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Severe Hypoxia and Acidosis Following Local Anesthetic-induced Convulsions

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We have suspected that in man severe hypoxia and acidosis precedes or occurs concomitantly with the convulsions of systemic toxicity from local anesthetic drugs.[1] To some extent, data from studies in monkeys and cats substantiate this suspicion.[2,3] Direct evidence obtained in man has not been reported. Nonetheless, our preparation for and treatment of toxic reactions assumes that severe hypoxia and acidosis do occur.[1,4,5] In support of this thesis, two cases of bupivacaine-induced convulsions in which arterial blood-gas analysis and bupivacaine plasma levels were obtained are presented.

REPORT OF TWO CASES

Patient 1. A 25-year-old woman, height 177 cm, weight 65 kg, ASA physical status 1, was scheduled for a bilateral oophorectomy for dermoid cysts. She was premedicated with diazepam, 10 mg (0.15 mg/kg), orally, at 0800, and morphine sulfate, 15 mg, with scopolamine, 0.4 mg, im, at 0830. At 0930, intravenous fluids were started. Blood pressure was 100/60 torr with a pulse rate of 60/min. A 19-gauge needle was introduced into the epidural space at the second lumbar interspace. Aspiration for blood and cerebrospinal fluid had negative results. A 3-ml test dose of 0.75 per cent bupivacaine with 1:200,000 epinephrine was judged to be innocuous. The patient's condition was not monitored electrocardiographically. At 0949, we completed the injection of 23 ml 0.75 per cent bupivacaine (173 mg) with 1:200,000 epinephrine. Aspiration for blood and cerebrospinal fluid was again negative. The needle was withdrawn, and on instruction, the patient at once turned herself supine. Within a minute convulsions occurred, and help was immediately summoned. Between convulsions, respiratory efforts were present, and they were instantly assisted with oxygen (10 l/min), using an Ambu bag and mask and an oral airway; intubation was not performed. Blood pressure rose to 150/80 torr and pulse rate was 176/min. Arterial blood samples were drawn (table 1). After the third convulsion, diazepam, 10 mg, and thiopental, 200 mg, were given intravenously, each as a bolus dose. At 0954 and 30 sec, the patient's respirations stopped, as did the convulsions, and the blood pressure was 110/66 torr with a pulse rate of 72/min. Ventilation with oxygen was continued. When adequate respirations returned, she was sent to the recovery unit for observation. At 1115, she was not responsive, and she was given naloxone, 0.4 mg, iv. She awakened, but was restless, and physostigmine, 1.5 mg, was given intravenously at 1130. At 1135 the restlessness ceased, and at 1145 the patient became oriented as to time and place. No analgesia occurred from the block. At 1205, with general anesthesia, the operation was performed without further incident.

Patient 2. A 15-year-old boy, height 175 cm, weight 66 kg, a ASA physical status 1, was scheduled for a meniscectomy. He was premedicated with diazepam, 10 mg (0.15 mg/kg), orally, at 0800 and meperidine, 125 mg, im, at 0900. Blood pressure was 110/60 torr, with a pulse rate of 62/min. A 19-gauge needle was introduced into the epidural space at the second lumbar interspace. Aspiration for blood and cerebrospinal fluid was negative. A test dose was not administered. At 0946, we completed the injection of 20 ml 0.75 per cent bupivacaine (150 mg) with 1:200,000 epinephrine. Aspiration for blood and cerebrospinal fluid again had negative results, and the patient, on instruction, at once turned himself supine. Within a minute the patient had convulsions. Instantly he was ventilated with oxygen (10 l/min) by use of a bag and mask and an oral airway. Intubation was not performed. No spontaneous respiratory attempts occurred between convulsions. Blood pressure was 140/800 torr and pulse rate was 120/min. At 0949 and 30 sec, diazepam, 15 mg, was given intravenously. Arterial blood samples were drawn (table 1). Convulsions ceased at 0950. The systolic blood pressure was 120 torr, and pulse rate was 70/min. Adequate respirations returned. The patient was sent to the recovery unit and was oriented as to time and place at 1045. No analgesia occurred from the block. At 1130, the meniscectomy was performed with general anesthesia, without further incident.

DISCUSSION

The most impressive event in both of these cases was the rapid development of hypoxia, hypercarbia and acidosis that accompanied the convulsions (table 1). In the first patient, Pa_{O_2} fell to 33 torr within 3 min of the onset of convulsions, in spite of efforts to oxygenate the patient, and this was accompanied by an increase in Pa_{CO_2} to 59 torr and a blood *p*H of 7.09. Two and a half minutes later, with the cessation of convulsions, although oxygenation was adequate (Pa_{O_2} now 210 torr), blood *p*H had fallen to 7.01. This acidosis had both a respiratory and a metabolic component (Pa_{CO_2} = 71 torr; base excess [BE];= −11 mEq/l). Adequate ventilation alone corrected both components of the acidosis within an hour. Patient 2 showed an almost identical pattern, with development of profound hypercapnia and acidosis (Pa_{CO_2} = 76 torr; *p*H 6.99) within a minute of the onset of convulsions.

These observations suggest that during local anesthetic-induced seizures, as with seizures of any cause, carbon dioxide production (and oxygen consumption) is considerably increased. It is difficult, if not im-

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TABLE 1. Arterial Blood Levels of Bupivacaine and Results of Concomitant Blood-gas Determinations during and after Convulsions

	Arterial Blood Levels			Blood-gas Values				
	Time	Bupivacaine Base (μg/ml)	O_2 l/min	pH	P_{CO_2} (torr)	P_{O_2} (torr)	HCO_3 (mEq/l)	BE mEq/l
Patient 1								
First convulsion	0950		10*					
Second convulsion	0950 plus 30 sec	3.2		7.27	48	48	21.5	−4
Third convulsion	0951							
Fourth convulsion	0953	2.97		7.09	59	33	17.2	−10
Convulsions ceased	0954							
	0955 plus 30 sec	2.98		7.01	71	210	17.2	−11
	1022	2.58	6†	7.25	48	99	20.5	−5
	1150	0.89	Room air	7.56	25	106	22.5	0
Patient 2								
First convulsion	0947		10*					
Second convulsion	0947 plus 30 sec	4.4		6.99	76	87	17.4	−10.2
Third convulsion	0948							
Convulsions ceased	0950							
	1003	3.0		7.16	54	140	18.5	−6.9
	1018	2.2	6†	7.26	42	141	18.4	−5.8

* Bag and mask with oral Guedel airway and artificial respirations. † Nasal prongs.

possible, to provide adequate ventilation for the convulsing patient; therefore, hypoxia, hypercapnia, and acidosis quickly develop. Our data indicate that even if ventilation between convulsions provides adequate oxygenation, it cannot compensate for the increased carbon dioxide production, and hypercapnia and acidosis progress until the convulsions cease.

If efforts to ventilate were not instituted immediately, one would expect that the hypoxia, hypercapnia and acidosis would be even more severe. Add to this direct myocardial depression by the local anesthetic agent itself and by a barbiturate or other drugs given for treatment of the convulsions, and a real potential for cardiac arrest has been created. It would seem obvious, then, that when a convulsion occurs, whether it be the result of an injection of a local anesthetic agent or from any other cause, the very first efforts should be directed toward assuring adequate ventilation by whatever means possible.

No test or procedure to prevent systemic toxic reactions from local anesthetic drugs is foolproof. Therefore, the anesthetist must: 1) know the signs and symptoms of systemic toxicity; 2) monitor for them; 3) institute correct therapy immediately to avert their sequelae. At the first sign of a systemic toxic reaction, oxygen must be administered. With convulsions caused by local anesthetics, spontaneous respiration between convulsions may or may not be present; regardless, severe hypoxia and acidosis occur or are already present. Initial treatment should include establishment of an airway and ventilation, preferably with oxygen. Such treatment should not be delayed by giving depressant drugs and/or attempting to intubate the trachea. Then, while maintaining proper oxygenation or attempting to, drug therapy (succinylcholine, diazepam, and/or thiopental) may be employed, and if necessary, the trachea of the oxygenated patient can be intubated. Should cardiac arrest result anyway, manual systole must be instituted. Reversing this sequence and administering drugs or intubating the trachea of a hypoxic patient first before ventilation is established invites cardiac arrest.

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