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.

References

1. Kefouer AK, Feingold A: Collapse of a disposable endotracheal tube by its high pressure cuff. ANESTHESIOLOGY 43:108–110, 1975
2. Kemmotsu O: Six cases of endotracheal tube obstructions. Jpn J Anesthesiol 20:259–264, 1971

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

DANIEL C. MOORE, M.D.,* GALE E. THOMPSON, M.D.,* ROBERT D. CRAWFORD, M.D.*

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 concomittantly with convulsions do not support Albrights' pro-

* Staff Anesthesiologists.

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Address reprint requests to Dr. Moore.

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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)	*p*H	P_{aCO_2} (mmHg)	P_{aO_2} (mmHg)	BE	
Bilateral Intercostal Nerve														
Patient 1. Renal endarterectomy and nephropexy	1140	3	140/82	60	12									
1) block completed	1200					396	88	0.25						
2) 1st convulsion	1210 + 15 s		120/80	60	0									thiopental, 40 mg, iv
3) 2nd convulsion	1211		120/80	60	0									thiopental, 40 mg, iv
4) convulsions ceased	1212													
5) pt observed			120/80	68	12					7.12	63	590	−7.7	
6) general anesthesia started	1300		120/80	65	14					7.48	27	206	−3.3	
7) surgery, bilateral sensory level T5–12	1400		110/80	65	14					7.44	33	97	−1.3	
Epidural														
Patient 2. Marshall Marchetti	0945	1	108/68	55	16									
1) block completed	0958					165	22	0.11						
2) 1st convulsion	0959		110/60	102	12*									diazepam, 10 mg, iv
3) 2nd convulsion	0959 + 50 s		110/60	102	12*				2.74	7.33	50	42	+0.6	
4) convulsions ceased	1001		96/60	92	16*									
5) pt responding	1005		96/60	68	16*				2.31	7.31	48	280	−1.4	
6) surgery, sensory level T-8	1015		96/60	68	16				2.16	7.34	44	97†	−1.4	
Patient 3. Anterio-posterior colporrhaphy	1540	2	122/74	68	18									
1) block completed	1600					180	24	0.125						
2) 1st convulsion	1601		140/?	100	0									thiopental, 60 mg, iv
3) 2nd convulsion	1601 + 30 s		140/?	100	0									thiopental, 60 mg, iv
4) convulsions ceased	1602									7.23	53	459	−4.5	
5) pt responding	1615		110/?	98	16									
6) surgery, sensory level T5	1700		120/80	90	16					7.36	50	159‡	+3.3	
Patient 4. Rectal prolapse and appendectomy	0725	2	94/60	90	18									
1) block completed	0745					188	25	0.125						
2) 1st convulsion	0745 + 45 s		76/?	100	16*									thiopental, 80 mg, iv
3) 2nd convulsion	not recorded		80/?	100	18*									thiopental, 80 mg, iv
4) 3rd convulsion	not recorded		80/?	98	18*									
5) 4th convulsion	not recorded		80/?	90	18*									thiopental, 80 mg, iv
6) convulsions ceased	0748 + 15 s		80/?	90	18*									
7) pt observed	0750		80/?	90	18				2.03	7.00	59	229	−12.3	
8) pt observed	0805		80/?	90	18					7.11	38	339	−15.9	sodium bicarbonate 100 mEq
9) pt responding	0840		80/?	80	18					7.33	35	166‡	−6.7	
10) to special care unit, sensory level T6	0855		98/?	80	16					7.39	37	83†	−2.0	

* 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 concomittantly 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, Pa_{O_2} was 76, Pa_{CO_2} 109, *p*H 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.

References

1. Albright GA: Cardiac arrest following regional anesthesia with etidocaine or bupivacaine. ANESTHESIOLOGY 51:285–287, 1979
2. Moore DC, Crawford RD, Scurlock JE: Severe hypoxia and acidosis following local anesthetic-induced convulsions. ANESTHESIOLOGY 53:259–260, 1980
3. Moore DC: Administer oxygen first in the treatment of local anesthetic-induced convulsions. ANESTHESIOLOGY 53:346–347, 1980
4. Moore DC, deJong RH: Toxic effects of local anesthetics. JAMA 240:434, 1978
5. Liu P, Feldman H, Covino BG: Acute cardiovascular toxicity of lidocaine, bupivacaine and etidocaine in anesthesized, ventilated dogs. ANESTHESIOLOGY 53S:231, 1980
6. Block A, Covino BG: Effect of local anesthetic agents on cardiac conduction and contractility. Regional Anesth 6:55–61, 1981
7. Moore DC, Balfour RI, Fitzgibbon D: Convulsive arterial plasma levels of bupivacaine and response to diazepam therapy. ANESTHESIOLOGY 50:454–456, 1979