Effect of the local anesthetic agent bupivacaine prior to application of the skull-pin holder for craniotomies

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Object. The authors conducted a double-blind prospective randomized study to determine whether infiltration of Mayfield skull-pin sites with 0.5% bupivacaine, compared with placebo, would prevent hemodynamic stimulation, thus allowing for a reduction in the quantity of anesthetic agents required.

Methods. Thirty patients were randomized into two groups. There was a significant increase in blood pressure (mean systolic blood pressure 10 mm Hg, p = 0.003) in patients in the placebo group compared with that in patients in the bupivacaine group 1 minute after securing the head holder.

Conclusions. The local administration of bupivacaine for anesthetic purposes before skull-pin application may prevent potentially hazardous hemodynamic stimulation.

KEY WORDS • bupivacaine • craniotomy • skull pin • hemodynamics

CLOSE monitoring of the heart rate and arterial pressure is mandatory during craniotomy. Even a moderate increase in arterial pressure can trigger an increase in blood flow, resulting in raised intracranial pressure or rupture of an unsecured intracranial aneurysm. Incision of the skin and placement of the Mayfield skull-pin holder into the pericranium are major cardiovascular stimuli. Deepening the state of anesthesia to compensate for these phenomena may reduce cerebral perfusion pressure and delay consciousness of the patient after surgery.

We performed a randomized prospective double-blind study to compare the use of 0.5% bupivacaine with that of normal saline to infiltrate head-pinning sites. The hemodynamic response, the quantity of anesthetic agents applied, and the time required for the patient to awaken were measured to assess the effect of bupivacaine in the study group.

Clinical Material and Methods

Approval from the Institutional Review Board as well as clear and informed consent was obtained in 30 patients, who were then randomly assigned to one of two groups. Members of the test group received 0.5% bupivacaine, whereas those in the control group were given a placebo of normal saline. Eligible patients consisted of adults undergoing elective craniotomy with the aid of the Mayfield skull-pin holder while in a state of general anesthesia.

Patients were monitored using continuous electrocardiography and an automatic pressure cuff as well as intraarterial blood pressure monitoring, capnography, pulse oximetry, and temperature and neuromuscular blockade monitoring. Intravenous sufentanil citrate (0.5 μg/kg), thiopental sodium (5 mg/kg), and rocuronium bromide (0.6 mg/kg) were used for induction of anesthesia and curarization. After orotracheal intubation, anesthesia was maintained with a volatile mixture of N₂O/O₂ (70%/30%) and 0.25 to 0.5% isoflurane (inhaled fraction) as well as a perfusion of sufentanil citrate (0.5 μg/kg/hr). The neuromuscular blockade was maintained with the administration of either repeated bolus doses or continuous perfusion of rocuronium bromide.

Following a blinded infiltration of the pin sites with 30 ml of either 0.5% bupivacaine (without epinephrine) or normal saline, 2 minutes were allowed to elapse before attaching skull pins. Incision sites in patients in both groups were also infiltrated with 1% lidocaine with epinephrine, as is our normal practice to reduce scalp bleeding.

Alfentanil hydrochloride (10 μg/kg) was administered as a bolus dose when necessary, that is, if systolic blood pressure increased by 20 mm Hg or more during or after pin insertion, and during surgery.

We recorded each patient’s blood pressure and heart rate at baseline, 5 minutes after intubation, immediately before infiltration, during infiltration, and at 1 and 5 minutes after skull-pin insertion. We also recorded the quantity of alfentanil hydrochloride given in bolus form during the procedure.

The sufentanil citrate perfusion was stopped at the beginning of closure of the dura mater and isoflurane administration was halted at the beginning of skin closure. The N₂O/O₂,
administration was stopped when the skull-pin holder was removed. If necessary, the neuromuscular blockade was reversed with a mixture of neostigmine and glycopyrrolate. The time required for this reversal was noted according to the following parameters: time to spontaneous breathing, breathing rate greater than eight breaths per minute, extubation, eye opening, obeying orders, name recognition, and a score of 9 or 10 on an in-house consciousness scale based on five criteria (Table 1) measured at the moment N₂O was stopped.

Statistical analysis of the results for both groups was performed using the Student t-test for group and unpaired data.

### Results

Both patient groups were well matched for age, height, weight, duration of surgery, and quantity and type of anesthetic agent used (Table 2). The resection of a brain tumor was the most frequent reason for undertaking a craniotomy (seven patients in each group), and various other intracranial procedures necessitated the rest.

There was no difference in hemodynamic data between the groups prior to application of the Mayfield skull-pin holder. No significant increase in blood pressure was observed in the group of patients who received bupivacaine after application of the head holder. One minute after pin insertion, there was a statistically significant rise in arterial pressure in the group of patients who received placebo (a mean rise of 10 mm Hg in systolic pressure [\(p = 0.003\]), 7 mm Hg in diastolic pressure [\(p = 0.02\)], and 9 mm Hg in mean arterial pressure [\(p = 0.004\); Fig. 1 and Table 3]. This increase in pressure had resolved at 5 minutes. A slight decrease in heart rate (a mean of four beats/minute) occurred in patients in both groups (bupivacaine \(p = 0.01\); placebo \(p = 0.02\) following pin insertion.

There was no statistical difference in the amount of alfentanil hydrochloride received by patients in either group, even though blood pressure increased more in those in the placebo group. Seven patients in each group required one or more bolus doses of alfentanil hydrochloride due to increased blood pressure.

Two patients were excluded from statistical analysis of the reversal of general anesthesia: one patient in the placebo group self-extubated, and one patient in the bupivacaine group had a surgical complication unrelated to the study. The time for reversal in the remaining 28 patients was not statistically different, but we observed a trend toward quicker awakening times for each of the criteria recorded among patients in the bupivacaine group.

### Discussion

The application of a Mayfield head holder has been reported to cause major variation in arterial pressure, which may increase 20 to 40 mm Hg compared with prestimulus systolic values. A rise of this magnitude has been associated with adverse reactions, including rupture of unsecured intracranial aneurysms and worsening of intracranial hypertension. Data from a recent study demonstrated that a better overall patient outcome was associated with lower overall arterial pressure during surgery for ruptured aneurysms. In an effort to alleviate hypertensive responses, classic neuroanesthesia protocols involve combinations of high doses of anesthetic agents with long half-lives; delayed clearance and possible accumulation of these drugs contribute to an increased awakening time after surgery. Newer agents, including propofol, which have faster turnover and clearance, are increasingly used to replace longer-acting drugs. A better way to promote awakening may simply be to use less sedation during the procedure. Administering a local anesthetic agent to anatomical sites subject to potential pain is one way to achieve this goal. Given that local infiltration of the incision sites is standard practice in many centers, including ours, we wished to evaluate the additional impact of blunting the stimulation associated with skull-pin placement.

Results of our study demonstrate that 0.5% bupivacaine infiltration of the skin and pericranium before securing the Mayfield head holder can effectively blunt the hemodynamic response associated with this stimulus. We assert that it is a safe procedure given that no increase in heart rate or blood pressure was noted during infiltration in patients in either group in our study. Our results are in agreement with those of other studies reported in the literature. Levin, et al., had similar results using 0.5% mepivacaine with epinephrine. Researchers of another study conducted in a pediatric population reached the same conclusion by using 0.125% and 0.25% bupivacaine with epinephrine. Pinosky, et al., found that a skull block with the aid of 0.5% bupivacaine
was also effective. Hillman, et al., \(^7\) demonstrated that infiltration of the incision line and scalp reflection line, which is common practice for the control of scalp bleeding, prevented hemodynamic stimulation at the beginning of surgery. Note that all changes in our patients occurred prior to incision. The magnitude of the difference in arterial pressure (systolic, diastolic, and mean) between the two groups 1 minute after securing the head holder, although statistically significant, was much less than we expected. A likely explanation might stem from the fact that although we infiltrated the Mayfield pin sites, we did not decrease the dose of the anesthetic drugs used. This could very well have blunted the patient’s response to the securing of his or her head to a greater extent in the placebo group, the members of which were not protected by the effect of bupivacaine, compared with those in the anesthesia group. We did not use epinephrine in our protocol because of its possible role in elevating blood pressure,\(^3,9\) although Levin, et al., \(^8\) found no such association in their study.

There was no increase in heart rate among patients in the placebo group after attaching the head holder, as would have been expected, and in fact we noted a slight decrease. This bradycardic response occurred in both groups. We have no satisfactory explanation for this finding, especially given that investigators of other studies demonstrated a tachycardic response to head pinning or incision, which was attenuated by local anesthesia.\(^7\)

We chose to include bupivacaine in our protocol because of its long half-life (1.5–5.5 hours). We anticipated that its effect would last for the entire surgical procedure and into the postoperative period, providing for a more rapid reversal in patients who received bupivacaine compared with those who received placebo. No statistical difference between the two groups was found. We observed a definite tendency for a quicker consciousness in the patients in whom bupivacaine was administered for each of the criteria evaluated. That this did not reach statistical significance was perhaps related to the small size of our patient groups.

The intraoperative use of supplementary doses of alfentanil hydrochloride for increases in blood pressure resulted in both groups receiving the same amount of that drug, independent of the consequences of securing the Mayfield head holder. This could be another reason why the trend toward a more rapid awakening in patients in the bupivacaine group did not reach statistical significance.

### Conclusions

Infiltration of head-pinning sites with 0.5% bupivacaine is a simple and safe procedure that blunts the hemodynamic response associated with this stimulus. A larger-scale study could help in demonstrating whether this procedure has a real effect on the awakening of patients after surgery.
Administration of bupivacaine during craniotomies


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