to the left brachial artery. Preoperative evaluation was significant for a heart rate of 105 and a hematocrit of 25%. The patient was brought to the operating room where an electrocardiogram, a pulse oximeter, and a blood pressure cuff were applied. The chest tube was attached to continuous wall suction. After preoxygenation, a rapid-sequence induction was performed using 375 mg intravenous thiopental, 140 mg succinylcholine, and 150 μg fentanyl. The trachea was easily intubated with a size 8 cuffed endotracheal tube. The position of the tube was confirmed by bilateral equal breath sounds and by presence of end-tidal CO_2 as noted by capnometer.

Anesthesia was maintained by 1–2% enflurane in an air/oxygen mixture, as well as intravenous fentanyl and vecuronium. The lungs were ventilated *via* a semiclosed circle anesthesia breathing circuit using an Ohmeda® 7700 ventilator. The inspiratory-to-expiratory (I:E) ratio was set at 1:2. The fresh gas flows were set as follows: air, 1 l/min, and O_2, 1 l/min. Once the operation had begun, the capnograph was noted to have the shape seen in figure 1. Of note was the I:E ratio of 1.6:1. A leak was therefore suspected distal to the CO_2 sampling site. Partial deflation of the endotracheal tube balloon lead to an audible leak, which disappeared on full inflation. This confirmed the balloon to be intact. Further search for the leak was unrevealing. By chance, when the chest tube suction was disconnected for a short time, the capnograph was noted to return to normal (figure 2).

The patient's brachial artery was repaired with an autologous vein graft. The remainder of the anesthetic was uneventful. Neuromuscular blockade was antagonized, the trachea was extubated and the patient made an uneventful recovery.

The capnograph consists of four phases; the inspiratory baseline, the expiratory upstroke, the expiratory plateau and the inspiratory downstroke.* The expiratory plateau is flat only when a circle system with intact valves is utilized. When a Mapleson D or other continuous flow circuit is used, exhaled gas is continuously washed out. In the case described, the patient had a continuous leak of air into his left hemithorax caused by the chest tube suction. Had this leak been large, a failure of the ventilator bellows to return to the inspiratory position would have been seen. As this did not occur, we speculate that the leak was less than the fresh gas flow. During exhalation, a fraction of the patient's exhaled gas exited *via* the chest tube rather than the endotracheal tube. During the expiratory pause, the continued flow through the chest tube resulting from the suction led to the flow through the endotracheal tube being inward rather than outward. Because gas flowing past the end-tidal CO_2 monitoring device contained

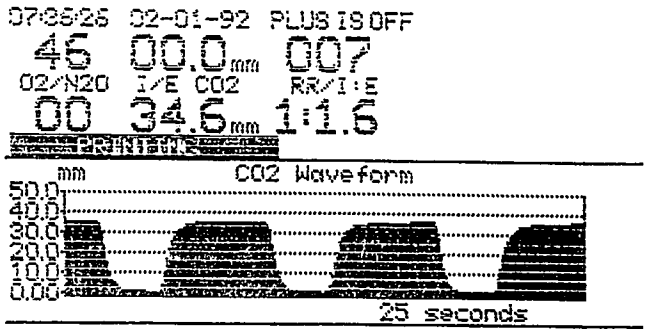

FIG. 2. Capnograph after chest tube suction disconnected.

no CO_2, the device incorrectly interpreted this as the beginning of the inspiratory phase, thereby yielding an abnormal value for the I:E ratio. When suction to the chest tube was disconnected, the air leak decreased, there was no inward flow of gases through the endotracheal tube during the expiratory pause, and the capnograph returned to normal. At the beginning of the case, only the presence of CO_2 was sought from the capnograph. Only later when the operation had begun was the capnograph noted to be abnormal.

This case demonstrates another use of the capnograph. It displayed a characteristic abnormal I:E ratio when a continuous leak of gas from the breathing circuit occurred. An important lesson to be drawn is that the shape and I:E ratio as well as the presence of CO_2 on the capnograph should be noted after intubation.

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Electroencephalographic Effects of Sedative Hypnotics

To the Editor:—We read with interest the recent report by Drummond *et al.*[1] We have conducted volunteer studies examining the electroencephalographic (EEG) effects of midazolam, thiopental, and propofol and their relation to cognitive effects in sedative doses. All three agents appear to produce similar cognitive[2] and EEG effects. All three agents produce increases in high-frequency EEG beta (13–30 Hz) activity, which, by and large, are statistically identical at these dose ranges. All subjects in these experiments were sedated but able to perform moderately complex cognitive tasks (*e.g.*, memorizing word lists). Propofol clearly causes high-frequency EEG beta activity during sedation, and the effect appears to be related to the serum concentration.[3–5] The phenomenon is not a transient effect, and beta activity appears to be maintained as long as the serum concentration remains constant.

In a separate report, Kalkman *et al.* show that the electrophysiologic effects of propofol appear to be prolonged in relation to the clinical sedative effects of the drug.[6] We have noted a similar discrepancy with midazolam at sedative concentrations on long latency evoked potentials (P300).[7] Preliminary analysis of our data during propofol sedation show a similar effect, with depression of P300 amplitude lasting more than 2.5 hr after the termination of the infusion.

With the introduction of new surgical technology, more anesthetic practice will be targeted toward the production of a satisfactory state

of sedation, and usually amnesia. The apparent dissociation of electrophysiologic effects from clinical signs is an interesting observation that deserves more thorough study. Further investigation into the effects of anesthetics at sedative concentrations should be encouraged.

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