TABLE 2. Examples Comparing HCO_3^- and BE_3 in Acid–Base Imbalance

	pH	P_{CO_2} (torr)	$[HCO_3^-]$ (mEq/l)	BE_3 (mEq/l)
Normal	7.4	40	24.5	0
Hypercapnia				
Acute	7.16	80	27.6	0
Chronic	7.33	80	41.2	+14.5
+Ventilator	7.585	40	38.5	+14.5
Metabolic alkalosis				
Acute	7.56	42	38.4	+14.5
Chronic	7.50	50	39.2	+14.5
Metabolic acidosis				
Acute	7.27	35	15.5	−10
Chronic	7.315	30	15.0	−10
Hypoxia (Pa_{O_2} = 50 torr) giving respiratory alkalosis				
Acute	7.43	37	24.2	0
Chronic	7.415	30	19.0	−4.9
+O_2, 10 min	7.39	32	19.2	−4.9
Hyperventilation	7.635	20	21.6	0

hagen," but as "pure" chronic respiratory acidosis, and thus no metabolic alkalosis, by "Boston." For example, when P_{CO_2} is stable at 80 torr, and HCO_3^- is 41.5 mEq/l, this is reported as BE_3 = 14.5 mEq/l in Copenhagen parlance and a zero metabolic alkalosis in Boston.

It seems to me that both are right, and both answers are needed. I believe the laboratory report cannot make clinical judgments, but should provide chemical characterizations. The report should include P_{CO_2}, P_{O_2}, *p*H, BE_3, and HCO_3^-. Clinical interpretation may then proceed from these data to reason as follows:

1) BE_3 measures total non-respiratory or "metabolic" deviation from normal, whether renal, compensatory or metabolic in origin.

2) ΔHCO_3^- calculated as described by Dr. Levesque herein measures the deviation of this patient's state from that found in average patients with similar steady states.

3) 100 BE_3/BE_c estimates percentage compensation to an observed hypercapnia.

JOHN W. SEVERINGHAUS, M.D.
Professor of Anesthesia
University of California
San Francisco, California 94143

(Accepted for publication April 8, 1977.)

A Safer IV Catheter

To the Editor:—A recent article by Drabinsky[1] describing two cases of operative intervention for removal of an intracardiac "cath-embolus" prompts me to call attention to recent improvements in catheter technology that should make obsolete the problem of cut catheters and resultant catheter emboli. This accident usually occurs during catheterization of the subclavian or internal jugular vein when a catheter is inserted and manipulated through a cutting metal needle. The introduction of a closed catheter system enables the removal and discarding of the cutting metal needle after percutaneous venipuncture and prior to insertion of the preattached long catheter into the vascular system.*

In this design (fig. 1), a metal needle protrudes through a flexible self-sealing rubber tube, which is attached to a short "around-needle" introduction catheter. A long placement catheter is encased in a malleable plastic cover connected to the distal end of the rubber tube. Upon venipuncture, the needle

* Advanset, C. R. Bard Company, Murray Hill, New Jersey.

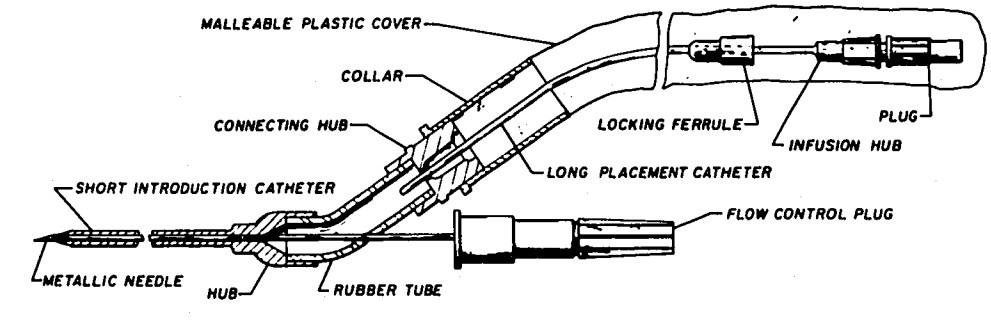

FIG. 1 (*above*). Design of the system.

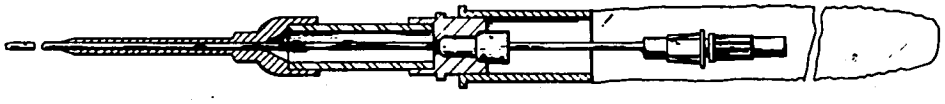

FIG. 2 (*below*). Insertion of the placement catheter.

is removed and discarded and as the rubber tube straightens (fig. 2), the placement catheter is inserted through the rubber tube and short introduction catheter into the vein. Should the long catheter require manipulation, there is no danger of cutting it, as the placement tube through which it protrudes has no cutting edge. The presence of a closed system decreases the danger of air embolus and the long catheter, being already attached to the system, decreases blood loss and the danger of contamination of the hub.

BORIS SCHWARTZ, M.D.
Attending Surgeon
Greater Paterson General Hospital
Wayne, New Jersey 07470

REFERENCE

1. Drabinsky M: Retrieval of embolized central venous catheters by a Dormia ureteral stone dislodger with a straight filiform tip. Chest 69:435–437, 1976

(Accepted for publication March 31, 1977.)

Fasting and Metabolism

To the Editor:—It has been my experience when working with nephrectomized rats that the trauma associated with nephrectomy causes them to stop eating. I am curious to know whether Dr. Ghoneim *et al.* had the same experience.[1] If the nephrectomized rats used in their study did stop eating, then they would have been "fasted" animals, and this would have had considerable impact on the interpretation of the results. It is well known that fasting, even for short periods, greatly alters hepatic metabolism of barbiturates, causing a significant increase in the "sleep time" of rats and presumably increasing plasma free barbiturate.[2] In addition, fasting causes blood free fatty acids to increase. Free fatty acids have been shown to be direct competitive inhibitors with thiopental for albumin protein-binding sites.[3] While I agree with the authors concerning the conclusions of their study, other interpretations are possible, depending on the nutritional state of the rats.

DAVID J. MILETICH, PH.D.
Director, Anesthesia Research Laboratories
Assistant Professor of Anesthesiology
Michael Reese Hospital and Medical Center
Pritzker School of Medicine
University of Chicago
Chicago, Illinois 60616

REFERENCES

1. Ghoneim MM, Pandya HB, Kelley SE, et al: Binding of thiopental to plasma proteins: Effects on distribution in the brain and heart. ANESTHESIOLOGY 45:635–639, 1976
2. Campbell TC, Hayes J: Role of nutrition in the drug-metabolising enzyme system. Pharmacol Rev 26:171–197, 1974
3. Rudman D, Bixler TJ, Del Rio AE: Effect of free fatty acids on binding of drugs by bovine serum albumin, by human serum albumin and by rabbit serum. J Pharmacol Exp Ther 176:261–272, 1971

(Accepted for publication April 1, 1977.)

To the Editor:—I thank Dr. Miletich for his interest in our paper. He suggests that the increases in sleep times and the unbound thiopental levels in nephrectomized rats may have been due to alteration of hepatic metabolism and increase of the plasma free fatty acid concentration by fasting.

It is unlikely that alteration of hepatic metabolism would increase sleep time. Novelli *et al.*,[1] in a recent study in rats, found that portal injection of thiopental or pretreatment of the animals with microsomal enzyme inducers or inhibitors did not modify the duration of action of the drug.

Increased plasma free fatty acid concentrations can compete with many acidic drugs for albumin-binding sites. The contribution of plasma free fatty acids to the reduction of plasma protein binding of thiopental in our experiments is unknown, since we did not measure plasma free fatty acids. There is, however, evidence for a qualitative change in the plasma proteins in uremia.[2]

M. M. GHONEIM, M.D.
Professor
Department of Anesthesia
University of Iowa
College of Medicine
Iowa City, Iowa 52242

REFERENCES

1. Novelli GP, Marsili M, Lorenzi P: Influence of liver metabolism on the action of althesin and thiopentone. Br J Anaesth 47:913–916, 1975
2. Shoeman DW, Azarnoff DL: The alteration of plasma proteins in uremia as reflected in their ability to bind digitoxin and diphenylhydantoin. Pharmacology 7:169–177, 1972

(Accepted for publication April 1, 1977.)