Although a definite stop, as at the zero position, was felt, the smell of halothane was present in the gas coming from the common outlet. Initially, the oxygen flush was used to dilute the in-circle halothane concentration. However, since another machine was readily available, it was used for the balance of the procedure.

Discussion

The hazard in this situation is twofold. First, delivery of 1.2 per cent halothane in some situations will provide a concentration too high to be tolerated by the patient. Second, unless it is noticed that the firm stop to dial rotation did not actually occur at the zero point, the anesthetist may fail to realize that the vaporizer is still delivering clinical levels of anesthetic vapor. Cook *et al.* have recently reported that trace concentrations of halothane may continue to be delivered even though the vaporizer is nominally off.[2]

In our case failure occurred because the plastic dial detent had worn down to the point where it escaped from its correct position (Fig. 1). The detent normally provides the clicking sensation at each labeled position of the concentration selector dial by pushing a small plastic dowel against each notch in the lower rim of the dial as it is rotated. Since the worn detent was spring-loaded, the assembly was then pushed against the underside of the concentration selector dial. There is a control arm fixed to the underside of the dial that normally engages the pin of a rotating drum outlet valve and closes the valve as "off" is approached. In rotating the dial counterclockwise to shut off the vaporizer, this arm impinged on the detent assembly, preventing the dial from rotating below the 1.2 per cent halothane position (Fig. 2).

Since the vaporizer was permanently fastened to a Foretrend 300 machine in the flow path between the oxygen flowmeter and common outlet, a method of bypassing the vaporizer is necessary in the event another machine is unavailable. Activation of the oxygen flush provides oxygen while cutting off flow to the flowmeters and therefore through the vaporizer. Intermittent use to fill the breathing circuit with the flowmeter needle valves closed will also prevent continued vaporization. Intermittent oxygen flush with flowmeters on will dilute the in-circle anesthetic concentration. Emptying the vaporizer is not an adequate acute remedy, since a saturated Fluomatic wick was found to contain about 60 ml liquid halothane, which would be vaporized after apparent emptying.

The key to prevention of untoward sequelae is to understand machine function sufficiently well to deal with a hazard of this type. From a broader viewpoint, prevention of such a hazard may be accomplished by careful original design and regular preventive maintenance.

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Prolonged Apnea Following Trimethaphan and Succinylcholine

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Plasma cholinesterase activity declines during pregnancy, reaches a nadir on the third postpartum day, and returns to normal during the following six weeks.[1] Some investigators have cautioned that low cholinesterase activity may result in prolonged apnea following succinylcholine administration.[2–5] Recently, Blitt *et al.*[6] found no correlation ($r < 0.5$) between serum cholinesterase activity (0.81 to 1.03 units/ml) and duration of paralysis following succinylcholine. They concluded that single-dose administration of succinylcholine (40–80 mg/m^2) should not be associated with prolonged apnea in parturients. We report the case of a parturient pretreated with a ganglionic blocking drug who experienced prolonged apnea following succinylcholine.

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The patient described in this case report was cared for by Drs. Poulton and James at The Bowman Gray School of Medicine. Dr. Lockridge performed serum cholinesterase determinations at the University of Michigan.

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Report of a Case

A 26-year-old primigravida was referred for treatment of severe pre-eclampsia at 33 weeks' gestation. She had no history of chronic drug therapy or previous anesthesia. Pregnancy had been uncomplicated until ten days prior to admission, when severe pre-eclampsia had developed. Initial treatment had consisted of rest in bed, sodium restriction, and, for 48 hours prior to transfer to this institution, iv administration of magnesium sulfate.

On admission the patient was alert and oriented, with hyperreflexia, edema, and blood pressure 170/120 torr. Complete blood count and serum electrolyte, calcium, and magnesium levels were normal. Magnesium sulfate was discontinued and a regimen of parenteral hydralazine and alpha-methyldopa was begun. Blood pressure proved difficult to control, climbing to 180/120 torr on the seventh hospital day. Non-stress fetal monitoring disclosed diminished beat-to-beat variability and occasional late decelerations; the lecithin:sphingomyelin ratio was 1.6:1. Labor was induced with oxytocin, iv, and a continuous infusion of trimethaphan camsylate (Arfonad®) was started. Labor began promptly, and over the next two hours, during which the patient received 1,700 mg trimethaphan, blood pressure dropped to 160/100. At that time persistent late fetal decelerations necessitated urgent cesarean section.

Three minutes after iv administration of 3 mg *d*-tubocurarine, rapid-sequence induction–intubation was accomplished with 200 mg (3 mg/kg) thiopental and 140 mg (2 mg/kg, 80 mg/m^2) succinylcholine. The 1,850-gram infant had 1- and 5-min Apgar scores of 7 and 10. Anesthesia was maintained with nitrous oxide (3.5 l/min) and oxygen (1.5 l/min) and, following delivery of the infant, 0.10 mg fentanyl, iv. The patient remained fully relaxed and apneic throughout the 45-min procedure.

At the end of the operation, peripheral-nerve stimulation demonstrated a profound depolarization type of neuromuscular blockade. Arterial blood gases were normal. The patient was transferred to the intensive care unit, where mechanical ventilation was continued. Two hours later she began to make weak respiratory efforts; peripheral-nerve stimulation confirmed the continued presence of a depolarization type of neuromuscular blockade. Six hours after the induction of anesthesia, the patient regained sufficient strength to permit extubation of the trachea.

Plasma cholinesterase evaluation on the second postpartum day revealed qualitatively normal pseudocholinesterase with decreased activity (table 1). Six weeks post partum the cholinesterase activity had returned to normal.

Table 1. Serum Cholinesterase Activity and Phenotype of the Patient

	Cholinesterase Activity* (units/ml)	Dibucaine Number†	Fluoride Number‡
Postpartum values			
Two days	0.60	80	59
Six weeks	0.80	82	55
Normal	0.8 to 1.1	80	60

* Determined by the method of Kalow W, Lindsay HA: A comparison of optical and manometric methods for the assay of human serum cholinesterase. Can J Biochem Physiol 33:568–574, 1955.

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Discussion

Although prolonged apnea following the administration of succinylcholine to parturients has been reported previously, postpartum cholinesterase activity was in every instance much lower, and duration of apnea much shorter, than in our patient.[1,2,4,5] Furthermore, since none of the parturients in the study of Blitt *et al.* (all of whom had cholinesterase activities similar to that of our patient) experienced prolonged apnea after the same dose (80 mg/m^2) of succinylcholine, we doubt that decreased cholinesterase activity alone accounted for our patient's paralysis.

Trimethaphan is a ganglionic blocking drug used in a variety of clinical situations. Direct interference with neuromuscular transmission by trimethaphan has been documented in animals[7–9] and in man.[10,11] This dose-dependent effect produces nondepolarizing neuromuscular blockade. A possible prolongation of succinylcholine-induced apnea in persons pretreated with trimethaphan was suggested in 1957 by Tewfik.[12] More recently, Sklar and Lanks[13] demonstrated trimethaphan to be a potent noncompetitive inhibitor of plasma cholinesterase *in vitro*. Sodium nitroprusside had no effect on cholinesterase activity.

In summary, we believe this represents a case of cholinesterase inhibition *in vivo* by trimethaphan. This association needs to be confirmed and more widely recognized. The recommendation of Sklar and Lanks that nitroprusside rather than trimethaphan be used as a rapidly-acting hypotensive agent in patients who may receive succinylcholine deserves consideration. Since prolonged use of nitroprusside in hypertensive parturients may lead to fetal cyanide toxicity,[14] we suggest that alternative drugs such as nitroglycerine be considered for parenteral treatment of severe hypertension in pregnant patients who may need succinylcholine as part of their anesthetic management.

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Prevention of Anaphylaxis to Contrast Medium

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Anaphylactic reactions to the injection of contrast medium are rare, but potentially catastrophic. This is a report of the prevention of an anaphylactic reaction in a patient who had had documented anaphylaxis in response to contrast medium only eight hours earlier.

REPORT OF A CASE

A 50-year-old white woman who had a history of progressive angina was admitted for diagnostic cardiac catheterization. She specifically denied allergies to iodine and iodine-containing products. After premedication with meperidine, 75 mg, and promethazine, 25 mg, im, a left ventriculogram was performed through a catheter inserted via the right femoral artery. A pressure-injected 45-ml bolus of meglumine diatrizoate was given, after which the patient complained of a "cold, clammy feeling," and a diffuse maculopapular rash developed. The patient was treated with diphenhydramine, 50 mg, and methylprednisolone, 100 mg, iv, which relieved her symptoms. Catheterization of the coronary arteries was then performed, and 10 ml contrast medium injected. The patient immediately became dyspneic, and wheezing was heard over the lung fields. Arterial blood pressure was 40/0 torr. The patient was placed in Trendelenburg position and iv fluids were given rapidly. Oxygen was administered by face mask, and 10 ml epinephrine (1:10,000) were administered iv. Blood pressure improved and the wheezing subsided. The study was terminated.

Eight hours after the episode of anaphylaxis, the patient's right leg was discovered to be cold and pulseless. The decision was made to perform a femoral-artery thrombectomy with local anesthesia. Because of difficulty in locating the thrombus, angiography was necessary. Methylprednisolone, 1 g, iv, was given 30 min, and diphenhydramine, 25 mg, 5 min prior to injection of the contrast medium. Meglumine diatrizoate, 30 ml, was injected into the femoral artery, with no change in arterial blood pressure or other vital signs. The patient's chest remained clear to auscultation and no rash appeared. Two subsequent injections of 50 ml of each contrast medium were preceded by diphenhydramine 25 mg, iv, and likewise resulted in no evidence of a reaction. The patient was discharged from the hospital on the fourth postoperative day.

DISCUSSION

Anaphylaxis to contrast medium was first attributed by Mann[1] to histamine release. Subsequent work has shown that additional chemical mediators, such as serotonin, plasma kinins (kallidin I, kallidin II) and slow-reacting substance (SRS-A) play major roles in this reaction.[2] Manifestations of anaphylaxis characteristically may include urticaria, bronchospasm, laryngeal edema, hypotension, nausea, vomiting, diarrhea, and motor convulsions.[3,4] The treatment of anaphylaxis includes administration of epinephrine, aminophylline, fluids, corticosteroids and vasopressors, when necessary. Upper-airway obstruction from laryngeal edema may necessitate tracheal intubation. Pathologic studies by James and Austen[5] showed that upper airway edema and obstruction were the predominant abnormalities in fatal cases of anaphylaxis.

Until recently, this reaction was not reproducible in an animal model as a response to a specific contrast medium,[6] although it was well known as a clinical entity. Although the reaction to contrast medium has not been shown to be IgE-mediated[7] and no antibody has been identified, histamine is released.[8,9] Pretreatment with antihistamines will prevent some of the symptoms of anaphylaxis, but antihistamines

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The opinions or assertions expressed herein are those of the author and are not to be construed as official or as reflecting the views of the Navy Department or the Naval Service at large.

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