on Relaxation Times

*To the Editor:*—Several recent papers and an accompanying editorial, all of which discussed nuclear magnetic resonance (NMR) as a measurement technique,[1–3] did not mention the fact that anesthetics can measurably change the relaxation times of NMR nuclei because they fluidize lipids. The effects of anesthetics on NMR relaxation times are not well understood. It would seem appropriate for anesthesiologists to contemplate the anesthesia-related findings both *in vivo* and *in vitro*.

Several theories and models have been proposed to account for the dissimilarities of relaxation behavior in normal and pathological tissues. Each is fraught with pro and con arguments. Recently, Akber has proposed an alternative approach to explain the enigma of relax-

ation behavior in biological systems.[4-6] The hypothesis states that the dissimilarities in relaxation time in normal and pathological tissue are due to the concentration of paramagnetic molecular oxygen ($O_2$) in cell-associated water.

NMR relaxation time is the length of time it takes for the spins to return to their equilibrium distribution, in which transverse magnetism is zero and longitudinal magnetization is at its maximum value, aligned in the direction of the static magnetic field. The transverse magnetization decays toward zero with a characteristic time constant $T_2$, and the longitudinal magnetization returns toward the equilibrium distribution with a characteristic time constant $T_1$. To comprehend the concept of $T_1$ in simple terms, consider the trunk of a tree as the static magnetic field. If a branch of this tree which is aligned to the trunk at a certain angle is pulled down and then released, the time it takes for the branch to return to its original position is called relaxation time. The tipping of spins out of alignment in the NMR system is done by radiofrequency radiation (RF), and when RF has ceased or is stopped, the nuclei tends to return to its original position with a time constant $T_1$.

Schneindlin *et al.*[7] reported the effects of ketamine and sodium pentobarbital on increased hepatic $T_1$, $T_2$ in rats *in vitro*. Similarly, Barany *et al.*,[8] assessing C-13 spectra, reported increased $T_1$ of methylene (30.5 ppm) and polyunsaturated (129 ppm) resonance in phospholipids of brain excised from halothane-anesthetized rats, and in halothane-treated brains, with respect to control.

Lipid-mediated mechanisms of anesthesia require paramagnetic molecular $O_2$, either directly as a substrate, or indirectly because of their dependence upon cellular chemistry. For example, oxidative metabolism of halothane consumes two $O_2$ atoms for each molecule of halothane metabolized. The oxidative process is more rapid than the reductive metabolism of halothane at all $P_{O_2}$ levels.[9,10] The oxidative metabolism of halothane initiates the hypoxic process, whereas reductive metabolism produces free radicals, and $O_2$ is by far the best proton acceptor.[11] Rosenberg *et al.** also observed anesthesia-related hypoxia in H-1 NMR spectra of brain, similar to unanesthetized hypoxia, thereby confirming initiation of the hypoxic process with anesthetics. I propose that it is the increase in $O_2$ consumption in the presence of anesthetics, together with the decreased concentration of $O_2$ in the cell water, that influences the relaxation mechanism.

With the increasing role of NMR spectroscopy and NMR imaging in clinical investigation, it should be worthwhile for anesthesiologists to investigate the influence of anesthetics on relaxation times, $O_2$ consumption, and the correlation between relaxation time and $O_2$ concentration in different organs. Clinical anesthesiologists should also be aware of changes in NMR spectra and image intensities under the influence of anesthetics.[8,12]

Further experimental work on the complicated mechanisms of relaxation time and NMR spectroscopy under the influence of anesthetics will reveal new insight into and understanding of both biological and physical mechanisms of relaxation times in normal and pathological tissues.

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