

Title : EFFECTS OF BUPIVACAINE CERVICAL EPIDURAL BLOCKADE ON RESPIRATORY FUNCTION

Authors : M. Chauvin, M.D., C. Lebrault, M.D., P. Gauneau, M.D., O. Goeau, M.D., P. Duvaldestin, M.D.

Affiliation : Département d'Anesthésie-Réanimation, Hôpital Ambroise-Paré - 92100 Boulogne, France

Introduction. The use of cervical epidural anesthesia has been reported for carotid endarterectomy (1) and neck cancer surgery (2). Since these patients frequently suffer from chronic obstructive pulmonary disease (COPD) it is important to evaluate the respiratory effect of this anesthetic technique which may induce phrenic and intercostal nerve paralysis. Cervical epidural anesthesia with mepivacaïne has already been shown to reduce ventilatory capacity in patients with normal lungs (3).

The present study using a noninvasive technique was designed to measure the respiratory effects of cervical block produced by bupivacaïne.

Methods. After institutional approval and informed consent had been obtained, 18 patients undergoing carotid endarterectomy, mean age 69 ± 12 years ($X \pm SD$), mean weight 65 ± 14 kg, were studied preoperatively. None of the patients had preoperative pulmonary dysfunction. All patients were premedicated with lorazepam (2 mg PO) two hours prior to operation. With the patient sitting erect and the neck flexed, the epidural space was identified by the hanging-drop technique using a 18-gauge Tuohy needle inserted into the C7-D1 interspace. A catheter was inserted cranially to a distance of 10 cm. Respiratory measurements were performed in the supine 15° head-down position. The measurements were obtained before and 10, 20 and 30 min after the injection of 15 ml of 0.33 % plain bupivacaïne through the epidural catheter. Levels of anesthesia were determined at the same times by the pinprick method. Tidal volume (VT), respiratory rate (RR) and minute ventilation (MV) were measured with a Fleisch pneumotachograph. The ratio of the abdomen contribution to VT (ABD/VT) was continuously monitored with a noninvasive method which has been previously described (4). Two bellow pneumographs (Hewlett-Packard (HP) model 108) attached circumferentially around the rib-cage (RC) and the abdomen (ABD) were connected to two differential pressure transducers (Validyne) and their signals stored with a digital analogic interface (Adalab) in a micro-computer (Apple II E, 64 K). After calibration procedure which was performed by matching volumes measured with a pneumotachograph and signals obtained with the bellow pneumographs, each patient's respiratory variables were monitored for 2 minutes in a quiet semi-dark room. End-tidal CO_2 (ET CO_2) was monitored from a nasopharyngeal tube, using a capnograph (Diamant 6000, Cosma).

All respiratory data are presented as mean \pm SD values of 2 minutes of monitoring for each period. Statistical significance was assessed with a paired Student's t-test. Significance was assumed at a $P < 0.05$ level.

Results. The neck was anesthetized in all patients, the lowest level of anesthesia was between T1 and T10 (4 ± 3) and was obtained at 23 ± 5 min. There was no significant change in any respiratory variable during cervical epidural block, when

compared with control values, as shown in table 1. However, there were large interindividual variations. In four out of 18 patients, MV was decreased and ET CO_2 was increased by more than 10 % of control value. One of these patients had a paradoxal abdominal motion during inspiratory cycle following epidural block with a negative ABD contribution to VT (ABD/VT = -0.14 instead of 0.54 before epidural). On the other hand, ABD/VT increased by 11, 38, 60 % in the three other patients. These three patients had the lowest levels of analgesia, respectively T8, T8 and T10.

Table 1. Ventilatory variables (mean \pm SD) before and after bupivacaïne epidural injection

Variable	Control	10 (min after bupivacaïne injection)	20	30
RR (breath/min)	15.0 \pm 3.7	16.0 \pm 3.6	15.9 \pm 3.9	15.6 \pm 4.4
VT (ml)	483 \pm 192	470 \pm 195	450 \pm 174	473 \pm 188
ML (ml)	6852 \pm 2353	7252 \pm 2690	6865 \pm 2673	7083 \pm 3203
ABD/VT	0.78 \pm 0.16	0.78 \pm 0.21	0.73 \pm 0.26	0.74 \pm 0.26
ET CO_2 (%)	4.7 \pm 0.7	4.6 \pm 0.7	4.6 \pm 0.8	4.7 \pm 0.8

Discussion. This study shows that cervical epidural anesthesia by 0.33 % bupivacaïne induces inconstant change in ventilatory parameters. In comparison, mepivacaïne has been demonstrated to decrease MV, inspiratory capacity and vital capacity in all patients. In the present study, 4 out of 18 patients presented an increase in ET CO_2 . In one patient the negative ABD/VT ratio observed, suggested the presence of complete diaphragmatic paralysis. In the three other patients, hypercapnia was concomitant with a decrease in RC contribution to breathing, suggesting simultaneous partial phrenic and intercostal paralysis. Another hypothesis is that a paralysis of accessory inspiratory muscles will decompensate respiratory function in patients with preexisting poor diaphragmatic contractility. However this appears unlikely since none of these patients had preoperative pulmonary dysfunction. In conclusion, cervical epidural 0.33 % bupivacaïne may occasionally induce inspiratory muscle impairment which would be particularly deleterious in COPD patients.

References.

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