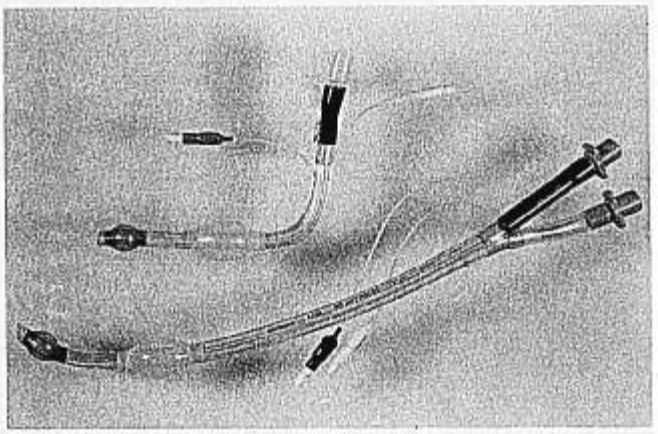

FIG. 1. Modified left-sided polyvinyl chloride (PVC) tracheostomy double-lumen endobronchial tube (DLT) next to a standard left-sided PVC DLT (Broncho-Trach, Sheridan, Argyle, NY).

to prevent kinking and subsequent lumenal obstruction.[4] Fixation of the tube to prevent movement and displacement may also be a problem with the standard DLT. These problems are greatly magnified if the tube is required for extended periods, as may occur when selective split-lung ventilation in the intensive care unit is indicated.[5]

Therefore, we designed and the manufacturer modified to our specifications a left-sided 41F Broncho-Trach DLT (Sheridan, Argyle, NY), which we have used successfully in patients with tracheostomies (Figure 1).

The distance between the distal tip of the endobronchial lumen and the bifurcation of the tracheal and endobronchial lumens is shortened to 18.5 cm from 32.0 cm to reflect the markedly reduced length of the upper airway. The proximal length of both lumens after they bifurcate are also shortened, to 3.0 cm from 7.5 cm, to reduce the chance of kinking. A 90° bend is placed approximately 2.5 cm proximal to the proximal edge of the tracheal cuff to allow the tube to exit from the neck at a less awkward angle. The tubings to the pilot balloons are shortened to 11.0 cm from 23.0 cm for convenience. In all other aspects the tube is identical to a standard 41F Sheridan PVC DLT.

A plastic DLT intended for tracheostomies has been described previously.[6] It consists of two conventional endotracheal tubes (6-mm ID, 8-mm OD) solvent-welded together side by side. In contrast to modern DLTs, this tube has low-volume/high-pressure cuffs. Our tube offers all the advantages of a standard PVC DLT and in addition has been shortened and its body further modified with a bend to more closely conform to the altered anatomy of the upper airway and neck in patients with tracheostomies. A 41F DLT (13.7-mm OD) is just slightly larger than a #10 Shiley (Shiley Inc., Irvine, CA.) single-lumen tracheostomy (39F, 13.0-mm OD) tube and can be used in most adult patients with tracheostomies. If necessary, other (37F and 39F) PVC DLTs may be similarly modified for smaller patients.

JAY B. BRODSKY, M.D.
Professor (Clinical), Department of Anesthesia

H. GARETH TOBLER, M.D.
Chief Resident, Department of Cardiovascular Surgery

JAMES B. D. MARK, M.D.
Professor, Department of Surgery
Head, Division of Thoracic Surgery

Stanford University School of Medicine
Stanford, California 94305

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(Accepted for publication October 30, 1990.)

Anesthesiology
74:388–389, 1991

Fentanyl and Sufentanil Anesthesia Revisited: Establish an Effective Plasma Concentration and Achieve It at the Right Time

To the Editor:—In our opinion, the study entitled "Fentanyl and Sufentanil Anesthesia Revisited: How Much Is Enough?"[1] and the accompanying editorial[2] miss the mark on certain issues. The study attempts to address the question of whether there is dose-related suppression of hemodynamic and hormonal responses to surgical stimulation and uses two protocols. However, only in protocol II were plasma opioid (sufentanil) concentrations measured.

The technique used in this protocol resulted in the highest plasma sufentanil concentrations (means ranging from 23 to 54 ng/ml) at the time of the least painful stimulation (during induction) and the lowest plasma sufentanil concentrations at possibly the times of greatest stimulation (after sternotomy and cannulation). The magnitude of the decreases in plasma sufentanil between the end of the loading dose and intubation (ranging between 57 and 69%) indicates that the authors must have delayed tracheal intubation for some time after induction of anesthesia.

If the object of a study is to demonstrate the effectiveness of sufentanil at blocking hemodynamic and hormonal stress responses, laryngoscopy and intubation would best be accomplished at the time that plasma sufentanil concentrations are at or closest to their highest value (*i.e.*, immediately after the loading dose was administered). Furthermore, subsequent infusion rates of sufentanil should be calculated so that plasma concentrations do not decrease by 80% or more by sternotomy, and additional boluses of sufentanil should be administered just prior to sternotomy or cannulation, in anticipation of these more stressful events. The study design of Philbin and co-workers did none of the

above and therefore seems to us predestined to demonstrate that opioids as sole anesthetics are ineffective in blocking hemodynamic and hormonal stress responses.

Despite a "less than ideal anesthetic technique" for blocking stress responses to stimulation, Philbin *et al.*[1] may still have shown that higher plasma concentrations of sufentanil are more effective than lower concentrations at preventing hemodynamic responses to stressful events. If hemodynamic responders with plasma concentrations of sufentanil greater than 10 ng/ml are compared to responders with plasma concentrations of less than 10 ng/ml for all three events (postintubation, poststernotomy, and postcannulation) (fig. 3 in the article by Philbin *et al.*) only 14.3% (6 of 42) of the responders had plasma sufentanil concentrations above 10 ng/ml, *versus* 35.2% (19 of 54) with plasma concentrations below 10 ng/ml. This difference is statistically significant; according to Fisher's Exact test, $P = 0.034$.

The issue addressed by Hug[2] is whether high doses of μ-opioid agonists, because of their limited ability to block autonomic and endocrine responses during surgery, can ever be considered more than incomplete or partial anesthetics. Clearly, opioids seem to produce their anesthetic effects *via* different mechanisms than those causing anesthesia with the classical inhalation and even other intravenous agents. What Philbin and co-workers and Hug have not emphasized is that subtleties in anesthetic technique, including timing of opioid administration, are more important with opioids than with other anesthetics in explaining differences in measured outcomes.

Dr. Hug is incorrect in stating that highly potent opioids such as fentanyl and sufentanil are incapable of preventing movement in all subjects, whether patients or dogs. Bailey *et al*[3] have shown that 100% of unparalyzed dogs receiving 3,000 μg/kg fentanyl did not move in response to a supramaximal tail-clamp stimulus of 30 s. Some readers may suggest that 3,000 μg/kg is an excessively high dose of fentanyl. Undoubtedly, it is excessive in humans, but it is the only dose that Bailey *et al*[3] found that would reliably produce a plasma fentanyl concentration above 500 ng/ml 5 min after injection in the dog. If plasma fentanyl concentrations were above 500 ng/ml, all dogs in Bailey's experiments did not move regardless of the stimulus. If plasma fentanyl concentrations were less than 500 ng/ml, the incidence of movement increased as the plasma concentration decreased.

Published and unpublished data obtained from studies in a variety of animal species have demonstrated that, although within any species individual variability may be extremely large, there is a plasma concentration of all potent μ-receptor agonists that blocks movement in all individuals.[3-7,*,†] The plasma concentration of sufentanil that blocks movement in response to a painful stimulus in "all dogs" has not yet been determined. Likewise, the plasma concentration of fentanyl or sufentanil that blocks undesirable responses (including awareness) to painful stimuli in all human patients also has not been determined. Furthermore, whether there are advantages in attempting to achieve anesthesia with only an extremely high dose of an opioid (and achieving high plasma and CNS concentrations), *versus* using an opioid plus one or more supplements, has not been carefully studied and is unknown.

The upshot of all of this is that while Philbin *et al.*[1] and Hug's[2] contention that suppression of hemodynamic and hormonal stress responses is not related to opioid plasma concentration may be correct, we believe the study has not adequately tested this hypothesis. Unfortunately, despite its popularity in anesthesia, the subtleties of opioid anesthetic actions are still not well known, and therefore, clinical anesthetic techniques with opioids probably are still quite primitive.

THEODORE H. STANLEY, M.D.
Professor of Anesthesiology

PETER L. BAILEY, M.D.
Assistant Professor of Anesthesiology
University of Utah School of Medicine
50 North Medical Drive
Salt Lake City, Utah 84132

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(Accepted for publication November 2, 1990.)

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Anesthesiology
74:389–390, 1991

In Reply:—The critical issue underlying our study is the practical limits of the dose–response relationship. Stanley and Bailey assume that higher opioid blood levels, and therefore, timing of periodic doses in anticipation of stimulation, will abolish responses. This also assumes that there is a continuous dose–response relationship that does not plateau. Our data clearly demonstrate that within the dose range tested, such a plateau exists. We could discern no difference in response between patients receiving 50 μg/kg fentanyl and those receiving 40 μg/kg sufentanil plus infusion. Since this latter dose is already well beyond that recommended for clinical use, we have no evidence that changes in or timing of doses predictably decrease response. We stand by our statement that it is unlikely that any *clinically useful dose* will successfully abolish responses in *all patients.*

In addition, Stanley and Bailey have misinterpreted our data in figure