TABLE 1. Sample Program

```
10: "D" print "dopamine: 200 mg/500 ml D5W"
20: Print "Dose=2-25 μg/kg/min": GOSUB 400
30: X=Q/400:GOSUB 500
40: End
400: Input "Lbs=";L,"Dose=";D
405: Q=(L/2.2)*D:Return
500: Y=X*60
505: Print using "###.##"; X; "ML OR"; Using "####"; Y;
     "MGT/MIN": Return
```

recommended dilution and dose range, and prompts for the patient's weight in pounds and the desired rate of administration. In a few microseconds, the rate is given in both milliliters and microdrops. While not as elegant as some of the programs available on larger computers, it is eminently useful, and economical as to both price and space.

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Ketamine Treatment of Status Epilepticus

To the Editor:—Recent publications regarding ketamine fail to deal with an old controversy regarding its use; namely, its convulsant/anticonvulsant properties.[1,2]

We recently were asked to consult regarding three patients in status epilepticus. Conventional therapy had failed in these patients and the use of a general anesthetic[3] presented too great a risk due to both severe hypotension and dysrhythmias. We noted an apparent temporal relationship between the cessation of both physical and electroencephalographic evidence of seizure activity and the intravenous administration of ketamine.[4]

Whether this truly represents seizure suppression by ketamine HCl is difficult to say. It is at least a consideration when faced with severely hemodynamically compromised patients in whom status epilepticus has proven refractory to conventional therapy.

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58:203–204, 1983

Inseparable Disposables

To the Editor:—I would like to share with you a recent experience of equipment malfunction and alert your readers to the potentially serious complications which might ensue.

During a recent hospital personnel strike, the black rubber face masks (Trimar-Ohio), normally used in this department, were replaced by single-use plastic face masks (Vital Signs—Product #5250). After induction of anesthesia, a patient was preoxygenated, paralyzed, and intubated.

When I planned to connect the patient to the anesthesia circuit, it quickly became evident that the mask

could not be separated from the single-use anesthesia circuit (Becton-Dickinson #3063). Repeated attempts failed to allow separation or even rotation of the mask around the patient-end of the circuit. With a final forceful attempt the circuit broke at the level of the swivel adapter (fig. 1) making any form of ventilation impossible. A replacement circuit was readily available and the patient suffered no harm. Using identical equipment, it was subsequently quite easy to reproduce a similar tight bond.

This incident suggests caution in the concomitant use of disposable equipment from different manufacturers and reemphasizes the need for a resuscitation bag in every operating room.

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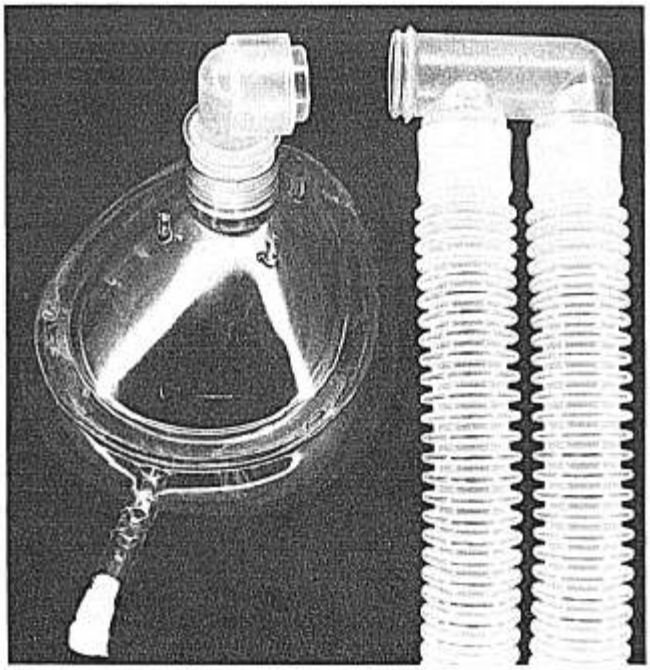

FIG. 1. Broken anesthesia circuit with tightly attached mask.

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Moyamoya Disease and Anesthesia

To the Editor:—Moyamoya (puff of tobacco smoke) disease is a rare, chronic, occlusive cerebrovascular disease with an unusual network of vessels in the base of the brain.[1] The clinical manifestations are variable and include hemiplegia, convulsive seizures, and speech disturbances.[2] In the last few years, this disease has been the object of surgical treatment, including superficial temporal-middle cerebral artery anastomosis[2] and encephalomyosynangiosis.[3]

We have anesthetized four patients seven times. They were 6, 10, 32, and 52 years old, and had no abnormal neurologic findings preoperatively. Two patients were anesthetized with neuroleptanesthesia with controlled ventilation. Frequent analysis of arterial blood gases revealed Pa_{CO_2} values ranging from 30 to 35 mmHg. These patients had delayed recovery of consciousness postoperatively, and also obvious neurologic deficits including hemiplegia, amnesia, dyscalculia, and speech disturbance in one case. These complications were severe and lasted for a month or more. On the other hand, the other patients, including the second anesthetics of three patients, were anesthetized with halothane and N_2O with spontaneous ventilation, except in one patient in whom ventilation was controlled. In these cases, an infrared CO_2 analyzer was used to monitor the end-tidal CO_2 that was maintained at 40–50 mmHg. Postoperatively, all of these patients showed prompt recovery of consciousness and no neurologic complications.

It seems unlikely that the surgical procedure itself caused the neurologic deficits, because in these operations, there was nothing we could identify that would cause a decrease in the cerebral blood flow. Furthermore, the direct arterial blood pressures were monitored in each patient and maintained an adequate range. Neuroleptanesthesia itself might not be an exacerbating factor in these two cases because, in humans, this type of anesthesia has not been shown to have a significant influence on either the cerebral blood flow (CBF) or the cerebral metabolic rate for oxygen.[4]

Hypocapnia may be the main cause of the neurologic deficits. The degree of hypocapnia in our patients should produce about a 30% decrease in the CBF[5] and may have been a critical effect on moyamoya disease.