patient manifesting anesthetic-associated hepatic dysfunction. The importance of the present data is to emphasize the absence of detectable differences between commonly used anesthetic drugs with respect to postoperative hepatic dysfunction. Indeed, evidence of hepatic dysfunction in our study, using a sensitive indicator of hepatocellular damage (LDH_5), was minimal even in those patients undergoing upper abdominal operations. For example, LDH_5 remained above normal in only four of 30 patients one day following cholecystectomy. Although these four patients all had received enflurane, the LDH_5 elevations were minimal, and we feel in this small series should not be interpreted as representing a specific anesthetic effect.

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Bronchoscopy and Reversal of Intracardiac Shunt

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The direction of shunt is from left to right in patients with atrial septal defect, because of the higher left atrial pressure (LAP) compared with the right atrial pressure (RAP).[1] Various conditions, including pulmonary embolism, hypercarbia, acidemia, systemic arterial hypotension, and increased airway pressure, may reverse the shunt and result in arterial hypoxemia.[2,4] The following case demonstrates the reversal of a left-to-right shunt with the institution of positive-pressure breathing during bronchoscopy.

Report of a Case

A 65-year-old man was admitted with a history of hemoptysis and was scheduled for bronchoscopy. Past history revealed that since birth he had had a heart murmur. He had no limitation of activity, and no history of cyanotic spells. Cardiac catheterization performed during the present admission revealed an atrial septal defect with 4-to-1 left-to-right shunt, with normal valves, coronary arteries, and pressures. Results of other laboratory investigations were within normal limits. Preoperative blood-gas values during breathing of room air were: *p*H 7.42, P_{O_2} 91 torr, P_{CO_2} 36 torr, and base excess 2.

The patient was premedicated with atropine sulfate, 0.4 mg, and secobarbital, 100 mg, im, an hour prior to the anticipated bronchoscopy. In the operating room, ECG and heart rate were continuously monitored. Blood pressure was monitored by an appropriate-sized cuff. Prior to the induction of anesthesia, vital signs were: heart rate 84/min, blood pressure 126/88 torr, respiratory rate 16/min. ECG revealed normal sinus rhythm. After preoxygenation, anesthesia was induced with a sleep dose of thiopental and endotracheal intubation was facilitated with succinylcholine. Anesthesia was maintained with 50 per cent nitrous oxide in oxygen, supplemented by fentanyl, and muscular relaxation was maintained by infusion of 0.2 per cent succinylcholine. Ventilation was controlled manually, and breath sounds were satisfactory. An adult-sized flexible fiberoptic bronchoscope was passed through the endotracheal tube, and bronchoscopy revealed a small mass in the right main bronchus. Throughout this time, patient was manually ventilated, and vital signs were stable. It was decided to perform a rigid bronchoscopy to obtain a biopsy of the mass. The patient's trachea was extubated and a 7-mm rigid bronchoscope passed into the trachea without difficulty. A Sanders ventilating attachment was connected to the proximal end of the bronchoscope, and the patient was ventilated at a rate of 15/min using a mixture of 50 per cent nitrous oxide in oxygen delivered by a high-flow N_2O–O_2 blender, which delivered the gas mixture at a constant pressure of 50 psi. Vital signs were stable, and chest expansions were good and bilaterally equal with each jet inflation. Analysis of a sample of arterial blood obtained at this time revealed *p*H 7.44, P_{O_2} 120 torr, and P_{CO_2} 32 torr. After 10 min of bronchoscopy, the patient was noticed to be cyanotic and tachycardic, with a rate of 130/min. An arterial blood sample was drawn, and analysis revealed *p*H 7.38, P_{O_2} 49 torr, and P_{CO_2} 42 torr. The patient was ventilated with 100 per cent oxygen with the Sander's jet ventilation attachment through the bronchoscope. Since the cyanosis did not abate, another arterial blood sample was drawn, and analysis revealed *p*H 7.19, P_{O_2} 59 torr, P_{CO_2} 48 torr, and a base deficit of 7. The bronchoscope was withdrawn, the trachea was reintubated with 9-mm endotracheal tube, and manual ventilation was instituted with 100 per cent oxygen. Sodium bicarbonate, 50 mEq, was given iv to correct the metabolic acidosis. Five minutes later, since the cyanosis was still present, another

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arterial blood sample was analyzed, revealing *p*H 7.4, P_{O_2} 56 torr, P_{CO_2} 49 torr, and a base excess of 3. A diagnosis of increased pulmonary vascular resistance with reversal of shunt was made, and trimethaphan camsylate, 0.01 per cent, solution was administered iv using an IVAC®—at a rate of 25 microdrops/min. Arterial pressure decreased from 140/86 torr to 124/76 torr in 4 min. Ten minutes after starting trimethaphan, administration cyanosis disappeared. Analysis of arterial blood with $F_{I_{O_2}}$ 1 revealed *p*H 7.38, P_{O_2} 148 torr, and P_{CO_2} 40 torr. At this time, the patient was awake, alert and obeying commands. He could not tolerate the endotracheal tube. After suctioning, the trachea was extubated and 50 per cent oxygen was administered by mask. Arterial blood-gas analyses repeated after 15 min, showed *p*H 7.46, P_{O_2} 210 torr, and P_{CO_2} 44 torr. The patient was transferred to the recovery room with continuation of infusion of 50 per cent oxygen by mask and trimethaphan infusion. The trimethaphan infusion was slowly discontinued over 45 min. An hour following cessation of trimethaphan administration, arterial blood-gas values at $F_{I_{O_2}}$ 0.5 were *p*H 7.42, P_{O_2} 184 torr, and P_{CO_2} 38 torr. Chest x-ray taken in the recovery room was normal, and there was no evidence of collapse of the lung or pneumothorax. Next day, during breathing of room air, arterial blood P_{O_2} was 89 torr, and patient was discharged from the recovery room.

Discussion

Shunting of the blood mainly in a left-to-right direction occurs in classic atrial septal defect because the mean pressure is higher in the left than in the right atrium.[1] However, the shunt flow through an atrial septal defect is pulsatile, and follows the pressure gradient generated between the two atria.[5] Thus, during the first half of systole, the tricuspid valve, with 60–75 per cent more area than the mitral valve, may bulge more, and causes an increase in RAP compared with LAP, with a right-to-left shunt. During the latter half of systole, which corresponds to venous inflow into the atria, LAP increases more than RAP because of the less compliant nature of the left atrium, and the direction of shunt flow is changed to left to right. The diastolic phase shows flow and gradients in a left-to-right direction. Thus, direction of shunt across an ASD changes with different phases in each cardiac cycle. Alexander *et al.* have also shown that a sustained airway pressure of 30 torr or more reverses the shunt flow in a right-to-left direction.[5]

When a Sanders ventilating attachment is used during bronchoscopy, a pressure of 25 to 36 torr can be attained at the distal end of the bronchoscope, depending on the size of the bronchoscope.[6] Chakravarthy *et al.* have shown that with the jet on, the distal airway pressure reaches values as high as 35 torr.[7] It has been demonstrated that the use of the Sanders ventilating attachment results in variable entrainment of air, with hypoxemia in critically ill patients. Also, the jet device does not have a means of reducing the flow rate of gases, which might result in production of excessive pressures in the airway.[8] During rigid bronchoscopy, our patient was ventilated with a Sander's attachment, which may have resulted in high airway pressure. High airway pressure then produced an increase in pulmonary vascular resistance, increasing right atrial pressure above that of the left atrium. This caused a reversal of left-to-right shunt, with hypoxemia and hypercarbia, which further increased pulmonary vascular resistance. Moorthy *et al.* have documented worsening of hypoxemia following 12.5 cm PEEP in a patient with a patent foramen ovale.[9] Ventilation with 100 per cent oxygen does not improve arterial oxygenation in the presence of an intracardiac right-to-left shunt. Following correction of acidosis and the use of trimethaphan, a potent pulmonary vasodilator, pulmonary vascular resistance decreased, leading to a reduction in right ventricular end-diastolic pressure (RVEDP) and RAP. This resulted in cessation of the right-to-left shunt and improvement of arterial oxygenation. Hypoxemia could have also resulted from the obstruction of the right main-stem bronchus, in which case breath sounds should have been absent or diminished. Throughout the procedure, however bilateral breath sounds were equal with normal chest expansion. Also, if the hypoxemia had been secondary to right main-stem bronchial obstruction, it would not have responded to trimethaphan therapy.

Reversal of a left-to-right shunt at the atrial level may occur following systemic hypotension or increased RVEDP, for whatever reason. If reversal of shunt is due to systemic hypotension, therapy should include either phenylephrine or norepinephrine, which will increase the systemic vascular resistance and convert the shunt into a left-to-right shunt. However, this patient did not experience systemic hypotension intraoperatively, so there was no need to use drugs that increase systemic vascular resistance. Various drugs, including trimethaphan, tolazoline, phentolamine, nitroglycerine, sodium nitroprusside, and chlorpromazine, have been utilized to produce pulmonary vasodilatation and thereby decrease RVEDP. Mixed vasodilators, which dilate both arterial and venous systems, produce maximum pulmonary vasodilatation.[10,11] Drugs that have this property are trimethaphan and nitroglycerine. These drugs should be controlled so that they produce maximum pulmonary vasodilatation with minimal changes in systemic pressure, however.

Other causes producing increased RVEDP include pulmonary embolism,[12] pulmonary stenosis,[13] pulmonary hypertension,[14] ventricular septal defect,[15] hypoxemia, positive-pressure ventilation and PEEP,[16] chronic congestive failure, and nodal rhythm with tricuspid regurgitation. None of the above-mentioned causes, including acidosis, hypercarbia, or hypoxemia,

played a role in the reversal of the shunt, because results of arterial blood-gas analysis 10 min prior to the onset of cyanosis were within normal limits.

In conclusion, if cyanosis appears suddenly with deterioration of Pa_{O_2} immediately following the institution of positive-pressure breathing or PEEP in patients with left-to-right shunts, the possibility of reversal of intracardiac shunt should be suspected. In such situations, administration of 100 per cent oxygen may not increase arterial blood P_{O_2}, and addition of PEEP may worsen the arterial oxygenation. Therapy should be aimed at reducing RAP in comparison with LAP. This could be achieved by careful administration of vasodilators, which both reduce the preload to the right atrium by systemic venous dilatation and reduce the afterload of the right ventricle by producing pulmonary vasodilatation, thereby decreasing the shunting of blood from right to left.

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Obstetric Anesthesia for a Quadriplegic Patient with Autonomic Hyperreflexia

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The quadriplegic patient with autonomic hyperreflexia represents an unusual and complex array of medical and surgical problems, necessitating highly individualized anesthetic care for successful management.[1,2] One case of labor and delivery in a quadriplegic patient complicated by autonomic hyperreflexia but without anesthetic intervention has been reported.[3] A single report of epidural anesthesia in a quadriplegic patient for urologic surgery exists.[4] We report here what is to our knowledge the first documented case of labor and delivery in a chronic quadriplegic patient managed with epidural anesthesia.

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Report of a Case

A 23-year-old quadriplegic woman, gravida 2, spontaneous ab 1, para 0, with an intrauterine pregnancy was admitted to the obstetrical unit for perinatal management and delivery. She had been quadriplegic since the age of 12 years following traumatic damage to the cervical spinal cord at the C5–6 level, leaving sensation to T4 intact. She had had an indwelling Foley catheter since that