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A Balloon Catheter for Nasal Intubation

To the Editor:—We recently experienced a complication from nasal intubation when a small nasal polyp was dislodged into the trachea by the endotracheal tube. Because of that experience, we now insert a Foley balloon catheter to obturate the end of the endotracheal tube so that it cannot carry a foreign body into the trachea (fig. 1). Because of its diameter, flexibility, length, and balloon size, a Foley 12 F catheter is the best for nasal intubation in adult patients. The catheter should be well lubricated, suitably placed in the endotracheal tube, and inflated with air using a three-way stopcock before intubation. After intubation is completed, the catheter should be removed with the balloon deflated. We have not experienced any problems with this technique and believe that this method will reduce the incidence of complications associated with nasal intubation.

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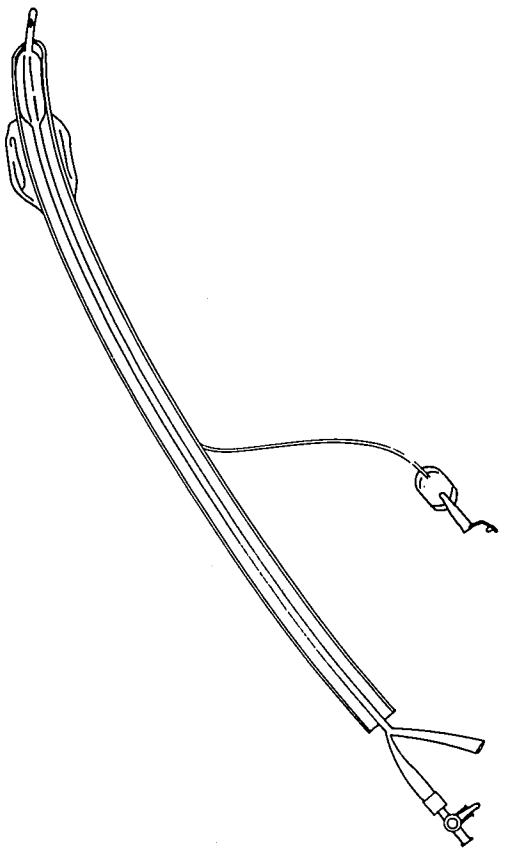

FIG. 1. A balloon catheter placed in the nasotracheal tube.

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Concepts of Thiopental Distribution and Metabolism, Old and New

To the Editor:—In a recent issue, Burch and Stanski report a study of thiopental (TPL) pharmacokinetics with frequent early arterial blood collection.[1] This study was done to ascertain the relative importance of redistribution and metabolism in recovery from TPL anesthesia. They reached opposite conclusions than those of Saidman and Eger, who used physiologic modeling to answer the same question 15 years ago.[2] As these two reports represent different approaches to the same question, I compared them further.

For the comparison, I recreated the model of Saidman and Eger on a digital computer. I then simulated blood TPL levels following a 6 mg/kg bolus injection at the blood collection times used by Burch and Stanski. Also, the metabolic loss/total ratio as defined by Burch was calculated from the simulations. Two simulations were done; one assuming a hepatic extraction ratio of 0.15, which is close to the accepted value and one assuming a hepatic extraction ratio of 0.30, which was the value used by Saidman and Eger for their conclusions.

The simulated blood levels assuming a hepatic extraction ratio of 0.15 are almost identical to the mean values reported by Burch and Stanski from the time period of 1–15 min. Also, simulated blood levels assuming a

TABLE 1. Measured and Simulated Thiopental Levels Using Physiologic Model of Saidman and Eger

Source	Concentration at 1 min	ML/TL at 1 min	Conc at 5 min	Conc at 15 min	ML/TL at 15 min
Burch (measured)	32 ± 12 μg/ml	0.14 ± 0.06	13 ± 1	6.9 ± 0.62	0.18 ± 0.04
Saidman (simulated hep. ext. 15%)	29.8	0.04	14.0	7.0	0.13
Saidman (simulated hep. ext. 30%)	28.7	0.08	12.8	5.8	0.23

hepatic extraction ratio of 0.30 are only slightly lower than the mean values reported by Burch and Stanski (table 1).

Simulated blood levels are lower at 0.5 min than those reported by Burch and Stanski. There are two probable reasons for this: 1) the model represents plasma and red blood cells (RBC) as one compartment; and 2) intravascular mixing may be incomplete at 0.5 min, causing higher measured serum levels. There is experimental evidence that these two factors are important in interpreting serum TPL levels within the first minute after administration. Igari *et al.* have shown that equilibrium between rat serum and RBC TPL is attained in 0.5 min.[3] Also, Crankshaw *et al.* have shown in dogs that peak TPL levels after a 5-s administration are twice levels seen after the same dose is given over 15 s.[4]

Different recovery criteria was a major factor leading Saidman and Eger to different conclusions from Burch and Stanski. Saidman and Eger chose as their end point the time at which blood levels of TPL were 3.5% of their initial value. This point is well past awakening after a 6 mg/kg dose and also past the rapid distribution phase and into the slow distribution phase. Thus, blood levels of TPL are influenced by metabolism and this time. Burch and Stanski chose 15 min after bolus injection as their definition of recovery from TPL anesthesia. At this point, the simulated ML/TL ratio, using Saidman's model assuming 30% hepatic extraction is only slightly higher (0.23 *vs.* 0.18) than that reported by Burch. Neither Saidman or Burch discuss criteria for recovery from TPL anesthesia.

Burch and Stanski state that one problem with a physiologic model is that many assumptions must be made in creating the model. This is true. But they also have made two crucial assumptions in deriving a metabolic loss/total loss ratio from their data: 1) clearance is constant over the range of drug concentrations involved in the calculation; and 2) mixing of drug within central compartment (presumably plasma) is instantaneous. Interestingly, Stanski *et al.*[5] previously reported Michelis-Menton kinetics in a few patients with serum TPL levels greater than 60 μg/ml. Mean serum TPL levels reported by Burch and Stanski during the first minute following TPL administration ranged from 32–93 μg/ml.

In summary, this comparison led to the following conclusions: 1) both the physiologic model of Saidman and Eger (even using a hepatic extraction ratio of 30%) and the experimental data of Burch and Stanski show the predominant role of redistribution in the rapid fall of arterial TPL levels in the first 15 min following bolus administration; 2) defining arterial levels (presumably reflecting brain levels) of TPL at which recovery occurs is crucial in assessing the relative impact of redistribution and metabolism on recovery; 3) the ability of a simple flow limited model to predict TPL levels in normal subjects during the first 15 min after bolus administration suggests that tissue binding of TPL and albumin-TPL dissociation must be rapid processes.

The work of Burch and Stanski is significant. It demonstrates experimentally in humans, the importance of redistribution in recovery from usual induction doses of TPL. Also, their method of early rapid arterial blood sampling should serve as a model for the design of future pharmacokinetic studies of drugs thought to have rapid distribution.

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REFERENCES

1. Burch PG, Stanski DR: The role of metabolism and protein binding in thiopental anesthesia. ANESTHESIOLOGY 58:146–152, 1983
2. Saidman LJ, Eger EI: The effect of thiopental metabolism on duration of anesthesia. ANESTHESIOLOGY 27:118–126, 1966
3. Igari Y, Sugiyama Y, Awazu S, Hanano M: Comparative physiologically based pharmacokinetics of hexobarbital, phenobarbital, and thiopental in the rat. J Pharmacokinet Biopharm 10:53, 1982
4. Crankshaw DP, Rosler A, Ware M: The short term distribution of thiopentone in the dog. Anaesth Intensive Care 7:148–151, 1979
5. Stanski DR, Mihm FG, Rosenthal MH, Kalman SM: Pharmacokinetics of high-dose thiopental used in cerebral resuscitation. ANESTHESIOLOGY 53:169–171, 1980

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