at 37 C is 105–120 min,[12] it is conceivable that the drug could have reached the endothelial cells, which were already compromised, and produced temporary changes that resulted in acute failure to maintain normal corneal hydration, with resultant onset of edema.

Although a single case fails to provide conclusive evidence, the available data suggest that acute corneal edema in our patient probably resulted from cytotoxic effects of melphalan, although the condition, once initiated, could have been exacerbated by anesthesia-related factors and prolonged closure of the eyes.

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A Coagulation Disorder during Reanastomosis of Small-intestinal Bypass

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Jejunoileal bypass is frequently employed for the treatment of massive exogenous obesity. Unfortunately, some intestinal bypass patients experience persistent severe diarrhea with electrolyte disturbances. Other serious complications also occur, namely, renal calculi, nephrocalcinosis, cholelithiasis, gastric hyperacidity, peripheral neuropathy, pulmonary embolism, vitamin deficiencies, hepatic steatosis, cirrhosis, and in some cases, hepatic failure.[1]

Reanastomosis of the small bowel to restore its integrity may become necessary in patients with severe hepatic dysfunction. This report emphasizes a potentially lethal coagulopathy that was encountered in such a patient.

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TABLE 1. Results of Laboratory Studies Performed during Admission for Reanastomosis of the Small Intestine

	Normal Range	Before Reanastomosis		After Reanastomosis		After Repair of Cecal Ulcer	
		Admission	Preop.	Day 1	Day 5	Day 1	Day 5
Blood count							
Hematocrit (per cent)	37 –47	34	30	37	31	26	34
Leukocyte count (thousands/cu mm)	4.8–10.8	3.1	4.2	14.9	14.6	27.3	13.2
Electrolytes (mEq/l)							
K	3.5– 4.5	2.5	3.5	2.7	4.2	5.2	4.5
CO_2	24 –32	20	19	23	28	15	22
Renal function variables							
BUN (mg/100 ml)	10 –20	8	10	6	10	46	97
Creatinine (mg/100 ml)	0.6– 1.1	.8	.8	.8	.8	5.2	4.6
Hepatic function variables							
Total bilirubin (mg/100 ml)	.25– .9	2.4	1.9	3.3	5.6	9.9	18.5 (D)
Total protein (g/100 ml)	6.5 –8.5	6.2	5.5	5.6	5.4	4.5	4.9
Albumin (g/100 ml)	3.5 –5.0	3.0	2.7	2.8	2.2	1.9	1.8
Hepatic enzymes							
SGOT (mu/ml)	5– 35	116	100	143	66	390 (D)	670 (D)
LDH (mu/ml)	90–210	268	236	600	289	449	845 (D)
Alkaline phosphatase (mu/ml)	25– 75	106	111	95	101	63	144
Coagulation studies							
Platelet count (thousands/cu mm)	150–400	95	80	156	264	550	135
Bleeding time (min)	To 7.5	10.3	10.5	—	—	—	—
Protime (per cent of control)	70–100	33	38	44	35	21	11
Partial thromboplastin time (sec)	20– 38	47	42.8	31.7	37.2	47.2	56.5
Thromboplastin time (sec) (5 units)		—	11.7	—	—	—	—
Fibrinogen (mg/100 ml)	200–400	—	95	355	195	—	—
Fibrin split products (micro gm/ml)	To 4.0	—	4	4	—	—	—
Factors (per cent of normal)							
II		—	45				
V		—	55				
VII–X		—	32				

(D) = diluted.

Report of a Case

A 36-year-old woman (weight 79.6 kg, height 162.6 cm) was admitted for routine follow-up seven months after jejunoileal bypass for morbid exogenous obesity. Her weight loss during seven months had been 75 pounds; she also complained of persistent diarrhea (12 times per day), intermittent nausea and vomiting, and generalized weakness. There was no history of exposure to hepatitis, hepatotoxic drugs, chemical agents, or excessive ethanol consumption. Examination of a biopsy specimen taken from the liver during the original bypass procedure revealed significant fibrosis, nodule formation, and fatty infiltration.

Vital signs on admission were within normal limits. Physical examination disclosed no abnormality except a systolic ejection murmur along the left sternal border, spleen palpable 7 cm below the left costal margin, and an area of ecchymosis around a venipuncture site. All laboratory tests were noncontributory except those listed in table 1.

The primary preoperative problem was severe hepatic dysfunction with marked abnormality of coagulation factors. Intravenous hyperalimentation was considered, but it was elected to proceed with reanastomosis of the small intestine and splenectomy. Preoperative preparation included 3 units of fresh frozen plasma, iv, the evening before operation. Platelets (10 units) and adequate fresh frozen plasma were available for intraoperative use. The identified fluid and electrolyte abnormalities were corrected.

Premedication was with Innovar, 2 ml, and diphenhydramine, 50 mg, im, 65 minutes prior to induction of anesthesia. Induction was accomplished with thiopental (2 mg/kg), diazepam (0.15 mg/kg), and fentanyl (0.002 mg/kg). Anesthesia was maintained with N_2O:O_2 4l/2l. Endotracheal intubation and subsequent muscle relaxation were achieved with pancuronium bromide, while ventilation was mechanically controlled with a tidal volume of 10 ml/kg at a respiratory rate of 8–10/min.

Immediately upon making an upper midline operative incision, excessive oozing was noted. This bleeding became uncontrollable as the peritoneum was exposed. Four units of fresh frozen plasma and three units of packed erythrocytes were rapidly infused. The bleeding continued and surgical hemostasis was unsuccessful. A splenectomy was performed as expeditiously as possible. Following removal of the spleen, eight platelet units were promptly administered. The oozing subsided, allowing the operation to proceed. Subsequently, reanastomosis of the small bowel and a liver biopsy were performed. Blood loss within the 10–15 minutes prior to splenectomy was measured at 1,500 ml, while total loss during the ensuing operative procedure (2.5 hours) was 500 ml.

Immediate postanesthetic recovery was uneventful and unusual bleeding was not encountered. Maintenance of the prothrombin time at greater than 40 per cent of control value by administration of fresh frozen plasma was attempted. However, progressive hepatic and renal failure supervened. On the fifteenth day after intestinal reanastomosis, free air under the diaphragm, peritoneal localizing signs, fever, and sepsis necessitated re-exploration. A perforated ulcer of the cecum was found and closed. The postoperative course was downhill, and death occurred four days later.

Discussion

With the advent of operative management of the markedly obese patient, the anesthesiologist can anticipate that some of these individuals will need further unrelated operative procedures. Although many potential complications may be encountered, certainly the most serious is severe hepatic dysfunction. Small-intestinal bypass typically leads to striking hepatic fat accumulation, and occasionally to frank cirrhosis.[3] Moxley has demonstrated that shortly after bypass when weight loss is rapid, hepatic dysfunction and marked steatosis uniformly occur, in association with protein malnutrition. Scott *et al.*[5] reported improvement in hepatic function and morphology as weight stabilized (approximately 18 months postoperatively).

Our patient's severe hepatic dysfunction was apparent from laboratory findings. However, the relative disparity between the coagulation factor (II, V, VII, IX, X, and fibrinogen) deficiencies and the results of other liver enzyme tests (SGOT), alkaline phosphatase, and SGPT) was unexplained. The presence of cirrhosis with portal hypertension resulting in splenomegaly would explain the observed leukopenia and thrombocytopenia.

This case demonstrates that caution should be exercised when anticipating the anesthetic care of patients who have been treated by jejunoileal bypass. Careful assessment of electrolytes, acid–base status, fluid replacement, renal function, and specific liver profiles seems advisable. One could also argue against the use of halogenated anesthetics in these cases, where hepatic dysfunction is likely to develop. Adequate preoperative communication with the blood bank may well prove life-saving when dealing with such a potential coagulopathy.

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Unsuspected Pheochromocytoma in a Surgical Patient

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Hypertensive patients have been shown to have an increased incidence of cardiac arrest during operation.[1] Additional risks may be involved, depending on the cause of the hypertension. If it is due to pheochromocytoma, there may be a fatal hypertensive crisis, arrhythmia, cerebrovascular accident, or acute left heart failure and pulmonary edema without preoperative management.[2] These potential complications and the fact that pheochromocytoma is a correctable cause of hypertension are important reasons to exclude the presence of this tumor in hypertensive patients about to undergo anesthesia. The following case illustrates problems that may occur when this is not done.

REPORT OF A CASE

A 70-year-old man, seen in January 1967 complaining of hoarseness of 4 months' duration, had erosion of the right vocal cord posteriorly and lesions along the left vocal cord. Malignancy was suspected, and laryngeal suspension and biopsy under general anesthesia was planned. The preanesthetic medical evaluation was essentially negative except for a vague history of recent increase in blood pressure. At examination, the blood pressure was 150/90 mm Hg, pulse rate 74/min, and weight 121 kg. The heart and lungs were normal. Routine laboratory results were within normal limits. No electrocardiogram was obtained.

When the patient was placed on the operating table, the blood pressure was 150/90 mm Hg and pulse rate, 75/min. During preparation for induction of anesthesia, the blood pressure increased to 210/90 mm Hg. Atropine (0.4 mg) was given iv, and anesthesia was induced with thiopental (375 mg, iv). A mixture of halothane, nitrous oxide, and oxygen was given for maintenance anesthesia, with spontaneous ventilation. Shortly after induction, cardiac arrest occurred. An endotracheal tube was inserted immediately, controlled ventilation with 100 per cent oxygen was started, and external cardiac massage was instituted. Within a few seconds, a heartbeat was heard and spontaneous respiration resumed. Nevertheless, two additional cardiac arrests occurred during the next few minutes. Ten defibrillations were necessary before cardiovascular stability was achieved. The surgical procedure was cancelled and the patient was transferred to the intensive care unit (ICU). While there, his blood pressure varied between 270/150 and 160/90 mm Hg but stabilized at about 190/90 mm Hg. No treatment was thought necessary, and the cause of his hypertension or of his cardiac arrest was not determined. He was dismissed from the hospital on the eleventh day after cardiac arrest.

Two weeks later, laryngeal suspension with biopsy was completed under local anesthesia. Examination of the biopsy specimen revealed carcinoma of the right vocal cord. Radiotherapy rendered this disease process inactive, and periodic checks confirmed its inactivity for nearly 7 years.

During this interval, general medical care was provided by the patient's local physician, who prescribed an antihypertensive agent and potassium supplementation in 1973. Four months after the biopsy procedure, the patient had reported having "spells," lasting as long as 2 hours, when he would feel "mean," but these were not evaluated further.

In late 1973, an anterior web and granularity of the cords were noted. Laryngeal suspension with biopsy under general anesthesia was considered necessary. During this preanesthetic medical evaluation, the blood pressure was 190/110 mm Hg, pulse rate 70/min, and weight 112 kg. Examinations

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