

syncratic reaction to the bupivacaine. Another possible method of treating the phantom pain in this case might have been the use of general anesthesia as described when it occurred under spinal anesthesia^{2,3}; however, this would have been inappropriate in this case as the patient was already in the recovery room by the time the phantom pain appeared. Other possible forms of treatment might have been the use of anticonvulsants or antidepressants as described previously for the treatment of chronic phantom pain, but such extrapolation from chronic to acute pain is unreliable because of the great psychologic and possibly physiologic differences between chronic and acute pain.

Spinal narcotics are thought to provide analgesia by neuronal blockade in the substantia gelatinosa of the spinal cord¹¹ and so do not cause anesthesia. We consider that this makes spinal narcotics the treatment of choice in cases such as this where acute phantom pain is associated with epidural anesthesia, and possibly in similar cases associated with spinal anesthesia.

We conclude that epidural anesthesia did not produce phantom pain during its period of action as has been reported under spinal anesthesia.¹⁻³ Because of these reports we had felt some reluctance in agreeing to this patient's request for epidural blockade. Yet, perhaps by the mechanism described previously, the epidural anesthesia did seem to cause phantom limb pain as the block wore off, because a pain appeared which was both more severe and in a different site to the patient's usual phantom pain. This was readily relieved by the epidural opiate and no

doubt would also have been temporarily relieved by further epidural local anesthetic. Faced with a similar request again, not only would we be prepared to use an epidural block for the operation, but we would ensure that the epidural catheter was retained into the postoperative period as a means of providing pain relief, preferably by use of a narcotic.

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65:221-224, 1986

Increased Perioperative Risk Following Repair of Congenital Heart Disease in Down's Syndrome

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Patients with Down's syndrome (DS) have an incidence of congenital heart disease (CHD) of approximately 40%.¹ Early surgical intervention can prevent associated congestive heart failure, pulmonary hypertension, and pulmonary vascular obstruction.^{1,2} Children with DS may have a propensity for early development of pulmonary

vascular obstruction,^{3,4} although this conclusion has been disputed.^{5,6} Perioperative mortality is a function of both age at the time of surgery and complexity of the cardiac defect. In one series of DS patients with complete atrioventricular canal (AVC), mortality was 50% for patients less than 3 months of age, and 17% for patients at 12 months.⁷ Another series reported a mortality rate of 52% for all patients with DS with AVC, compared with 20%

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Received from the Departments of Anesthesiology and Pediatrics, University of Washington School of Medicine, Children's Orthopedic Hospital and Medical Center, Seattle, Washington. Accepted for publication March 19, 1986.

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Key words: Anesthesia: cardiac. Heart: congenital defects, Down's Syndrome.

TABLE 1. Comparison of Weight, Cardiovascular Variables, and Duration of Care in Patients With and Without Down's Syndrome

	Down's (n = 23) (mean \pm SD)	Non-Down's (n = 23) (mean \pm SD)	P*
Weight (kg)	11.8 \pm 9.7	14.9 \pm 13.2	<0.01
Qp/Qs	2.5 \pm 0.7	2.8 \pm 1.0	NS
CPB (min)	71.3 \pm 16.4	61.0 \pm 21.9	NS
PAP (mm Hg)	47.6 \pm 11.9	27.0 \pm 11.2	<0.001
Ventilator days	3.9 \pm 4.5	1.4 \pm 1.2	<0.01
ICU days	7.8 \pm 4.5	4.0 \pm 2.3	<0.001
Hospital days	13.7 \pm 6.4	8.9 \pm 2.4	<0.001

NS = not significant. See text for additional abbreviations.

* Student's *t* test for paired data.

for those with simple ventricular septal defect (VSD).² Perioperative mortality for all DS patients with CHD is approximately 30%.^{8,9} No cause for this high perioperative mortality rate has been identified, although perhaps a different standard of care exists for patients with DS, since they may be offered surgical repair at an older age than their non-Down's syndrome (NDS) counterparts.¹⁰ No one has compared the perioperative morbidity and mortality in a DS population with CHD to that of an NDS population matched for age and congenital heart lesion. This was the purpose of our retrospective study.

METHODS

Between January 1980 and June 1984, 23 children with DS were admitted for surgical closure of a VSD during cardiopulmonary bypass. These children ranged in age from 3 to 216 months (mean \pm SD = 38.8 \pm 48.8 months). Following surgical repair under moderate hypothermia (temperature 26–28°C), the patients were rewarmed and weaned from cardiopulmonary bypass (CPB). After cardiopulmonary stability was achieved and the chest closed, the patients were transferred to the pediatric intensive care unit (ICU) for mechanical ventilation and postoperative care. Patients were considered eligible for tracheal extubation when they were awake and had stable hemodynamics and an arterial oxygen tension greater than 80 mmHg while breathing spontaneously in an inspired oxygen concentration of 50% or less. Transfer from the ICU to the ward was accomplished once the patients were hemodynamically stable without need for inotropic drugs; required less than 30% inspired oxygen, had all chest tubes and intravascular monitors removed; and had no significant dysrhythmias or signs of sepsis. Discharge from ward to home occurred when the patients no longer required supplemental O₂, were eating well, were afebrile for 24 h, and had pacing wires and skin sutures removed.

The hospital record of each of these patients was reviewed and the following information was recorded: age; weight; cardiac defect; preoperative mean pulmonary ar-

tery pressure ($\overline{\text{PAP}}$) and ratio between pulmonary and systemic flow (Qp/Qs); preoperative medications; presence of preoperative respiratory or congestive heart failure; surgical procedure; CPB time; number of days of mechanical ventilation, intensive care, and total hospitalization; need for pacing or inotropic drugs; presence of sepsis (defined as temperature greater than 38°C, white blood cell count greater than 10,000 cell/mm³, and positive blood culture); pulmonary parenchymal disease (defined as radiographic evidence of atelectasis, pulmonary edema, pneumonitis, or pleural effusion as read by a radiologist); and final disposition.

For each DS patient, an NDS patient of the same age§ with a VSD operated on during the same period of time was selected for an identical review. These children ranged in age from 2.5 months to 240 months (mean \pm SD = 40.7 \pm 53.3 months). The clinical management of the NDS patients followed the same procedures and guidelines listed previously. Four of the DS patients had an associated patent ductus arteriosus and were matched with NDS patients with the same associated lesion. The DS and NDS groups were compared using Student's paired *t* test for ordinal data and the chi-square analysis for nominal data. Differences were considered significant if *P* < 0.05. The association between $\overline{\text{PAP}}$ and duration of mechanical ventilation, ICU stay, and total hospitalization in DS patients was analyzed using linear regression.

RESULTS

Data from the DS and NDS groups are summarized in table 1. The DS group, compared with their age-matched controls, were significantly smaller, consistent with the growth retardation seen in this syndrome.¹¹ Ten DS patients and eight NDS patients were receiving digitalis and diuretics in the preoperative period, although only four DS patients and three NDS patients had overt signs of congestive heart failure. No patient in either group had respiratory failure requiring mechanical ventilation. Preoperatively, the DS group had significantly higher $\overline{\text{PAP}}$ than the NDS group, and postoperatively required more ventilator, ICU, and hospital days. There was no difference between the groups in preoperative Qp/Qs or CPB time. No correlation could be demonstrated in DS patients between $\overline{\text{PAP}}$ and ventilator days (*r* = .01), ICU days (*r* = .17), or hospital days (*r* = .09).

One of the DS patients died of sepsis (4%). None of the NDS patients died. Other postoperative complications are shown in table 2. The DS group had a significantly higher incidence of radiographically diagnosed atelectasis

§ Within 1 month for patients less than 2 yr of age and within 6 months for patients greater than 2 yr of age.

and pulmonary edema. There was no difference between the groups in the incidence of other complications.

DISCUSSION

In this study, children with DS and VSD, when compared with age-matched NDS children with the same cardiac lesion, had higher preoperative pulmonary artery pressures. The similarity in preoperative Qp/Qs and CPB time suggests that the cardiac lesions were similar in both groups. The finding of increased pulmonary artery pressures in the DS group supports the contention that children with DS are predisposed to the early development of pulmonary vascular obstructive disease.^{3,4} Perhaps DS children with CHD have a higher incidence of pulmonary hypertension because they are referred for corrective surgery at a later age than NDS children with CHD.¹⁰ The results of this study, in which age was a controlled variable, suggest that factors other than age must be invoked to explain the difference. For example, DS patients have an abnormal upper airway, which predisposes to chronic upper airway obstruction, hypoxemia, and elevated pulmonary artery pressures.^{12,13} Cooney and Thurlbeck¹⁴ studied the lungs of seven patients with DS with or without congenital heart disease and in six found a diminished number of alveoli in relation to acini, a smaller number of alveoli, and a smaller alveolar surface area compared with NDS controls. They speculated that the diminished alveolar number resulted in a reduced cross-sectional area of the pulmonary capillary bed and aggravated pulmonary hypertension. Yamaki *et al.*¹⁵ performed morphometric examinations of the pulmonary vascular bed in 21 patients with DS and cardiac defects. They found that intimal changes developed at an earlier age and were more severe than in NDS patients with cardiac lesions at the same level of pulmonary artery pressure. However, in the DS group, the media of small pulmonary arteries was thinner at the same vessel radius and pulmonary artery pressure than the NDS group with cardiac lesions. They speculated that abnormal development of the medial layer in the DS group in response to increased pulmonary artery pressures exposed the distal pulmonary vascular bed to high pressure and flow, resulting in accelerated development of intimal thickening and vascular obstruction.

Our study also identified an increased duration of mechanical ventilation, intensive care, and total hospitalization in the DS patients compared with NDS controls. Of the potential sources of morbidity and need for prolonged care, only a higher incidence of atelectasis and pulmonary edema, defined by radiographic criteria, could be identified. Similarly, a recent study of 46 patients with AVC, including both DS and NDS patients, noted that the DS group had a higher incidence of postoperative atelectasis

TABLE 2. Comparison of Complications in Patients With and Without Down's Syndrome

	Down's (n = 23) No. Patients	Non-Down's (n = 23) No. Patients	P*
Sepsis	2	0	NS
Cardiac pacing	5	1	NS
Congestive heart failure	3	0	NS
Pleural effusion	5	1	NS
Pneumonia	2	0	NS
Atelectasis	19	13	0.05
Pulmonary edema	10	3	0.02

NS = not significant.

* Chi-square analysis.

as well as congestive heart failure.¹¹ We suggest that impaired development of alveoli and the pulmonary vascular bed may predispose DS patients to both preoperative pulmonary hypertension and to pulmonary complications in the perioperative period, although no statistical correlation was found in this group of patients between preoperative pulmonary artery pressure and the incidence of postoperative complications. Any cause-and-effect relationship in DS patients between increased pulmonary artery pressure, increased risk of pulmonary complications, and the need for more ventilator, ICU, and total hospital days must remain speculative.

In summary, children with DS presenting for closure of a VSD, compared with NDS children of the same age and with the same cardiac lesion, had a higher preoperative pulmonary artery pressure, higher incidence of postoperative atelectasis and pulmonary edema, and longer requirement for mechanical ventilation, intensive care, and hospitalization.

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Anesthesiology
65:224-226, 1986

Use of Local Anesthetics with Epinephrine for Epidural Anesthesia in Preeclampsia

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Fear of excessive hypertension in preeclamptic parturients has led to the notion that epidurally administered local anesthetics should not be mixed with epinephrine.^{1-3,†} However, this seems to be a theoretical concern and actual occurrence of excessive hypertension in this situation has not been reported. We describe four cases in which epinephrine-containing solutions were used in such patients without any deterioration in the condition of the parturient or the fetus.

REPORT OF FOUR CASES

Case 1. A 25-yr-old woman G₅P₂, 40-weeks gestation with preeclampsia, requested lumbar epidural anesthesia (LEA) for labor analgesia. She was asthmatic and had a past history of iv drug abuse (heroin) and hepatitis B. Medications included: anhydrous theophylline (sustained release), 300 mg q 8h and metaproterenol inhaler, 2 puffs qid prn. She had received LEA for vaginal delivery in the past without incident. Height was 170 cm, weight was 75 kg. Arterial blood pressure was 180/92 mmHg and heart rate 90 beats/min. Physical examination revealed mild expiratory wheezing, without acute respiratory distress, 3+/4 deep tendon reflexes and 1+/4 clonus. Clotting profile was normal. She was receiving a magnesium sulfate infusion at 2 g/h. After receiving 1000 ml of lactated Ringer's solution, LEA was performed uneventfully at L4-5, using 2% lidocaine with 1:200,000 epinephrine.

A total dose of 15 ml (75 µg epinephrine) was given incrementally to achieve a T-10 level. Arterial blood pressure was 180/90 mmHg and heart rate 80 beats/min before LEA and remained unchanged 30 min post-LEA. Shortly afterward a vigorous female infant, with apgar scores of 9/9 (at 1 and 5 min, respectively), was born. The preeclampsia resolved shortly thereafter.

Case 2. A 20-yr-old woman, 41-weeks gestation, with preeclampsia presented for a cesarean section for failure to progress during labor. She had no significant past medical history. On physical examination, she was 163 cm, 79.5 kg, with moderate generalized edema. Arterial blood pressure varied between 140/80 to 170/100 mmHg, and she had 2+/4 clonus. Clotting profile was normal. Magnesium sulfate was administered iv at a rate of 2 g/h. LEA was performed uneventfully at the L3-4 interspace, using 2% lidocaine with 1:200,000 epinephrine following infusion of 1500 ml lactated Ringer's solution. A total of 26 ml (130 µg epinephrine) was given incrementally to achieve a T4 level of anesthesia. At the start of the procedure, arterial blood pressure was 130/90 mmHg and heart rate 85 beats/min. Thirty minutes later, arterial blood pressure decreased to 100/60 mmHg with a heart rate of 130 beats/min. Treatment with ephedrine 5 mg iv resulted in a return to baseline values. During delivery, the arterial blood pressure remained at 110/70 mmHg and a heart rate of 100-130 beats/min was sustained for the remainder of the procedure. A vigorous male infant, apgar scores of 9 at both 1 min and 5 min following delivery, was born. The preeclampsia resolved after 2 days, at which time the magnesium sulfate was discontinued.

Case 3. A 13-yr-old girl with preeclampsia requested LEA for labor analgesia at 5 cm dilation. She had no significant past medical history. On examination, she was 157 cm tall and weighed 85 kg. Arterial blood pressure was 140/104 mmHg, and she had mild generalized edema. Clotting studies were normal. No magnesium sulfate was administered because reflexes were normal. LEA was performed uneventfully at the L3-4 interspace, after receiving 1000 ml lactated Ringer's solution, using a combination of bupivacaine 0.5% plus lidocaine 2% with 1:200,000 epinephrine mixed in equal volumes (to yield bupivacaine 0.25%, lidocaine 1%, and epinephrine 1:400,000). A total of 8 ml (20 µg epinephrine) was given incrementally to achieve a T9 level. The pre-LEA arterial blood pressure was 140/100 mmHg with a heart rate of 100 beats/min initially, and they did not change

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Received from the Department of Anesthesiology, Yale University School of Medicine, 333 Cedar Street, New Haven, Connecticut 06510. Accepted for publication March 24, 1986.

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Key words: Anesthesia: obstetrics, eclampsia. Anesthetic techniques: epidural. Sympathetic nervous system: epinephrine.

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