brospinal fluid would then approximate twice normal, or 300 ml, if there were no transfer of isotonic fluid out of the subarachnoid space. A volume increase of this magnitude within the bony confines of the skull and spine could lead to a marked increase in intracranial pressure and set the stage for pulmonary edema, as indicated in the experimental work cited above.

Anesthesiologists should be aware that pulmonary edema can occur following intrathecal blocks with hypertonic saline solution as well as in neurosurgical patients with head injuries. Conversations with other anesthesiologists reveal that the "wet lung" syndrome following blocks with hypertonic saline solution has been observed in other hospitals. Blood pressure must be monitored closely and enriched oxygen mixtures given. Should pulmonary edema become manifest, a potent intravenous diuretic such as furosemide is indicated. Treatment of hypertension must be tempered with an awareness that the hypertensive response may not be entirely detrimental and, in fact, it may even be beneficial when it ensures cerebral perfusion in a patient in whom the Cushing reflex is operative.

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Hypoxia, Hyperdynamic Circulation, and the Hazards of General Anesthesia in Patients with Hepatic Cirrhosis

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The association of arterial hypoxemia with hepatic cirrhosis has been known for almost a century.[1] More recently, it has been determined that at least some patients with cirrhosis show evidence of a hyperdynamic state of the systemic circulation, with increased cardiac output, decreased total peripheral resistance, a bounding pulse and, often, widened pulse pressure.[2, 3] The hypoxemia and the circulatory changes probably have a common etiology, namely, shunting in the systemic and pulmonary beds.

Below we describe the cases of two patients with Laennec's cirrhosis. The preoperative status of the first patient did not raise our index of suspicion sufficiently, yet his course

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Received from the University of Pennsylvania School of Medicine, Department of Anesthesia, Philadelphia, Pennsylvania 19104. Supported in part by USPHS Research Grant 5-P01-GM-15430-04 and USPHS Training Grant 5-T01-GM-00215-13.

during anesthesia and operation was obviously that of the type of patient described above. Alerted by our experience with the first patient, we managed the second with two different anesthetic techniques, and we believe we obtained useful information. A plan for preanesthetic identification and anesthetic management of such individuals is outlined.

Report of Two Cases

Patient 1. A 46-year-old Caucasian man had been admitted to the Philadelphia Veterans Administration Hospital repeatedly with a diagnosis of Laennec's cirrhosis and bleeding esophageal varices. Following a month of intensive medical therapy, the patient was scheduled for an elective portacaval shunt. Past history revealed no cardiopulmonary diseases or symptoms. The only previous operations were a left inguinal herniorrhaphy and hemorrhoidectomy performed under spinal anesthesia. Physical examination showed the patient to be thin, with "liver palms" and spider nevi, but without cyanosis, clubbing or edema. The heart sounds were normal without murmurs and the lungs were clear to auscultation. Blood pressure was 130/80 mm Hg and pulse was 80 beats/min and regular. Medications in the preoperative period were hydrochlorothiazide, spironolactone, and vitamins.

Immediately preoperatively hemoglobin was 14 gm/100 ml, hematocrit 42 per cent, prothrombin time 17 sec with a control value of 13 sec, SGOT 100 units/ml, alkaline phosphatase 125 international units/l (normal = 12–40), bilirubin 3 mg/100 ml, BUN 10 mg/100 ml, sodium 140 mEq/l, potassium 4.5 mEq/l, chloride 100 mEq/l. Electrocardiogram and chest x-ray were normal. Arterial blood sampled during breathing of room air had a P_{O_2} of 100 mm Hg, P_{CO_2} 38 mm Hg, *p*H 7.47. Pulmonary function test results were: FEV, 76 per cent, MMEFR 65 per cent, MVV 60 per cent, and vital capacity 95 per cent of predicted normal values.

The patient was premedicated with diazepam, 5 mg, and atropine, 0.4 mg, intramuscularly, an hour before induction of anesthesia with halothane in 60 per cent nitrous oxide and 40 per cent oxygen. After ten minutes, succinylcholine, 100 mg iv, facilitated endotracheal intubation. This led to ventricular tachycardia, recorded by electrocardiogram, which returned immediately to normal sinus rhythm upon treatment with 100 per cent oxygen and lidocaine, 100 mg iv. Anesthesia was maintained with nitrous oxide, 3 l/min, oxygen, 2 l/min, and 1 per cent halothane. At the time of the incision the blood was very dark. Nitrous oxide was discontinued and anesthesia was changed to 2 per cent halothane in oxygen. An arterial blood sample was drawn ten minutes later. $F_{I_{O_2}}$ was 0.98 as confirmed by a Pauling oxygen analyzer; P_{O_2} was 64 mm Hg, P_{CO_2} 33 mm Hg, *p*H 7.50. Mechanical problems were ruled out, and the two sides of the chest were being ventilated equally. Repeated blood samples throughout the procedure found P_{O_2} in the range of 60 to 75 mm Hg despite administration of essentially 100 per cent oxygen.

Portal pressure was found to be 42 cm H_2O (normal 6–10 cm H_2O), and a side-to-side portacaval shunt, which lowered the portal pressure to 24 cm H_2O, was constructed. Blood loss during the eight-hour procedure was 3,000 ml and was replaced with 2,000 ml whole blood, 1,800 ml Ringer's lactate solution, 600 ml 5 per cent dextrose in water, and 25 gm albumin. Urinary output was 600 ml. During the last two hours of the operation, the cardiovascular system became hyperdynamic in association with signs of congestive heart failure. Central venous pressure rose from 15 to 25 cm H_2O, urinary output decreased, and bilateral rales appeared. Treatment with furosemide, 40 mg, and digoxin, 0.5 mg, iv, resulted in clearing of the lungs, return of central venous pressure to 15 cm H_2O, and increased urinary output.

Postoperatively, hypoxemia persisted, with a wide alveolar-to-arterial oxygen gradient and P_{O_2} 50 mm Hg during breathing of 100 per cent oxygen. This was probably due to remaining shunts, postoperative atelectasis, and congestive heart failure. The patient needed assisted ventilation for a week. Positive end-expiratory pressure, 10 cm H_2O, was tried on several occasions, but it caused hypotension each time. Congestive heart failure was markedly alleviated within two days, but gradual improvement of the hyperdynamic state took five more days.

Patient 2. A 48-year-old Caucasian man was admitted to the Hospital of the University of Pennsylvania with a diagnosis of upper gastrointestinal bleeding. Esophagoscopy demonstrated the source of the bleeding to be esophageal varices, and the patient was scheduled for a portacaval shunt. Preoperatively, he was treated with ampicillin, Aquamephyton, and neomycin orally and rectally, and the bleeding was controlled with Pitressin, iv, and a Sengstaken-Blakemore tube. Past history revealed no cardiopulmonary diseases or symptoms. The only previous operation was an appendectomy in 1950, performed under general anesthesia. Physical examination showed the patient to be obese, weighing 210 pounds, with palmar erythema, spider nevi, atrophic testicles, gynecomastia, ascites, and hepatosplenomegaly. There was no cyanosis or clubbing. He had a quiet precordium with normal heart sounds and no murmurs. Blood pressure was 110/71 mm Hg and pulse 90 beats/min. There were dry rales at both lung bases.

Laboratory values immediately preoperatively were: hemoglobin 11 gm/100 ml, hematocrit 35 per cent, prothrombin time 55 per cent, platelet count 63,000, bilirubin 2.4 mg/100 ml, SGOT 39 units/ml, SGPT 42 units/ml, LDH 440 units/ml, alkaline phosphatase 24 international units/l, blood

ammonia 2.6 μg/nitrogen (normal = 0.3–0.8), BUN 17 mg/100 ml, sodium 135 mEq/l, potassium 3.4 mEq/l, chloride 101 mEq/l. Chest x-ray showed bibasilar atelectasis, and an electrocardiogram was normal.

On arrival in the operating room, without premedication, the patient was alert and cooperative. An 18-gauge Argyle catheter was placed in the left radial artery. A large intracath had previously been placed in the left subclavian vein for monitoring of central venous pressure. Arterial blood sampled during breathing of air had a P_{O_2} of 51.2 mm Hg, P_{CO_2} 33.7 mm Hg, pH 7.43 (table 1). The cardiac index was 4.91 l/m/m² as measured by the dye dilution technique using indocyanine green dye, and total peripheral resistance was 630 dynes/sec/cm⁻⁵. At the end of five minutes of preoxygenation, with an oxygen flow of 5 l/min and an imperfect mask fit, arterial P_{O_2} was 105 mm Hg and central mixed venous oxygen saturation 70 per cent.

Anesthesia was induced with halothane in 50 per cent nitrous oxide and 50 per cent oxygen. Fifteen minutes later, an arterial blood sample taken with $F_{IO_2} = 0.5$ had a P_{O_2} of 50 mm Hg. The cardiac index decreased to 4.7 l/m/m² and total peripheral resistance to 570 dynes/sec/cm⁻⁵. The trachea was intubated without difficulty and the electrocardiogram continued to record a normal sinus rhythm. At the time of the incision the blood was dark, and N_2O was discontinued. Fifteen minutes later, with $F_{IO_2} = 0.95$, arterial P_{O_2} was 65 mm Hg, P_{CO_2} 34 mm Hg, pH 7.43. Mechanical problems were ruled out, and the two sides of the chest were evenly ventilated.

Ninety minutes after induction, the cardiac index was 4.95 l/m/m² and total peripheral resistance had decreased to 500 dynes/sec/cm⁻⁵. Halothane was discontinued, and 10 per cent cyclopropane in oxygen was administered in a high-flow semiclosed circle absorption system in order to wash out halothane. Forty-five minutes later, an arterial blood sample had a P_{O_2} of 101 mm Hg. At the same time, total peripheral resistance had risen to 800 dynes/sec/cm⁻⁵ and cardiac index declined to 4.0 l/m/m². After 90 minutes of 10 per cent cyclopropane and oxygen, P_{O_2} was 120 mm Hg. Cyclopropane was then shut off and halothane in oxygen administered. This was followed by a gradual decrease in arterial oxygen tension to 100 mm Hg and total peripheral resistance to 600 dynes/sec/cm⁻⁵, while cardiac index increased to 4.65 l/m/m² over the next hour.

Central venous pressure remained between 15 and 25 cm H_2O, and the patient showed no signs of congestive heart failure. A side-to-side portacaval shunt was established and the procedure was completed uneventfully.

Postoperatively, the patient needed assisted ventilation for two days. In the recovery room, an F_{IO_2} of 0.6 was necessary to maintain P_{O_2} at 60 mm Hg. By the second postoperative day, P_{O_2} was 87 mm Hg with F_{IO_2} 0.4, and the endotracheal tube was removed. The patient was treated

TABLE 1. Pertinent Hemodynamic Values

Time	Anesthetic	Mean Arterial Blood Pressure (mm Hg)	Heart Rate (beats/min)	CVP (cm H_2O)	Cardiac Index (l/m/m²)	TPR (dynes/sec/cm⁻⁵)	P_{O_2} (mm Hg)	P_{CO_2} (mm Hg)	pH	Event
1000		80	90	15	4.91	630	51.2	33.7	7.43	Preoperatively; room air
1015	Oxygen						105			5 minutes of preoxygenation
1030	N_2O 50 per cent; halothane 1 per cent; oxygen	70	100	16	4.70	570	50	50	7.20	15 minutes into induction
1120	Halothane 1 per cent; oxygen	65	95	20	4.95	500	65	34	7.43	15 minutes after incision
1215	Cyclopropane 10 per cent; oxygen	85	85	18	4.00	800	101	34	7.41	Cyclopropane on for 45 minutes
1300	Cyclopropane 10 per cent; oxygen	90	80	20	4.30	800	120	31	7.43	Cyclopropane on for 90 minutes
1400	Halothane 1 per cent; oxygen	80	80	24	4.65	600	100	30	7.45	Halothane on for 90 minutes

with 30 per cent oxygen by mask for two days. Recovery was otherwise uneventful.

Discussion

The preoperative evaluation of the first patient, including normal Pa_{O_2}, electrocardiogram, and chest x-ray and a negative cardiac history, lulled us into a false sense of security. Problems started with the intubation of a presumably hypoxic patient and continued throughout a week of postoperative ventilation. The cause of the hypoxemia was believed to be induction of anesthesia with an agent that dilates the peripheral vasculature, opens more shunts, and depresses the myocardium.

Two types of shunt are found in these patients: 1) a systemic left-to-right shunt similar to an arteriovenous fistula in the periphery, and 2) a pulmonary right-to-left shunt. The high cardiac output and small arteriovenous oxygen content differences result from the decreased total peripheral resistance due to the shunts. Siegel postulated that the abnormal vascular tone may be caused by a vasoactive substance normally metabolized by the liver.[2] Recently, Said has isolated from dog intestine a vasoactive polypeptide (VIP) that dilates systemic blood vessels, increases cardiac output, and decreases pulmonary vascular resistance.[4] Patients who do well are able to maintain a sufficiently high cardiac output to supply both tissues and shunts without developing cardiac decompensation.[5, 6] Sugarman has shown that total peripheral resistance can be increased, the narrow arteriovenous oxygen content difference widened, and cardiac output decreased in these patients by a metaraminol intravenous drip.[7] Perhaps metaraminol closes some systemic arteriovenous shunts, thereby decreasing the demand for an elevated cardiac output.

Of the three causes of arterial unsaturation, two, diffusion block and ventilation–perfusion abnormalities, have been shown not to be the major factors in patients with hepatic cirrhosis.[2, 8, 9] The third is venous admixture, and right-to-left shunts amounting to 8–49 per cent of the total cardiac output have been found in these patients.[8, 10] The venous admixture occurs through either portopulmonary or intrapulmonary shunts.[11] Isselbacher believes the route of portopulmonary shunts to be "from the portal system into the mediastinal veins, the paraesophageal veins and then into the azygos system into which the bronchial veins empty. In the presence of portal hypertension there is retrograde flow of blood from the azygos system into the bronchial veins and thence via anastomoses into the pulmonary veins."[12] The intrapulmonary shunts cannot be demonstrated by gross or microscopic examination of the lung postmortem. Techniques of injection of the pulmonary vessels must be used, and when this is done spider nevi of the pleura are found.[13]

Anesthetic management of the cirrhotic patient for portacaval shunt must be planned to minimize hypoxemia and cardiac failure. Preoperatively, there should be a complete cardiopulmonary evaluation, including a thorough history and physical examination, electrocardiogram, chest x-ray, determination of arterial blood gases, pulmonary function tests, and cardiac output and total peripheral resistance measurements. Even when all results are normal, a high index of suspicion must be maintained.

The patient should be anesthetized with a high-oxygen technique, thus eliminating the use of nitrous oxide. Our second case suggests that cyclopropane is better than halothane for this purpose. Cyclopropane might close already-existing shunts, or at least not open as many new ones as a vasodilator such as halothane. Also, the myocardial depressant effects of cyclopropane are less than those of halothane in intact man.[14] Further studies comparing anesthetic agents in this type of patient are now in progress.

Monitoring during anesthesia and operation should include continuous determinations of arterial pressure, central venous pressure, temperature, and urinary output, electrocardiogram, and frequent determinations of arterial blood gases, in addition to use of an esophageal stethoscope. Throughout the procedure cardiac output can be measured using a dye-dilution technique and signs of cardiac decompensation can be detected and treated. Similar monitoring postoperatively is essential, and ventilation must be assisted for as long as necessary.

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Automatic Ultrasound Monitoring of Blood Pressure during Induced Hypotension

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Accurate measurement of arterial blood pressure is necessary for the safe management of patients undergoing hypotensive anesthesia. Under these conditions sphygmomanometric readings are often inaccurate or unobtainable, and oscillometry is unreliable, especially in obese patients.[1] Therefore, intra-arterial monitoring has frequently been necessary. While this method is accurate, it may be attended by difficulties and patient morbidity. In addition, the procedure is undesirably cumbersome for routine clinical use. For these reasons, the development of an indirect, noninvasive method of blood pressure measurement to provide accurate, easily interpreted, and reproducible results was urgently needed.

This paper describes the results obtained with an automatic ultrasonic Doppler blood pressure monitoring device (AUDM) § in patients before and during induced hypotensive anesthesia for radical surgery of the head and neck.

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Received from the Department of Anesthesiology, College of Physicians and Surgeons, Columbia University, New York, and the Anesthesiology Service, The Presbyterian Hospital in the City of New York, New York 10032. Supported in part by grant GM 09069 from the Institute of General Medical Sciences, National Institutes of Health, and in part by a grant from Roche Medical Electronics Division, Hoffmann-La Roche Inc., Cranbury, New Jersey.

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