

Obviously, severing the wire from the pull ring resulted in relieving this displacement.

We recommend the routine cutting of the pull ring from the wire in this type of endotracheal tube following successful nasotracheal intubation to prevent this complication.

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Long-acting Local Anesthetic Drugs and Convulsions with Hypoxia and Acidosis

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Albright theorizes that, "The newer local anesthetic agents (bupivacaine, etidocaine) may result in almost simultaneous seizures and cardiovascular collapse without antecedent hypoxia from typical clinical doses administered inadvertently intravenously."¹ Conversely, we reported two cases of severe hypoxia and acidosis occurring prior to or concomitantly with convulsions and proposed that any delay in the proper treatment of associated hypoxia and acidosis can trigger cardiac arrest.^{2,3}

We believe the following four cases of bupivacaine-induced convulsions with concomitant hypoxia and acidosis without cardiac arrest eliminates conjecture from our proposal.

REPORTS OF FOUR CASES

All patients were female, aged 40-60 years, and weighed 53-79 kg. The specific details of each case are summarized in table 1. These cases had the following similarities. No patient was given a test dose. In all four cases, the required dose contained epinephrine. The convulsions occurred within two minutes of the injection in three of the patients who received single-injection epidural blocks through 19-gauge Quincke spinal needles, and 10 min after completion of an intercostal nerve block in the other patient. All had hypoxia and/or acidosis. Patient 2 had only respiratory acidosis, but others had both a respiratory and metabolic component. Patient 4 had the most severe acidosis initially being both respiratory and metabolic, while later only being metabolic acidosis. Within 15 s of the onset of the convulsions, all were ventilated with 100 per cent oxygen via bag and mask. Endotracheal intubation was not performed in any patient. After adequate ventilation had been

instituted, appropriate drugs were administered. None of the patients had evidence of cardiotoxicity as indicated by changes in blood pressure or heart rate. In Patient 2 the electrocardiogram showed that her heart rate almost doubled during the convulsion and that postictally it approximated the control rate. Furthermore, on recovery from anesthesia none had any sequelae.

Interestingly, all of the patients had sensory analgesia adequate for the operative procedure. In the first patient, the systemic toxic reaction was judged to be from absorption and, therefore, analgesia was expected. Conversely, in the other patients the reactions appeared to result from unintentional intravascular injections. Evidently, in these cases a relatively small intravascular volume of the drug triggered the reaction and the rest remained in the epidural space. Only in Patients 2 and 4 were arterial plasma levels of bupivacaine obtained which were low and thus confirms only a small volume was injected intravascularly. Patients 2 and 3 received 10 mg diazepam, po, for premedication approximately 45 min prior to the completion of the blocks, and still had convulsions.⁴

After recovering from the convulsions and being oriented as to time, place, and person, Patients 1, 2, and 3 underwent surgery. For 2-6 hours following surgery, they were pain-free. The surgery for Patient 4 was canceled and performed the next day under general anesthesia. None had any sequelae from the convulsions.

DISCUSSION

Our experience from 1968 through 1980 with bupivacaine includes 20,748 regional blocks. Of these, 5,277 were intercostal nerve blocks, most of them bilateral, for surgical procedures using 290-400 mg bupivacaine in concentrations ranging from 0.375 to 0.5 per cent, with a final epinephrine content of 1:320,000 to 1:400,000. Another 11,839 were epidural blocks (7,393 lumbar, and 4,446 caudal), most with a final epinephrine content of 1:200,000. No cardiac arrests have followed the 28 convulsions from bupivacaine (25 epidural and 3 intercostal nerve). These experiences plus the now six documented cases of hypoxia and acidosis which occurred concomitantly with convulsions do not support Albright's pro-

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TABLE 1. Physical Status, Bupivacaine Solution and Concomitant Cardiovascular Blood-Gas Determinations during and after Convulsions

Type of Block	Time	ASA Physical Status	Blood Pressure (mmHg)	Pulse Rate (per min)	Respirations (per min)	Bupivacaine Solutions				Blood-gas				Drug Therapy					
						Mg	Vol (ml)	Epinephrine (mg)	Arterial Bupivacaine Base (µg/ml)	pH	P _{CO₂} (mmHg)	P _{O₂} (mmHg)	BE						
Bilateral Intercostal Nerve Patient 1. Renal endarterectomy and nephropexy 1) block completed 2) 1st convulsion 3) 2nd convulsion 4) convulsions ceased 5) pt observed 6) general anesthesia started 7) surgery, bilateral sensory level T5-12	1140	3	140/82	60	12	396	88	0.25		7.12	63	590	-7.7	thiopental, 40 mg, iv thiopental, 40 mg, iv					
	1200		120/80	60	0					7.48	27	206	-3.3						
	1210 + 15 s		120/80	60	0					7.44	33	97	-1.3						
	1211		120/80	68	12														
	1212		120/80	65	14														
	1300		110/80	65	14														
	1400		108/68	55	16														
	0945		110/60	102	12*					165	22	0.11	2.74		7.33	50	42	+0.6	diazepam, 10 mg, iv
	0958		110/60	102	12*										7.31	48	280	-1.4	
	0959 + 50 s		96/60	92	16*										7.34	44	97†	-1.4	
1001	96/60	68	16*																
1005	96/60	68	16*																
1015	96/60	68	16																
Patient 3. Anterio-posterior colporrhaphy 1) block completed 2) 1st convulsion 3) 2nd convulsion 4) convulsions ceased 5) pt responding 6) surgery, sensory level T5	1540	2	122/74	68	18	180	24	0.125		7.23	53	459	-4.5	thiopental, 60 mg, iv thiopental, 60 mg, iv					
	1600		140/?	100	0					7.36	50	159†	+3.3						
	1601		140/?	100	0														
	1601 + 30 s		110/?	98	16														
	1602		120/80	90	16														
	1615		94/60	90	18														
Patient 4. Rectal prolapse and appendectomy 1) block completed 2) 1st convulsion 3) 2nd convulsion 4) 3rd convulsion 5) 4th convulsion 6) convulsions ceased 7) pt observed 8) pt observed 9) pt responding 10) to special care unit, sensory level T6	0725	2	76/?	100	16*	188	25	0.125		7.00	59	229	-12.3	thiopental, 80 mg, iv thiopental, 80 mg, iv thiopental, 80 mg, iv sodium bicarbonate 100 mEq					
	0745		80/?	100	18*					7.11	38	339	-15.9						
	0745 + 45 s not recorded		80/?	98	18*					7.33	35	166†	-6.7						
	not recorded		80/?	90	18*					7.39	37	83†	-2.0						
	not recorded		80/?	90	18*														
	0748 + 15 s not recorded		80/?	90	18														
	0750		80/?	90	18														
	0805		80/?	90	18														

* Estimate respiratory rate between convulsions.

† Room air.

‡ Plastic bag and mask.

posal regarding the cardiotoxicity of bupivacaine.¹ If there are "susceptible individuals" in whom bupivacaine or any other local anesthetic results in convulsion and cardiac arrest without hypoxia and acidosis preceding or occurring concomitantly with the convulsion(s), we believe they are rare. Perhaps unidentified differences in preparation, precautions, and treatment can explain Albright's observation.

We conclude that presently, no data from humans or animals provide evidence that bupivacaine and etidocaine are more cardiotoxic than the shorter-acting local anesthetic drugs.^{5,6} Our experience is that immediate treatment of convulsions within 15–30 seconds of their onset especially correcting hypoxia and acidosis is not associated with a cardiac catastrophe.^{2,3,7}

Addendum

On November 20, 1981, another patient convulsed within one minute after injecting 18 ml of 0.75 per cent bupivacaine (135 mg) with 1:200,000 epinephrine while attempting an epidural block. She was immediately ventilated with oxygen and

succinylcholine 40 mg was administered iv. Two and one-half minutes after the convulsions ceased, P_{aO_2} was 76, P_{aCO_2} 109, pH 6.87, HCO_3 18.3, and BE -15.8 . No sequelae or analgesia occurred. One hour and fifteen minutes later, under general anesthesia, a colectomy was performed. The postoperative course was uneventful.

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