expect direct effects due to anesthetic agents; this is the first report of a demonstrably abnormal respiratory pattern in a full-term infant first noted following general anesthesia. This was not expected in a full-term healthy child who had gained weight and did not have a surgical procedure that would cause pain.

This child initially demonstrated a high frequency of periodic breathing in the operating room and the recovery room; this respiratory pattern is clearly abnormal in a full-term infant. This case emphasizes the importance of careful observation of all young infants who require surgery and anesthesia. This case has clinically important implications for patients scheduled for out-patient procedures because it is obvious that immaturity of the respiratory center is not limited to children with a history of prematurity, although the likelihood of such respiratory immaturity is inversely related to postconceptual age.[5,¶]

In conclusion, we suggest that neonates considered for surgery should ideally be the first case of the day so that the period of observation may be extended as long as is clinically indicated.[15] Although this is only one case in a full-term infant, it appears that abnormalities of respiration can be unmasked, just as in ex-premature infants, by the stress of surgery and/or administration of general anesthetic agents; such abnormalities will be most evident in the immediate postoperative period. Although our patient had a demonstrable abnormality of respiration, it would be premature to make specific recommendations regarding the management of all newborn patients as day surgical patients. We report this case to heighten the practitioner's awareness that such a problem may arise, albeit rarely; we are not suggesting that all infants be admitted to the hospital. The red flag in this case was persistent periodic breathing. If there is any question of abnormal breathing pattern in the infant, the infant should be admitted to the hospital for further observation of the respiratory pattern. Such infants should then undergo a more rigorous respiratory workup once the influences of

surgery and anesthesia have cleared. This infant may well represent a class of patients whose first episode of clinically apparent apnea is unmasked by anesthetic agents.

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Recurrent Paralysis Following Piperacillin Administration

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Several antibiotics have been shown to cause potentiation of the neuromuscular blockade produced by non-

depolarizing muscle relaxants.[1] Most clinical reports have involved the aminoglycosides, polymixins, and clinda-

mycin. Penicillins have been thought to be devoid of these actions at clinically relevant doses.[1] In this report, we present a case of recurrent paralysis following piperacillin administration in a patient who had previously undergone neuromuscular blockade with vecuronium.

CASE REPORT

A 29-yr-old, 80-kg man required exploratory laparotomy following two stab wounds to the abdomen. His medical history was remarkable for schizophrenia and poly drug abuse, and his sole prescribed medication was depo-haloperidol (last received 30 days prior to admission). Drug use the evening of presentation included cocaine and ethanol. Physical examination was unremarkable. A blood ethanol concentration of 158 mg% was noted.

Following 2 mg metacurine, anesthesia was induced with fentanyl 250 μg and thiopental 450 mg. Succinylcholine 100 mg was given and the patient's trachea was intubated without difficulty. Anesthesia was maintained with isoflurane and oxygen, and abdominal relaxation was effected using vecuronium 4 mg. Additional vecuronium 2 mg was given 35 min after the initial dose to facilitate abdominal closure. Fifteen minutes after the second dose of vecuronium he received atropine 0.8 mg and neostigmine 2.5 mg; 5 min later he exhibited sustained tetanus with 50 Hz stimulation of the ulnar nerve, spontaneous ventilation at a frequency of 18 per min, and an end-tidal P_{CO_2} of 39 mmHg.

Ten minutes later, he was taken to the recovery room (RR), breathing spontaneously, with his trachea intubated. Fifteen minutes after RR admission, piperacillin 3 g was begun as a 30 min infusion. Twenty-five minutes after admission he was able to be aroused, following commands, exhibited head lift sustained for more than 5 s and was able to move all extremities including elevation of his legs. His trachea was extubated.

Forty-five minutes following RR admission and 30 min after the start of piperacillin infusion, his heart was noted to have a rate of 40 beats per min, and he was making ineffectual respiratory efforts, blinking his eyes, and was unable to lift his extremities or head. His lungs were ventilated via bag and mask with 100% O_2. An arterial blood gas drawn shortly thereafter was pH 7.10, Pa_{CO_2} 80 mmHg, and Pa_{O_2} 335 mmHg. He received naloxone 0.2 mg iv with no effect. Upon reapplication of a nerve stimulator to the ulnar nerve distribution, a 50-Hz stimulus resulted in a barely discernable contraction with rapid fade to 0. This response continued over the next 15 min while the lungs were manually ventilated. Atropine 0.8 mg and neostigmine 2.5 mg were given. After 5 min he demonstrated a strong sustained tetanus to 50-Hz stimulation of his ulnar nerve, good respiratory efforts, as well as sustained extremity and head lifts. The remainder of his recovery room stay was unremarkable and he was discharged to the ward 2.5 h after admission. Blood samples drawn in the recovery room showed normal electrolytes and the patient had not been hypothermic at any-time.

DISCUSSION

Many clinical reports have documented the propensity of some antibiotics, including the aminoglycosides, polymixins, and clindamycin,[1] to increase the relaxant effects of nondepolarizing muscle relaxants (NDMR). The typical presentation is that of an antibiotic administered pre- or intraoperatively leading to prolonged relaxation in the presence of NDMR. Our report differs from most in two ways: 1) the antibiotic involved was a penicillin; and 2) administration of piperacillin postoperatively, after ob-

jective evidence of reversal of the effects of vecuronium, resulted in reinstitution of the blockade and respiratory failure.

The penicillins have been considered clinically safe antibiotics as far as causing neuromuscular blockade or enhancing that of NDMR. However, experimentally and in supraclinical doses, penicillins (penicillin V or penicillin G) can produce neuromuscular blockade (NMB), apparently by altering presynaptic sodium conductance.[2,3] As expected, this NMB is unaffected by neostigmine. This report describes an interaction of the neuromuscular junction with an acylaminopenicillin in which reversal was obtained with neostigmine, suggesting a different mechanism from that described above.

Tryba[4] found that the administration of various acylaminopenicillins both increased the intensity and prolonged the duration of vecuronium NMB. Using an integrated EMG technique, he studied anesthetized patients relaxed with vecuronium. When vecuronium and antibiotic were administered after 25% twitch, recovery from previously administered vecuronium, a more intense (2–3% vs. 50% control twitch) and more prolonged (20–50% increase until recovery of 25% twitch) block ensued. The author speculated that the site of action of the acylaminopenicillins was prejunctional as partial antagonism with calcium was observed in the two cases in which it was administered.

In the present report, the NMB was satisfactorily reversed with neostigmine, suggesting that the site of action of acylaminopenicillin enhanced NMB is postjunctional. However, further study will be needed to elucidate the mechanism.

Recent case reports have documented reinstitution of vecuronium NMB with polymyxin/amikacin[5] and prolongation of vecuronium NMB associated with gentamycin/clindamycin.[6] As these antibiotics have previously been shown to prolong NMB with other NDMR, such interactions are not unexpected. However, the current case report differs in that it involves a penicillin, a class of antibiotic not previously thought to prolong NMB in clinically relevant settings.

In this report, we have presented an apparent case of recurrent paralysis following administration of piperacillin in a patient previously paralyzed with vecuronium. It is notable that the patient had met clinical and electrophysiologic criteria for adequate reversal of vecuronium prior to administration of the antibiotic and reinstitution of NMB. The NMB resulting from administration of piperacillin was satisfactorily reversed with neostigmine. It is prudent to carefully monitor perioperative neuromuscular function in patients receiving intraoperative muscle relaxants and piperacillin.

Similar caution may be appropriate during administration of the structurally related antibiotics azlocillin, me-

zlocillin, and cefaperazone, but more detailed information is required.

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Severe Dynamic Left Ventricular Outflow Tract Obstruction Following Aortic Valve Replacement Diagnosed by Intraoperative Echocardiography

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Left ventricular hypertrophy may develop as a consequence of longstanding aortic stenosis. In patients with severe fixed aortic valvular stenosis, secondary left ventricular hypertrophy may also cause dynamic left ventricular outflow tract obstruction.[1,2] Recovery from aortic valve replacement may be complicated by the development of symptomatic outflow tract obstruction presenting weeks to months after surgery. We report a case in which severe dynamic left ventricular outflow tract obstruction developed in the immediate postoperative period in a patient who underwent aortic valve replacement for severe aortic stenosis. Intraoperative epicardial echocardiography was used to make the diagnosis and guide management.

CASE REPORT

A 71-y-old woman was referred to the Cleveland Clinic Foundation where she presented with the recent onset of congestive heart failure. She had a history of hypertension, but was otherwise free of previous cardiac symptoms.

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On admission, clinical features of severe aortic stenosis were present, including a grade III/VI harsh systolic ejection murmur at the right upper sternal border. Preoperative EKG showed normal sinus rhythm, right bundle branch block with a left anterior hemiblock, and Q waves in V_4–V_6 and AVL. Preoperative 2-D/Doppler/echocardiography demonstrated severe concentric left ventricular hypertrophy; left ventricular chamber size and systolic function were normal. The aortic valve was heavily calcified with a maximum Doppler derived gradient of 125 mmHg and a Doppler estimated aortic valve area of 0.65 cm^2. Cardiac catheterization showed mild (+1) aortic insufficiency and no significant coronary artery disease.

Because of severe aortic stenosis with recent congestive failure, the patient underwent aortic valve replacement with a 21-mm Carpentier-Edwards porcine bioprosthesis. Anesthetic management included fentanyl (75 μg/kg), diazepam, pancuronium, and metubine. Intraoperative monitors included a central venous catheter inserted prior to induction, and a left atrial catheter inserted prior to separation from cardiopulmonary bypass. Total bypass time was 114 min, and cross-clamp time was 74 min. The patient separated easily from cardiopulmonary bypass without inotropic support or vasodilators. On arrival in the intensive care unit (ICU) the mean arterial pressure (MAP) was 86 mmHg, central venous pressure (CVP) was 16 mmHg, and left atrial pressure (LAP) was 14 mmHg; nitroprusside was initiated and titrated to keep MAP 70–90 mmHg. Initial urine output was 80 ml/h. Approximately 8 h postoperatively, her urinary output decreased to 25 ml/h with CVP and LAP unchanged. Dopamine (4 μg · kg^{-1} · min^{-1}) was begun for renal effect. Urine output did not improve and CVP and LAP began to increase. As ionized calcium was low, a bolus of calcium (500 mg $CaCl_2$) was given, and a dobutamine infusion was started at 5 μg · kg^{-1} · min^{-1}. At this time, MAP declined precipitously to 55 mmHg in spite of discontinuing nitroprusside, and the CVP and LAP rose to 20 and 44 mmHg, respectively.

These hemodynamic developments, coupled with decreased chest tube drainage, led to a provisional diagnosis of cardiac tamponade and prompted a return to the operating room for re-exploration of the mediastinum. Upon opening the chest, there was no blood in the pericardial cavity and no significant bleeding was found. The heart was not enlarged and appeared to be contracting vigorously. Boluses of epinephrine were given and an infusion was started to maintain MAP over 60 mmHg. A pulmonary artery thermistor was inserted and the