Anesthesiology
61:478, 1984

A Simple Accurate Technique for Establishing Zero Reference Levels for Pressure Measurements

To the Editor:—When measuring pressures, the "zero" on the manometer scale or the transducer height must be at the same level as the cavity in which we wish to measure the pressure. Several devices have been described to avoid errors in extrapolating levels in the human body to the scale or the transducer. All have similar defects: they occupy too much space, they are difficult to keep clean, and they are awkward in use.

For the last 10 years we have been using a system that avoids these drawbacks, is inexpensive, and can be improvised with materials readily obtainable in any hospital. It is based on a technique widely used in construction.

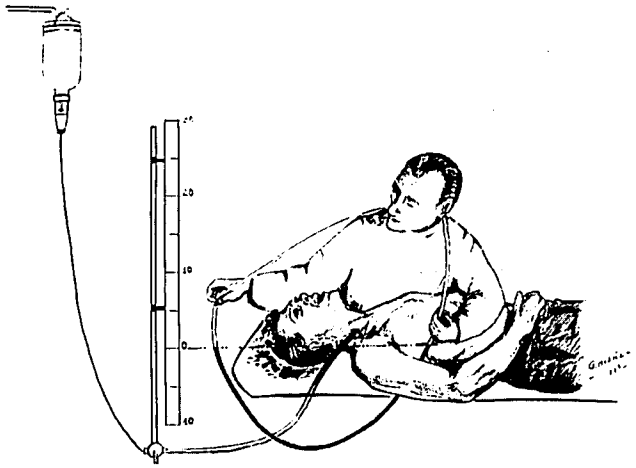

FIG. 1. Reference for central venous pressure is shown above.

The system consists of a transparent and highly flexible non-wettable tube. We use a silicone tube 250 cm with an internal diameter of 0.6 cm, although the diameter and length can vary. Colored fluid is introduced into the tube along with a small amount of alcohol in order to reduce surface tension and bubble formation during transportation or handling. Once the tube is half full of fluid, both ends are closed and then connected.

As can be seen in figure 1, the technique consists of setting one part of the tube at the reference body level and another part close to the scale or the transducer. The scale or transducer is lifted or lowered until the fluid level coincides with that at the body level. Placing this device around the operator's neck greatly facilitates the technique.

FERMÍN FERNÁNDEZ-CANO
Chief of Anesthesiology and Critical Care
Hospital Civil de Bilbao

Professor of the School of Medicine
Universidad del País Vasco
Bilbao, Spain

(Accepted for publication April 24, 1984.)

Anesthesiology
61:478–479, 1984

Emergency Equipment in the Labor Suite

To the Editor:—Epidural analgesia for labor has been a service provided by our Anesthesia Department for two decades. This usually has been performed by a physician in the labor suite with monitoring of the patient by the accompanying CRNA. To avoid aspiration pneumonitis, a reliable suction is necessary. The problem arises in determining beforehand what constitutes adequate preparation for an event that may occur only once in several years.

TABLE 1. Suction Assembly Time

Group	Number	Average Time (s)
CRNAs	10	70.3
SRNAs	3	91.0
Residents	6	121.0
Staff anesthesiologists	5	118.8

Our newly constructed labor rooms have wall suction plumbing, but the wall suction units are kept in the bottom shelf of a work cart, which is brought into the labor room for epidural anesthesia. We recently have measured how long it takes to assemble the wall units.

Twenty-four members of the anesthesia department assembled the wall unit (the CRD system of T. M. Medivac Corp. Abilene, Texas) from the components

on the cart. These components included a suction male quick coupler, a recepticle, a length of suction tubing, and a Yankauer suction. The participants were told of the test beforehand but were not provided with practice trials. A stop watch was used for timing each participant.

The average time required for assembling the suction unit was 95.8 s (table 1). No individual took less than 35 s, one took more than 200 s. Only three individuals were able to assemble the unit to function in less than 1 min.

It seems unlikely that aspiration could be avoided in an actual emergency when using this equipment considering the time delays noted. The presence of disassembled suction units on a cart should not be construed as "availability" of wall suction. We recommend that assembled wall suction units be kept in all labor rooms, ready for immediate use whenever regional anesthesia is administered. We further recommend that routine drills in the use of emergency equipment be carried out for all members of anesthesia departments, regardless of their level of prior training or experience.

CHARLES D. BOUCEK, M.D.
Staff Anesthesiologist, Montefiore Hospital
Assistant Professor of Anesthesiology

I. L. TYLER, M.D., PH.D.
Staff Anesthesiologist, Magee–Women's Hospital
Assistant Professor of Anesthesiology

SOFRONIO B. DE LA VEGA, M.D.
Associate Director of OB Anesthesiology
Assistant Professor of Anesthesiology

RAY MCKENZIE, M.D.
Chief of Anesthesiology
Magee-Women's Hospital
Professor of Anesthesiology

Received from the Department of Anesthesiology
Magee–Womens Hospital
University of Pittsburgh
School of Medicine
Pittsburgh, Pennsylvania 15213

(Accepted for publication April 26, 1984.)

Anesthesiology
61:479–480, 1984

Complex Effects of Isoflurane on Baroreceptor Reflex Compounded by Errors

To the Editor:—The article by Seagard *et al.*[1] on the effect of isoflurane on the baroreceptor reflex addresses an important physiologic mechanism. Although the methods are well designed and suggest sophisticated technical accomplishments, the data are presented poorly, with errors and omissions, making subsequent conclusions of dubious value.

The authors state that thiopental anesthesia significantly depressed the depressor but not the pressor response of the baroreceptor reflex. The data in table 1 show just the opposite, namely a significant reduction in bradycardic response–pressure slope to increasing blood pressure from 59.4 ± 16.5 ms/mmHg in conscious dogs to 23.4 ± 12.8 ms/mmHg in thiopental dogs. In their discussion, the authors reason that the vagolytic effect of thiopental was responsible for the depression of the depressor response. Any vagolytic effect would attenuate the bradycardic response of increasing pressures, not the depressor response as the authors suggest. Further in the discussion, they note "at 1 MAC (isoflurane) the bradycardic responses to decreases in pressure were not different from control." Surely they mean to suggest that the bradycardic response to *increase* in pressure is unaffected by 1 MAC isoflurane anesthesia as is indicated by the data.

In their results on the carotid sinus nerve recordings, the authors present no numeric data to clarify the confusing scatter of points on the nerve activity–carotid sinus pressure graph in figure 2 (we are referred to table 2 but the data are absent). The best fit lines, if indeed they are accurately drawn, would seem to have very wide 95% confidence limits. To conclude from this that "isoflurane produces a dose dependent increase in baroreceptor activity" is questionable.

In table 3 the legend states preganglionic and postganglionic nerve activity is expressed as per cent baseline level. Stating that sympathetic efferent nerve activity fell to 6.27 ± 3.07% of control in response to decreases in blood pressure must be an error. In an earlier publication,[2] these authors present similar data as a per cent of *change,* in which case the reader needs to know the direction of change.

The infusion rates of nitroprusside and phenylephrine (100–300 mg/min and 10–50 mg/min, respectively) seem lethal. Since we do not know the duration of infusion, however, total dosages are unknown.

Finally, we feel that considerable confusion is caused by the combination of four different methods into one publication. In our opinion, it would be more appropriate to present each as a separate publication so the reader can better analyze the data and conclusions presented.